INFECTIOUS DISEASES OF BRAZIL

Stephen Berger, MD

GIDEON
E-BOOK SERIES

Infectious Diseases of Brazil - 2013 edition
Stephen Berger, MD
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Scope of Content:
Disease designations may reflect a specific pathogen (ie, Adenovirus infection), generic pathology (Pneumonia - bacterial) or etiologic grouping (Coltiviruses - Old world). Such classification reflects the clinical approach to disease allocation in the Infectious Diseases Module of the GIDEON web application. Similarly, a number of diseases which are generally diagnosed and treated outside of the field of Infectious Diseases are not included, despite the fact that a clear infectious etiology exists. Examples include Peptic ulcer, Tropical spastic paraparesis, Hairy-cell leukemia, Creutzfeldt-Jakob disease, Human papilloma virus infections, etc. In contrast, a number of other entities of unknown etiology which do present to Infectious Diseases specialists have been included: Kawasaki's disease, Chronic fatigue syndrome, Kikuchi and Kimura diseases. Several minor infections having minimal relevance to the field of Geographic Medicine are not covered: Paronychia, Otitis externa, Molluscum contagiosum, etc.

Introduction: The GIDEON e-book series

Infectious Diseases of Brazil is one in a series of GIDEON ebooks which summarize the status of individual infectious diseases, in every country of the world. Data are based on the GIDEON web application (www.gideononline.com) which relies on standard text books, peer-review journals, Health Ministry reports and ProMED, supplemented by an ongoing search of the medical literature.

Chapters are arranged alphabetically, by disease name. Each section is divided into four sub-sections:
1. Descriptive epidemiology
2. Summary of clinical features
3. Status of the disease in Brazil
4. References

The initial items in the first section, Descriptive epidemiology, are defined as follows:

Agent	Classification (e.g., virus, parasite) and taxonomic designation.
Reservoir	Any animal, arthropod, plant, soil or substance in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such a manner that it can be transmitted to a susceptible host.
Vector	An arthropod or other living carrier which transports an infectious agent from an infected organism or reservoir to a susceptible individual or immediate surroundings.
Vehicle	The mode of transmission for an infectious agent. This generally implies a passive and inanimate (i.e., non-vector) mode.

There are 348 generic infectious diseases in the world today. 251 of these are endemic, or potentially endemic, to Brazil. A number of other diseases are not relevant to Brazil and have not been included in this book.

In addition to endemic diseases, we have included all published data regarding imported diseases and infection among expatriates from Brazil.

The availability and quality of literature regarding specific infectious diseases vary from country to country. As such, you may find that many of the sections in this book are limited to a general discussion of the disease itself - with no data regarding Brazil.

This is a book about the geography and epidemiology of Infection. Comprehensive and up-to-date information regarding the causes, diagnosis and treatment of each disease is available in the GIDEON web application. Many of the diseases are generic. For example, such designations as Pneumonia bacterial and Urinary tract infection include a number of individual diseases. These appear under the subheading, Synonyms, listed under each disease.

We welcome feedback, and will be pleased to add any relevant, sourced material. Email us at ebook@gideononline.com

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* Not endemic. Imported, expatriate or other context reported.

+ Country specific note exists for disease

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Actinomycosis

Agent	BACTERIUM. Actinomycetes, Actinomyces spp. An anaerobic gram-positive bacillus
Reservoir	Human - oral, fecal, vaginal flora
Vector	None
Vehicle	Endogenous
Incubation Period	Unknown
Diagnostic Tests	Gram stain and bacteriological culture using strict anaerobic technique. Growth is apparent in 3-7 days.
Typical Adult Therapy	Ampicillin 50 mg/kg/day IV X 4 to 6 weeks - then Amoxicillin 1.5 g/d PO X 6 months. OR Penicillin G 10 to 20 million units/day X 4 to 6w; then Penicillin V X 6 to 12m. Alternatives: Doxycycline, ceftriaxone, Erythromycin Excision/drainage
Typical Pediatric Therapy	Ampicillin 50 mg/kg/day IV X 4 to 6 weeks - then Amoxicillin 20 mg/kg/day PO X 6 months. Penicillin G 100,000 units/kg/day X 4 to 6w; then Penicillin V 25,000 units/day X 6 to 12m. Excision/drainage
Clinical Hints	Mandibular osteomyelitis with fistulae (sulfur granules) in the setting of poor dental hygiene [oral actinomycosis]; intrauterine device and pelvic abscesses [pelvic actinomycosis]; fever, right lower quadrant mass and fistulae [abdominal actinomycosis].
Synonyms	Actinomyces, Aktinomykose, Lumpy jaw. ICD9: 039. ICD10: A42

Clinical

Anatomic variants of Actinomycosis

Oral-cervical actinomycosis accounts for 55% of actinomycosis, and may be manifested as soft tissue swelling, an abscess, or a mass lesion. [1]
• Lesions may be multiple, and relapse following short courses of therapy.
• The disease often spreads to adjacent structures (masseter muscle, carotid artery, cranium, cervical spine, trachea, or thorax) without regard for normal tissue planes.
• Lymphatic spread and lymphadenopathy are rare.
• Infection is associated with pain, fever, and leukocytosis.

Periapical actinomycosis [2] is common and responds to dental care and antibiotics.
• The most common location for actinomycosis is the perimandibular region.
• Periapical infection often precedes infection, which is usually seen at the angle of the jaw; however, the cheek, submental space, retromandibular space, and temporomandibular joint may be affected.
• The overlying skin is often blue to red-purple in color, and sinuses may appear.
• An abscess may ensue, with trismus.
• Mandibular periostitis and osteomyelitis are rarely encountered.
• Maxillary or ethmoid disease, with or without osteomyelitis, is uncommon; but maxillary sinusitis and associated cutaneous fistulas can occur.
• Masses of the hard palate, tongue, nasal septum, head and neck, salivary glands, thyroglossal ducts, thyroid, branchial cleft cysts, lacrimal ducts, orbital structures and larynx have also been reported.
• The tonsils are rarely, if ever, involved; however, infection of the external or middle ear, temporal bone and mastoid may occur following spread of facial disease.

Thoracic actinomycosis [3] accounts for 15% of actinomycosis cases, and represents aspiration of organisms from the pharynx (rarely direct extension from the head and neck or abdominal cavity).
• Most cases present as an indolent, slowly progressive process involving the lung parenchyma and pleura.
• Chest pain, fever, and weight loss are common; occasionally with hemoptysis and a productive cough.
• X-ray findings are non-specific.
• The usual appearance is either a mass lesion or pneumonitis with or without pleural involvement.
• An air bronchogram within a mass lesion is suggestive when present, pleural thickening, effusion, or empyema is seen in more than 50% of cases.
• An isolated pleural effusion may drain spontaneously through the chest wall or produce a soft tissue or breast mass; or posteriorly, to involve the vertebrae or paraspinal structures or spinal cord
• Pulmonary disease may extend across fissures or pleura, and involve the mediastinum, pericardium (rarely endocardium)

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or contiguous bone.

Abdominal actinomycosis [4] accounts for 20% of actinomycosis and represents ingestion of bacteria, hematogenous infection or extension from the female pelvis.
• Associated fever, weight loss, abdominal pain or fullness and changing bowel habits may be present for months before the diagnosis is suspected.
• Physical findings include mass lesions and sinus tracts of the abdominal wall.
• Lymphadenopathy is uncommon.
• 65% of cases are associated with appendicitis, and 65% of lesions present in the right iliac fossa.
• Associated tuboovarian infection, hepatic abscesses [5] , diverticulitis or foreign body perforation in the transverse or sigmoid colon may also be encountered.
• Other associated factors include previous gastric of bowel surgery, typhoid fever, amebic dysentery, trauma, and pancreatitis.
• Abdominal infection may extend to the liver hematogenously; and perirectal or perianal infection is occasionally encountered, resulting in chronic fistulae, sinuses and strictures.

Pelvic actinomycosis [6] may represent spread from intra-abdominal infection; but is most often a complication of intra-uterine device (IUD) placement.
• Any type of IUD can cause infection; and on average, the device has been in place for eight years prior to the appearance of actinomycosis.
• Infection may even occur months following removal of the device.
• Infection is manifest as endometritis or a mass/abscess of the tubes or ovaries. [7]
• Presenting features consist of chronic fever, weight loss, abdominal pain, and vaginal bleeding .
• A "frozen pelvis" suggestive of malignancy or endometriosis is often encountered; and the infection may involve the ureters, bladder, rectum, small or large bowel or peritoneum.
• The diagnostic value of smears and cultures for Actinomyces among asymptomatic women with IUD's is controversial.

Other forms of actinomycosis include:
• brain abscess
• chronic meningitis
• urogenital infection
• musculoskeletal infection
• isolated skin [8] and muscle disease (including mycetoma)
• infected orthopedic prostheses
• thyroiditis
• disseminated hematogenous infection of multiple organs

This disease is endemic or potentially endemic to all countries.

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Adenovirus infection

Agent	VIRUS - DNA. Adenoviridae, Adenovirus Enteric strains classified in genus Mastadenovirus
Reservoir	Human Non-human primates
Vector	None
Vehicle	Droplet Water
Incubation Period	4d - 12d
Diagnostic Tests	Viral culture/serology or antigen assay. Direct fluorescence of secretions. Nucleic acid amplification.
Typical Adult Therapy	Enteric/secretion precautions. Cidofovir has been used in some cases. Symptomatic therapy
Typical Pediatric Therapy	As for adult
Vaccine	Adenovirus
Clinical Hints	Atypical pneumonia, upper respiratory infection, tracheitis, bronchiolitis or keratoconjunctivitis with preauricular adenopathy; uncomplicated illness usually lasts 3 to 5 days; this agent may also cause hemorrhagic cystitis.
Synonyms	Adenovirus gastroenteritis, Epidemic keratoconjunctivitis, Pharyngoconjunctival fever. ICD9: 047.9,077.1,077.2,008.62,480.0 ICD10: A08.2,B30.1,B34.0,J12.0

Clinical

Only 50% of Adenovirus infections are clinically apparent.
- Infection in children usually presents as mild pharyngitis or tracheitis.
- Adenovirus type 7 can cause fulminant bronchiolitis and pneumonia in infants.
- Severe respiratory infection is associated with serotype 14 [1]
- Adenoviruses have been isolated more often than any other nonbacterial pathogen from patients with the whooping cough syndrome; however, a causal relation has not been established.

Cough, fever, sore throat, tonsillitis [2] and rhinorrhea are the most common findings [3] , and usually last 3 to 5 days. [4] [5]
- Rales and rhonchi may be present.
- X-ray studies in patients with pneumonias reveal patchy ground-glass infiltrates primarily in the lower lung fields.
- Outbreaks among military personnel are characterized by tracheobronchitis, with 20% requiring hospitalization.
- The disease is usually is self-limited, superinfection and death are rare.
- Severe infections are increasingly reported among immunocompromised patients. [6-8]
- There are also case reports of severe Adenovirus pneumonia in immunocompetent adults. [9]
- Rare instances of fatal Adenovirus myocarditis have been reported. [10] [11]
- In one series of 3,298 adenoviral infections, pneumonia was found in only 2.4%. [12]
- Adenoviral pneumonia is often followed by bronchiolitis obliterans in children. [13] [14]

Pharyngoconjunctival fever:

Pharyngoconjunctival fever often occurs in the setting of small outbreaks.
- Illness is characterized by conjunctivitis, pharyngitis, rhinitis, cervical lymphadenitis, and fever to 38 C.
- The onset is acute, and symptoms last 3 to 5 days.
- Bulbar and palpebral conjunctivitis, usually bilateral, may be the only finding.
- The palpebral conjunctivae have a granular appearance.
- Bacterial superinfection and permanent residua are unusual.
- Respiratory involvement usually does not progress to the bronchi or lungs.
- Contaminated swimming pools and ponds have been implicated as sources of spread.

Epidemic keratoconjunctivitis:

Epidemic keratoconjunctivitis has an incubation period of 4 to 24 days, and lasts for 1 to 4 weeks.
- The conjunctivitis is often bilateral, and preauricular adenopathy is common. [15-17]
- Multiple subepithelial corneal infiltrates are often present. [18]
- Visual disturbance may persist for several months.
- Secondary spread to household contacts occurs in 10% of the cases.

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Hemorrhagic cystitis:
Hemorrhagic cystitis is two to three times more common in boys than girls (unlike bacterial cystitis which is predominantly seen in girls). [19]
• Hematuria usually persists for approximately three days.
• There was no seasonal preponderance.
• Adenoviral urethritis is also reported. [20]

Infantile adenoviral enteritis:
Infantile adenoviral enteritis is characterized by watery diarrhea is watery with fever, and may last for 1 to 2 weeks.
• Adenoviruses have also been implicated in the etiology of intussusception, encephalitis and meningoencephalitis. [21]
• Rare instances of intestinal intussusception have been associated with adenoviral gastroenteritis. [22]

Other forms of infection:
Adenoviruses have emerged as important pathogens in immunosuppressed patients, particularly those undergoing bone marrow or solid organ transplantation.
• Syndromes include infection of the transplanted organ, or disseminated infection involving the lung, colon (ie, chronic diarrhea [23]), and central nervous system.
• Infection, notably of the urinary [24] and gastrointestinal tracts, is also a common complication of AIDS.
• Adenoviral parotitis and encephalitis are also reported in AIDS patients.

This disease is endemic or potentially endemic to all countries.

Adenovirus infection in Brazil

Prevalence surveys:

6.5% of influenza-like illness with proven viral etiology among adults in Sao Paulo (2001 to 2003) [25]
3.6% of acute respiratory infections in the age group < 5 years (1984 to 1986)
3.7% of children below age 5 years with acute respiratory disease in Uberlandia (2001 to 2004) [26]
4.6% of childhood hospitalizations for lower respiratory tract infection (Sao Paulo, 1995 to 1996). [27]
8.2% of children hospitalized for respiratory tract infection (Sao Paulo, 1995 to 2000) [28]
3.37% of children below age 5 years hospitalized with respiratory infection (2005 to 2006) [29]
25% of children below age 5 years with acute respiratory infection, presenting to an emergency service in northeastern Brazil. (2011 publication) [30]
47.1% of children with tonsillar diseases (2912 publication) [31]
7.1% of nasopharyngeal aspirate samples from children with acute respiratory diseases in Uberlandia, Minas Gerais (2000 to 2007) [32]
10% of nasopharyngeal aspirates from patients hospitalized with acute respiratory disease (PCR, 2008) [33]
6% of acute respiratory infections in children below age 5 (Rio Grand do Sul, 1990 to 1992) [34]
2% of pediatric acute respiratory infection in Salvador (1998) [35]
0.5% of children with acute respiratory illness (Rio de Janeiro and Teresopolis, 2006 to 2007) [36]
6.8% of children (age below age 5 years) in Sao Paulo with lower respiratory tract infection (2003) [37]
9.9% of acute respiratory infections among ambulatory children in northeastern Brazil (1991 publication) [38]
2.2% of outpatient and 0.8% of hospitalized patients with respiratory infection (Curituba, 2005 publication) [39]
23.3% of fatal respiratory infection in children (1985 to 2005) [40]
1.55% of diarrhea among children below age 5 years (1998 to 2000) [41]
2% of children with diarrhea in Rio de Janeiro (1996 to 2003) and Salvador (2001 to 2003) [42]
10% of severe diarrhea among children in Sao Paulo (1993 publication) [43]
6.3% of children below age 6 years with diarrhea, in Porto Velho (Rondonia, 2006 publication) [44]
6.4% of children below age 5 with diarrhea and 1.7% of healthy controls, in Rondonia (2007 publication) [45]
Norovirus or enteric Adenovirus coinfection was found in 14% of rotavirus-positive samples from hospitalized patients (2012 publication) [46]
3.6% of hospitalized children ages <= 3 years with diarrhea (Mato Grosso do Sul, 2000 to 2004) [47]
7.5% of HIV-positive patients with diarrhea, and 14.4% of HIV-negative patients with diarrhea (1995 to 1999) [48]
66% of conjunctival and 27% of vitreous viral cultures (Sao Paulo, 1987 to 2001) [49]
30.8% of water samples in the area of Manaus (2007 publication) [50]
16.7% of recreational waters in a lagoon (Rio de Janeiro, 2012 publication) [51]
100% of water samples in Condordia, Santa Catarina (2012 publication) [52]
55% of seawater samples along beaches in Florianopolis, Santa Catarina (2009 to 2010) [53]
100% of sludge and 66.6% of wastewater samples in Floranopolis City (2010 publication) [54]
50% of sewage effluent samples in Porto Alegre (2012 publication) [55]

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64.2% of water samples and 87.5% of oyster samples in Florianopolis (2007 to 2008) [56]
21.43% of surface water samples in Arroio Diluvio, Porto Alegre (2009) [57]
96% of surface water samples in Florianopolis, Santa Catarina (2012 publication) [58]

Notable outbreaks:

1976 - An outbreak of pharyngoconjunctival fever caused by Adenovirus was reported in Belem. [59]

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Aeromonas & marine Vibrio infx.

Agent	BACTERIUM. Aeromonas hydrophila & Vibrio vulnificus, et al Facultative gram-negative bacilli
Reservoir	Salt or brackish water Fish
Vector	None
Vehicle	Water/shellfish - contact or ingestion
Incubation Period	Range 2d - 7d
Diagnostic Tests	Culture. Notify laboratory if these organisms are suspected in stool.
Typical Adult Therapy	Fluoroquinolone or Sulfamethoxazole/trimethoprim . Other antimicrobial agent as determined by susceptibility testing
Typical Pediatric Therapy	Sulfamethoxazole/trimethoprim . Or other antimicrobial agent as determined by susceptibility testing
Clinical Hints	Diarrhea, fever, vomiting or sepsis after marine injury or ingestion of raw oysters/contaminated fresh or brackish water; fecal leukocytes present; severe or fatal in immunosuppressed or alcoholic patients.
Synonyms	Aeromonas, Aeromonas hydrophila, Vibrio mimicus, Vibrio vulnificus. ICD9: 005.81,027.9 ICD10: A48.8

Clinical

***Aeromonas hydrophila* gastroenteritis:**
There is controversy as to whether *Aeromonas hydrophila* can cause gastroenteritis.
• Volunteer feeding studies using as many as 1 billion cells have failed to elicit illness.
• The presence of this species in the stools of individuals with diarrhea, in the absence of other known enteric pathogens, suggests that it has some role in disease. [1]
• *Aeromonas* species are often implicated in traumatic and surgical wound sepsis [2] [3] and a variety of localized infections. [4-8]
• *Aeromonas caviae* and *A. sobria* are considered by many as "putative pathogens," in diarrheal disease.

Two types of gastroenteritis have been associated with *A. hydrophila* [9] :
• a cholera-like illness with a watery diarrhea
• a dysenteric illness characterized by loose stools containing blood and mucus.
• cases of hemolytic uremic syndrome have followed *Aeromonas* infection [10]

Generalized systemic infection has been observed in individuals with underlying illness.

***Vibrio vulnificus*:**
Vibrio vulnificus causes septicemia in persons with chronic liver disease, alcoholism or hemochromatosis, and immunosuppressed patients. [11] [12]
• The disease appears 12 hours to 3 days after eating raw or undercooked seafood, especially oysters.
• One third of the patients are in shock within 12 hours after hospital admission.
• Three quarters have distinctive, bullous skin lesions which may be mistaken for pemphigus or pemphigoid.
• Thrombocytopenia is common and there is often evidence of disseminated intravascular coagulation.
• Over 50 percent of patients with septicemia die; and the mortality rate exceeds 90 percent among those with hypotension.

Relatively high mortality rates are associated with necrotizing fasciitis caused by *Aeromonas* or *Vibrio* species. [13]

V. vulnificus can also infect wounds sustained in coastal or estuarine waters.
• Infections range from mild self limited lesions to rapidly progressive cellulitis or myositis that can mimic clostridial myonecrosis clinically.

Additional species of *Aeromonas* and *Vibrio* are described in the Microbiology module.

This disease is endemic or potentially endemic to all countries.

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Aeromonas & marine Vibrio infx. in Brazil

Aeromonas caviae, *A. eucrenophila*, *A. media*, *A. sobria*, *A. trota*, *A. veronii bv. sobria*, *A. veronii bv. veronii* and *Aeromonas* sp. have been identified in oysters (*Crassostrea rhizophorea*) in the area of Fortaleza, Ceara. [14]
- *Vibrio parahaemolyticus*, *Vibrio carchariae*, *Vibrio alginolyticus* and *Vibrio vulnificus* have been isolated from oysters (*Crassostrea rhizophorae*) served raw in restaurants. (Rio de Janeiro, 2007 publication) [15]

Prevalence surveys:

Various *Aeromonas* species identified in rectal swabs from 2.4% of hospitalized neonates in Rio De Janeiro (2008 publication) [16]

V. vulnificus was detected in 80% of untreated and 60% of treated oysters from the Cananeia estuary; *Aeromonas* was found in 70% of untreated and 30% of treated oysters. (Sao Paulo, 1998 to 1999) [17]

Aeromonas was found in 2.6% of patients with diarrhea, vs. 0.3% of controls (2009 publication) [18]

Aeromonas species were found in 6% of drinking water samples in Sao Paulo (2007 publication) [19]

Aeromonas species were found in 91.6% of water samples from an integrated aquaculture system (2010 publication) [20]

Notable outbreaks:

1992 - An outbreak (6 cases) of diarrhea was ascribed to infection by *Vibrio metschnikovii*. [21]

2004 - An outbreak (2,170) of diarrhea in Pernambuco was caused by a variety of *Aeromonas* and other bacterial species. [22]

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African tick bite fever

Agent	BACTERIUM. Rickettsia africae
Reservoir	Sheep Goat Cattle Tick
Vector	Tick (Rhipicephalus, Haemaphysalis, Amblyomma)
Vehicle	None
Incubation Period	6d - 7d (range 3d - 18d)
Diagnostic Tests	Serology. Demonstration of rickettsiae by immunofluorescence or culture. Nucleic acid amplification
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3 to 5d. OR Chloramphenicol 500 mg PO QID X 3 to 5d
Typical Pediatric Therapy	Doxycycline 2 mg/kg PO BID X 3 to 5d (maximum 200 mg/day). OR Chloramphenicol 10 mg/kg PO QID X 3 to 5d
Clinical Hints	Fever and rash following a tick bite. Unlike Mediterranean spotted fever: 1) multiple eschars may be present; and 2) the rash is vesicular, and present in only 30% of patients.
Synonyms	Rickettsia africae, South African spotted fever. ICD9: 082.1 ICD10: A77.1

Clinical

As in other rickettsial spotted fevers, African tick bite fever is an acute illness associated with fever, lethargy, headache and myalgia. [1]

• Unlike *Rickettsia conorii*, *R. africae* infection is characterized by a low incidence of rash (usually vesicular) and the common finding of regional lymphadenopathy and multiple eschars. [2]

• The most common presentation is a flu-like illness. [3]

• An inoculation eschar is present in up to 50% of cases, with 20% to 45% having multiple eschars -they may be overlooked in dark skin, in the hair, or in the anogenital region. [4-6]

• Among elderly patients, rash is present in 87.5% (vesicular in 100%), enanthem in 50%, prolonged fever in 75%, chills 87.5%, asthenia 50%, anorexia 50% and weight loss (12.5%) [7]

• Reactive arthritis occurs occasionally. [8]

• Fever usually defervesces within 48 hours of anti-rickettsial therapy. [9]

• There are case reports of prolonged fever up to 3 weeks • consider in returned travelers from endemic areas with prolonged fever.

• Aphthous ulceration and lymphangitis have also been reported rarely. [10] [11]

Laboratory studies:

• Moderate lymphopenia, elevated CRP are seen at presentation in most cases. 40% have elevated liver enzymes, and 20% have thrombocytopenia. [12]

• Median time to development of IgM and IgG antibodies are 25 and 28 days respectively.

• Seroconversion may not occur in mild cases, or if treated early with doxycycline. [13]

• Complications are rare, and there have been no known fatal cases. [14]

This disease is endemic or potentially endemic to 31 countries. Although African tick bite fever is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

African tick bite fever in Brazil

A South African tourist died of spotted fever after arriving to Brazil (2008 publication). [15-20]

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AIDS

Agent	VIRUS - RNA. Retroviridae, Lentivirinae: Human Immunodeficiency Virus, HIV
Reservoir	Human
Vector	None
Vehicle	Blood Semen Sexual Transplacental Breast-feeding
Incubation Period	2m - 10y (50% within 10y)
Diagnostic Tests	HIV antibody (ELISA, Western blot). Nucleic acid amplification. Tests for HIV antigen & viral load as indicated.
Typical Adult Therapy	Two nucleosides + 1 protease inhibitor; or two nucleosides + 1 non-nucleoside; or 2 nucleosides + Ritonavir (alone or with lopinavir) + (indinavir, amprenavir, saquinavir or nelfinavir)
Typical Pediatric Therapy	As for adult
Clinical Hints	Most often associated with drug abuse, blood products, men who have sex with men, hemophilia. Hints: severe herpes simplex or moniliasis, chronic cough, diarrhea, weight loss, lymphadenopathy, retinitis, encephalitis or Kaposi's sarcoma.
Synonyms	ARC, Gay cancer, GRID, HIV-1, HIV-2, HIV-AIDS, SIDA, Slim disease. ICD9: 042 ICD10: B20,B21,B22,B23,B24

Clinical

CDC case surveillance definition:
As of 1993, the CDC (The United States Centers for Disease Control) surveillance case definition for AIDS includes all HIV-infected persons age 13 or over who have either. [1]
• a) a <200 CD4+ T-lymphocytes
• b) a CD4+ T-lymphocyte percentage of total lymphocytes of <14%
• or c) any of the following: pulmonary tuberculosis, recurrent pneumonia, or invasive cervical cancer; or any of the 23 clinical conditions defined in the case definition published in 1987. [2]
• For WHO case definition (1994) see reference [3]

The clinical features of AIDS are protean and often characterized by multisystem illness, evidence of immune suppression and the presence of one or more superinfections (tuberculosis [4] , Cytomegalovirus infection, cerebral toxoplasmosis [5] , pneumocystosis [6] [7] , penicilliosis [8] [9] , severe or recalcitrant candidiasis, disseminated *Acanthamoeba* infection [10] , etc).

Acute HIV infection is characterized by fever, generalized lymphadenopathy, headache, fatigue, myalgia, rash, nausea, vomiting, night sweats, sore throat, diarrhea or weight loss. [11]
• 40% to 90% of persons have symptoms suggestive of an acute viral infection.
• Symptoms tend to subside within two weeks; however, some patients continue to be ill for as long as ten weeks.
• In most cases, a history of likely acquisition within the past several weeks can be established: unprotected sex, extra-medical injection, transfusion, etc.

HIV infection and opportunistic pathogens:

HIV infection increases the incidence and severity of a wide variety of infectious diseases [12] caused by viruses, mycobacteria, actinomycetes, treponemes, fungi [13-17] , protozoa and helminths.
• HIV infection increases the incidence and severity of clinical malaria; however, in severe malaria the level of parasitemia is similar in HIV-positive and HIV-negative patients. [18-25]
• During pregnancy, HIV infection increases the incidence of clinical malaria, maternal morbidity, and fetal and neonatal morbi-mortality.
• HIV infection increases severity of malaria, the risk of malaria treatment failure, and for cerebral malaria in children. [26] [27]
• Some antimalarial drugs may inhibit HIV, while certain anti-retroviral drugs are effective against *Plasmodium* species. [28]
• Reactivation of Chagas disease encephalopathy has been reported among infected HIV-positive patients. [29]
• Acquired syphilis in patients with HIV infection is characterized by severe and accelerated infection, often with overt meningitis, hepatitis [30] and other forms of systemic involvement. [31-38] The presence of concurrent syphilis does not affect the progression of AIDS. [39]
• *Haemophilus ducreyi* has been associated with esophageal ulceration in HIV-positive patients. [40]

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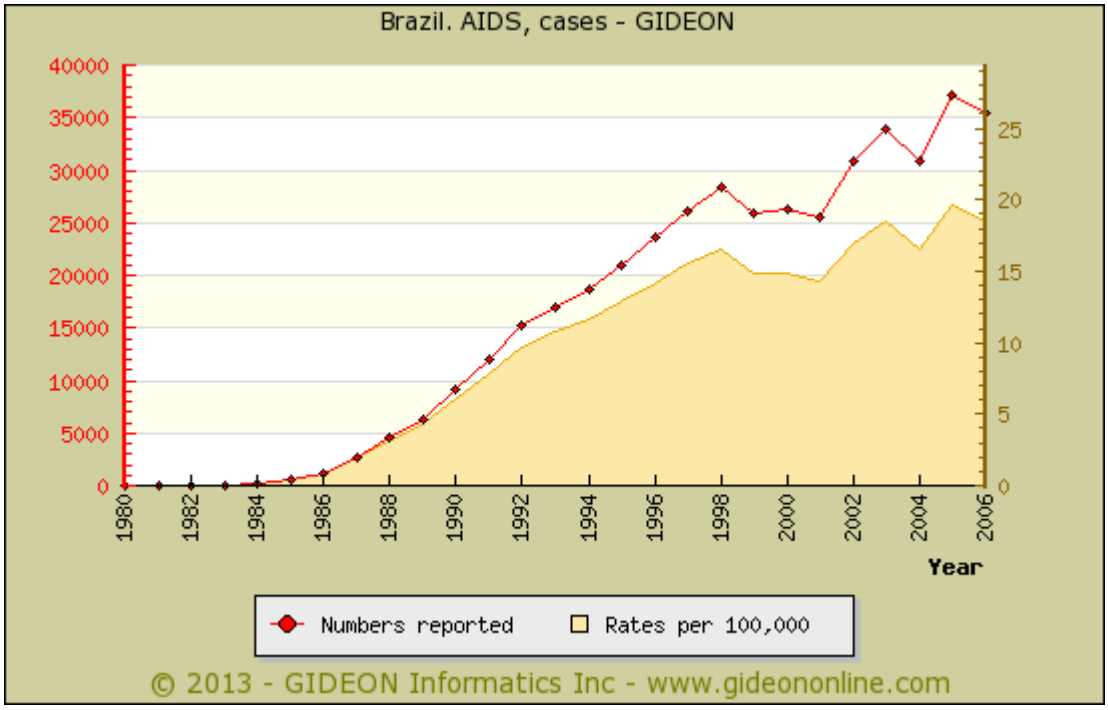
• Hepatitis G infection appears to improve survival among persons with concurrent HIV infection. [41] 41% of infants born to mothers with HIV-HGB-C coinfection acquired HGB-C infection (Thailand, 2009 publication) [42]
• Concurrent HIV infection increases the incidence of cirrhosis and HCC among Hepatitis B carriers [43] [44] ; and shortens the time to development of chronic liver disease in patients with Hepatitis C. [45]
• HIV-HCV coinfection is characterized by more rapid progression to cirrhosis and diminished response to peginterferon/ ribavirin therapy. [46-50]
• Hepatitis D is associated with relatively aggressive disease among patients with HIV-HBV coinfection. [51]
• Concurrent HIV infection may prolong the duration of viremia in patients with hepatitis A. [52]
• Lesions of Herpes simplex in HIV-positive patients may be vegetative, hypertrophic, condyloma-like, nodular, ulcerative, or tumor-like nodules or plaques. [53]

This disease is endemic or potentially endemic to all countries.

AIDS in Brazil

The first case of AIDS was registered in Sao Paulo in 1980.



Graph: Brazil. AIDS, cases

Notes:
1. 10,139 cases were reported in Sao Paulo in 1997; 7,290 in 2000.
2. 7,973 cases of AIDS were reported from the Brazilian border areas during 1990 to 2003 - 648 from the Amazon area, 1,579 from the Midwest and 5,746 from the Extreme south. [54]
3. Analysis of cases reported during 1991 to 2000 - see reference [55]

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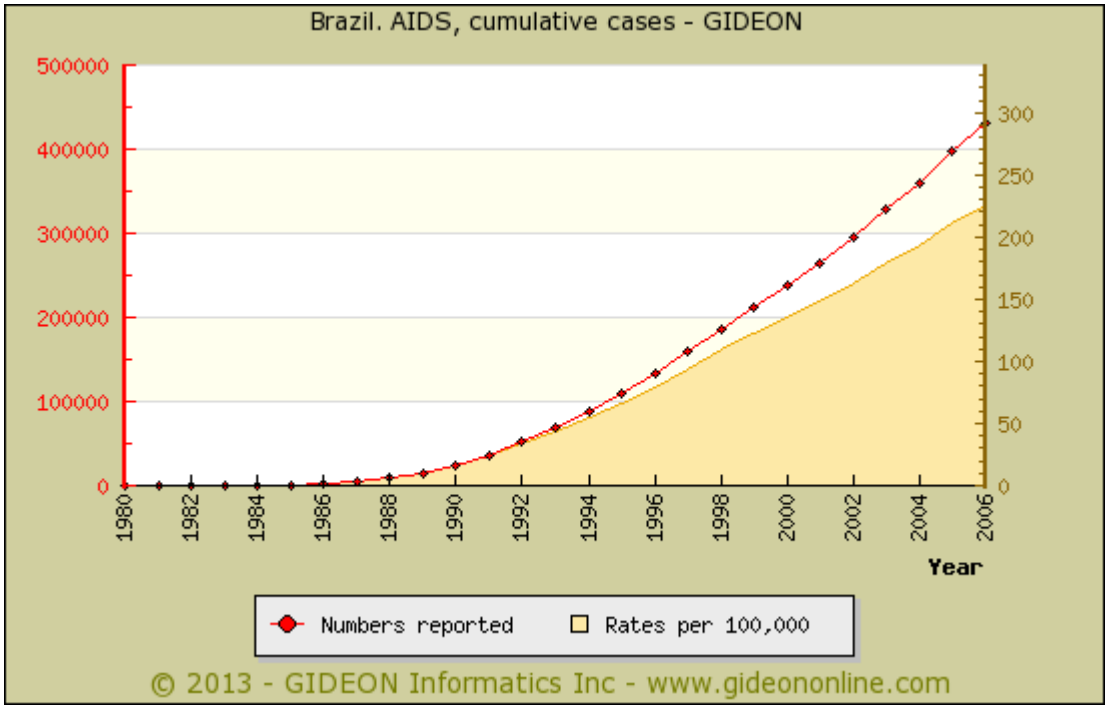

Graph: Brazil. AIDS, cumulative cases

Notes:

1. Analysis of cases reported during 1987 to 1996 - see reference [56]

Demography and risk factors:

- Cases reported during 1995 to 1997: 89% ages 15 to 49; 76% males; 38% heterosexual; 30% men who have sex with men 25% IDU; 3% transfusion or hemophilia-related; 4% mother to infant.
- Cases reported during 1996 to 1998: 89% ages 15 to 49; 71% males. During 1996 to 1998: 41% heterosexual; 29% men who have sex with men; 23% IDU; 2% transfusion/hemophilia; 5% mother to infant.
- Cases reported 1997 to 2001: 89% ages 15 to 49; 67% males; 50% heterosexual; 28% men who have sex with men; 18% IDU; 0% transfusion/hemophilia; 4% mother to infant.

105,595 AIDS deaths were officially reported to December 1999

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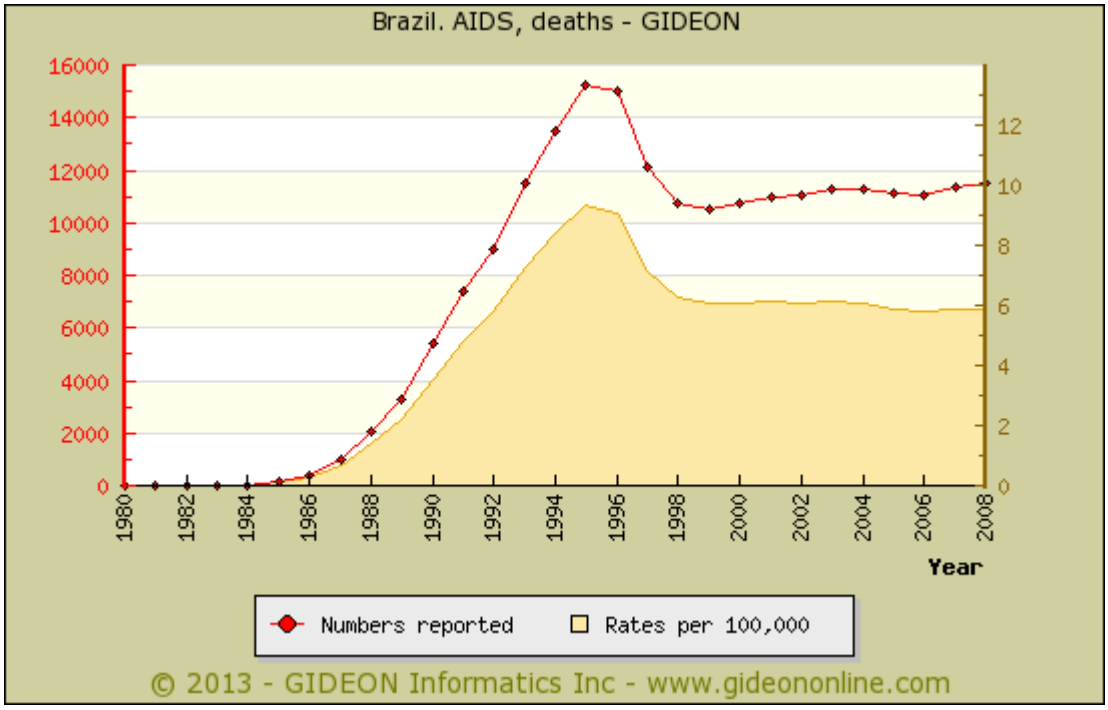

Graph: Brazil. AIDS, deaths

Notes:
1. 41,000 AIDS orphans were estimated to December 1999; 130,000 in December 2001.
2. Number for 1999 represents estimated AIDS deaths
3. 10,024 AIDS-related deaths were reported in Rio de Janeiro State during 1991 to 1995.

Rio and Sao Paulo have accounted for 58.6% of cases, with rates of 456.7 per 100,000 in Santos.
- In 1996, Sao Paulo reported the highest incidence of AIDS (23.0 per 100,000), while Rio de Janeiro reported the highest prevalence (62.4 per 100,000).
- AIDS rates in 1998 were 25.13 per 100,000 in Sao Paulo and 22.67 per 100,000 in Rio de Janeiro.
- AIDS rates in 1999 were 17.47 per 100,000 in Sao Paulo and 13.56 per 100,000 in Rio de Janeiro.

Seroprevalence surveys:

0.55% of the general population in Bahia (1998 to 2000) [57]
1.8% of homeless persons in Sao Paulo (2002 to 2003) [58]
5% of beggar blood donors in Rio de Janeiro (1987 publication) [59]
0.204% of blood donor candidates in Recife (1998 to 2003) [60]
4.9% of homeless people in Sao Paulo (2006 to 2007) [61]
12.6% of MSM (Rio de Janeiro, 2009)
55% of hemophilia patients (1990)
0.8% of sickle cell anemia patients in Bahia (1995 to 2009) [62]
0% of adults with psoriasis (Salvador, Bahia, 2012 publication) [63]
0.80% of patients with mental illness (2009 publication) [64]
0% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (2007 publication) [65]
0.6% of young women in Vitoria (2006) [66]
1.9% of women in Serra Pelada, Para State (2004) [67]
1.7% to 6.2% of CSW in 1994; 2.9% in 2001; 7% in 2003
0.6% of pregnant women (1997)
0.4% of rural pregnant women (1998)
0.6% of pregnant women in Parana (1996 to 1998); 3% to 6% in Rio Grande do Sul (non-clinic attendees)
0.3% of pregnant women in Parana (2010) [68]
0.5% of pregnant women in Cuiaba, Mato Grosso (2001 to 2002) [69]
0.5% of pregnant women in southern Brazil (prenatal clinic patients, 2003) [70]
0.2% of pregnant women in Mato Grosso do Sul (2002 to 2003) [71]
0.6% of pregnant women in Espiritu Santo (2007) [72]
0.09% of pregnant women in Goiania City, central Brazil (2004 to 2005) [73]
1.7% of pregnant women in Itajai, Santa Catarina State (2002 to 2007) [74]

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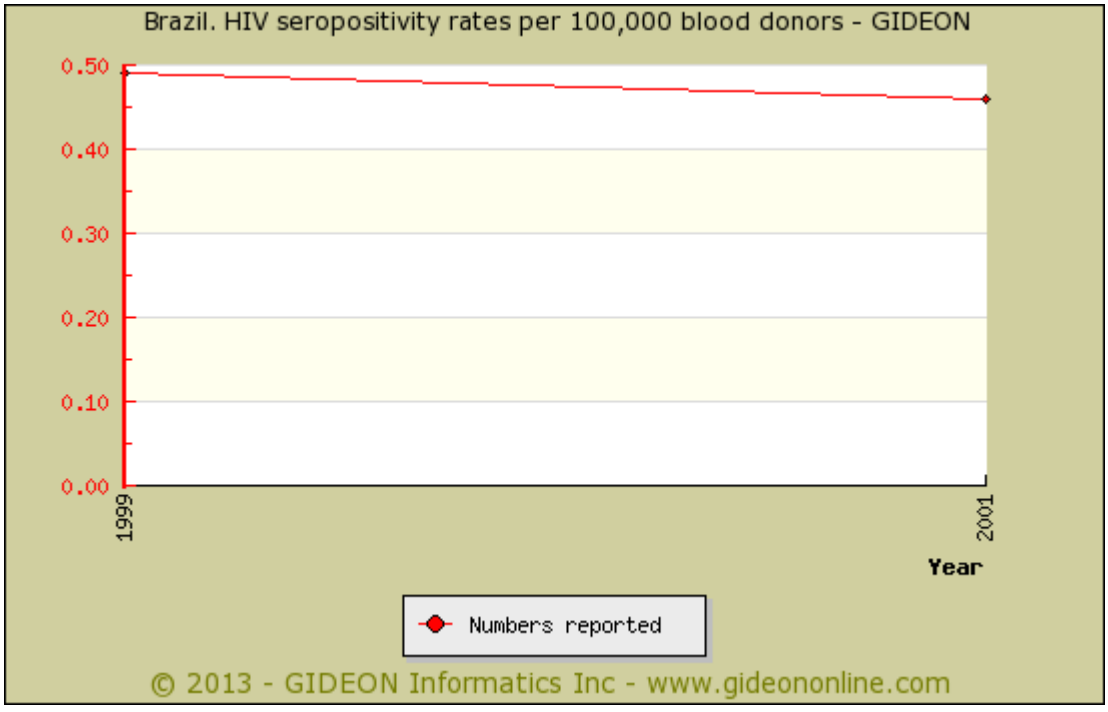
0.14% of pregnant women in Sergipe (2009 publication) [75]
2.1% of pregnant women in Sao Paulo (2011 publication) [76]
0.6% of pregnant women in the western Amazon (2008) [77]
0.9% of low-income postpartum and 0% of pregnant women treated in Greater Metropolitan Vitoria, Espirito Santo State (1999) [78]
6.2% of FSW, 13.6% of MSM and 23.1% of IDU (meta-analysis, 1999 to 2009) [79]
29% of IDU in 1996; 28% in 1998; 46.0% in 2000
5.9% of IDU in Rio de Janeiro (2009)
50% of IDU in Salvador in 1996, and only 7% in 2001
31% of IDU in Itagui and 64% in Porto Alegre (2003)
1.6% of female crack cocaine users in Bahia, Salvador (2007 publication) [80]
7.89% of ex-IDU in Rio de Janeiro (1999 to 2001) [81]
16% of prisoners (1993) [82]
0.34% of incarcerated adolescents (Salvador, 2008 publication) [83]
13.9% of female prisoners (Sao Paulo, 2006 publication) [84]
18% of STD patients (1995)
6.8% of STD clinic patients in Vitoria (2007 publication) [85]
2.5% of male STD clinic patients in Manaus (2008 publication) [86]
1.3% of STD clinic patients in Ceara (1999 to 2008) [87]
24.7% of patients referred to an Infectious Diseases clinic in Sao Paulo (2009 publication) [88]
4.8% of persons seeking voluntary HOV testing and counseling (Rio de Janeiro, 2004 to 2005) [89]
8.9% of recyclable waste collectors in Santos (2005) [90]
1.4% of men and 0.0% of women in Alto Solimoes, Amazon border region (2012 publication) [91]
0.3% of truck drivers crossing the southern Brazilian border at Foz do Iguacu (2003 to 2005) [92]

Rates of HIV infection among CSW are related to duration of prostitution, lower price charged for sex, and presence of concurrent STD (ten cities, 2008 to 2009) [93] [94]

Four cases of HIV infection from occupational exposure were documented among health care workers during 1981 to 2004. [95]



Graph: Brazil. HIV seropositivity rates per 100,000 blood donors

Notes:
Individual years:
2001 - 0.7% in Sao Paulo

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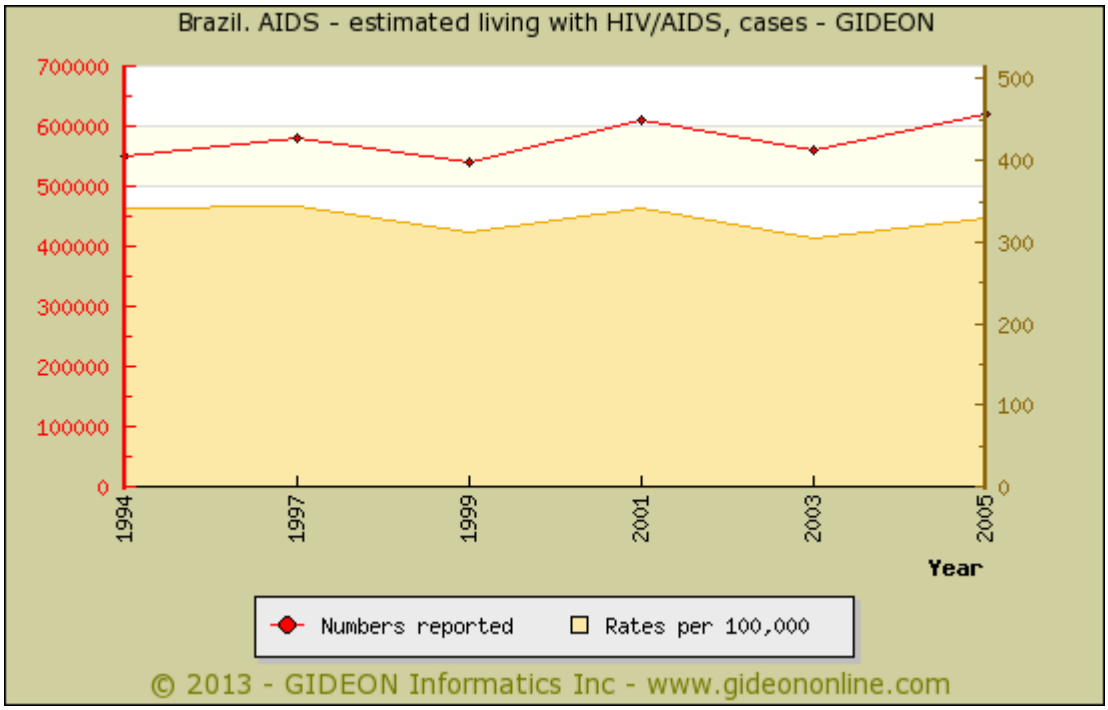

Graph: Brazil. AIDS - estimated living with HIV/AIDS, cases

Notes:
1. Figure for 1997 represents 0.63% of all adults; 0.65% in 2000; 0.7% in 2001; 0.7% in 2003.
2. The reported nationwide prevalence of AIDS was 32.5 per 100,000 in 1996
3. The rate of new HIV infection was 15.9 per 100,000 in 1998; 12.3 per 100,000 in 2001.
4. In 2006, 180,000 HIV-infected persons in Brazil were receiving HAART. [96]

Opportunistic infections:

Mycobacteriosis

- Tuberculosis was the cause of death in 28% of autopsied AIDS patients, bacterial pneumonia 17%, histoplasmosis 13%, toxoplasmosis 10%, pneumocystosis 8%, cryptococcosis 5% and bacterial septicemia 4% (Amazonas, 1996 to 2003) [97]

17% of AIDS patients have tuberculosis (30.6% in Ceara State, 1986 to 1992).
Five percent of tuberculosis patients were HIV-positive in 1997; 35.6% in 2001.
49.9% of prison inmates with tuberculosis in Campina, Sao Paulo are HIV-positive (1993 to 2000).
27.6% of patients with tuberculosis in Goias State were found to be HIV-positive - 67% of tuberculosis patients who were coinfected by HCV (2011 publication) [98]

Other bacteria

- 12.9% of AIDS patients have concurrent gonorrhea (1986 to 1992). [99]
- Syphilis coinfection was detected in 2.7% of HIV/AIDS hospital outpatients; ratio of men to women with coinfection was 4:1, with homosexual men the most effected. (Rio de Janeiro, 2005) [100]

Syphilis was found in 20.5% of HIV-positive patients in southern Brazil (1991 to 2008) [101]
Syphilis was found in 3.1% of pregnant HIV-positive women (Rio Grande do Sul, 2012 publication) [102]
Syphilis was found in 9.5% of HIV-positive women in Salvador, Bahia, HBV 3.2%, HCV 8.1%, *Chlamydia trachomatis* 11.1%, *Mycoplasma hominis* 2.1%, *Ureaplasma urealyticum* 2.1%, *Neisseria gonorrhoeae* 0% (2012 publication) [103]

- Bacterial vaginosis is present in 26.6% of HIV-positive women (2008 publication) [104]
- *Chlamydia trachomatis* infection was found in 4.3% of HIV-positive women in Manaus (2009 to 2010) [105]
- EPEC was found in 22.5% of HIV-positive patients with diarrhea, and 5% of control patients; salmonellosis in 5% vs. 1.7%; shigellosis in 0%; Calicivirus in 10% vs. 0%; amebiasis 2.5% vs. 1.7%; cryptosporidiosis 15% vs. 5% and giardiasis in 12.5% vs. 0% (southeastern Brazil, 2006 to 2007) [106]
- 38.4% in HIV positive individuals vs. 34% of controls are seropositive toward *Bartonella* species (Rio de Janeiro, 2010 publication) [107]
- 3.2% of HIV-positive males and 6.3% of HIV-positive females in Jacarepagua, Rio de Janeiro are seropositive toward *Coxiella burnetii* (2009 publication) [108]

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Viruses:

53% of men and 50.7% of women with AIDS in Niteroi, Rio de Janeiro were seropositive toward HSV-2. [109]

62.8% of HIV-positive patients in Rio de Janeiro were seropositive toward Parvovirus B-19 (2009 publication) [110]

24.4% of HIV-positive patients with diarrhea were found to be infected by Cytomegalovirus (2011 publication) [111]

6.4% of HIV-positive patients in the Amazon region were HBsAg-positive [112]

- 3.8% of HIV-positive patients in Vitoria were HBsAg-positive (1993 to 2004) [113]

36.9% of HIV-positive patients in Goiania City, Goias were HBsAg-positive (2008 to 2010) [114]

5.9% of HIV-positive patients in Sao Paulo were found to be HBsAg-positive and 14% seropositive toward HCV (2009 publication) [115]

- 10.8% of pregnant HIV-positive women were found to be positive for HCV, 2.34% HBV and 3.1% syphilis (Rio Grande do Sul, 2012 publication) [116]

5% of HIV-positive patients in the Amazon region carry Hepatitis C virus.

6% of HIV-positive patients in Mato Grosso State carry Hepatitis C antigen, and 10.9% were found to be seropositive (2007 publication) [117]

4.1% of HIV-positive patients in Recife were found to be seropositive toward Hepatitis C (2003) [118]

31.2% of HIV-positive patients in Rio Grande do Sul are seropositive toward Hepatitis C (2010 publication) [119]

4.42% of AIDS patients in Amazonas were found to be seropositive toward Hepatitis C (2000 to 2007) [120]

16.7% of HIV-positive outpatients were found to be seropositive toward Hepatitis C (2005 to 2007) [121]

- 27.6% of patients with tuberculosis in Goias State were found to be HIV-positive - 67% of tuberculosis patients who were coinfected by HCV (2011 publication) [122]

- 79.8% of HIV-positive patients are seropositive toward Hepatitis A (1988 to 2004) [123]

- Rotaviruses / Adenoviruses were found in 1.5% / 7.5% of HIV-positive patients with diarrhea, and 2.5% / 14.4% of HIV-negative patients with diarrhea (1995 to 1999) [124]

Protozoa

- Cryptosporidiosis is the most common cause of diarrhea among HIV-positive children in Southeastern Brazil (Sao Paulo State) [125]

- *Isospora belli* is responsible for 2% to 10.1% of AIDS-associated diarrhea in this country. [126-128]

11.1% and 9.4% of fecal samples from HIV/AIDS patients in the Triangulo Mineiro region were positive for *Isospora* and *Cryptosporidium*, respectively (1993 to 2003) [129]

Isospora was found in 4.7% of HIV-positive patients in northeastern Sao Paulo, *Giardia* 3.5% and *Cryptosporidium* 0.3% (1998 to 2008) [130]

- *Dientamoeba fragilis* infection has been identified in patients with HIV/AIDS. [131] [132]

- Visceral leishmaniasis has emerged as an important complication of HIV-AIDS, particularly in the northeastern region. [133]

25 cases of HIV-*Leishmania* coinfection were reported to November 1995; 100 to July 2003.

An additional 15 cases were reported in a 2009 publication. [134]

The estimated rate of visceral leishmaniasis among HIV-positive patients treated at a tertiary medical center in Brasilia was 16% (2009 publication) [135]

HIV-positive patients with visceral leishmaniasis were found to have a mean age of 37.3 years, vs. HIV-negative leishmaniasis patients; and were more likely to be male (88% vs. 65%, Rio Grande do Norte, 1990 to 2009). [136]

Asymptomatic leishmaniasis was identified in 10.8% of HIV-positive patients (ELISA, Minas Gerais, 1999 to 2001) [137]

- 27.5% to 46% of HIV-positive patients with diarrhea carry *Enterocytozoon bieneusi*. [138]

- 67% of HIV-negative and 72% of HIV-positive pregnant women in Rio Grande do Sul are seropositive toward *Toxoplasma gondii* (2002 to 2005) [139]

4.8% of HIV-positive patients in the southern region had a history of neurotoxoplasmosis, and 80% were seropositive toward *Toxoplasma gondii*. (2009 to 2010) [140]

- Trichomoniasis is present in 12.5% of HIV-positive women (2008 publication) [141]

Metazoa

- Prevalence of parasites in HIV-positive patients before / after HAART as follows: *Strongyloides stercoralis* 30.1% / 11%; *Ascaris lumbricoides* 15.6% / 2%; hookworm 13.7% / 2%; Trichuris trichiura 13.1% / 1%; *Hymenolepis nana* 0 / 1%; *Giardia duodenalis* 7.9% / 1%; *Entamoeba histolytica/dispar* 3.3% / 1%; *Cryptosporidium* 8.1% / 0% (Ceara, 2008 publication) [142]

- Hookworm was found in 1.4% of HIV-positive patients in northeastern Sao Paulo, *Taenia saginata* 0.7% and *Strongyloides* 0.7%. (1998 to 2008) [143]

Fungi

- Systemic fungal diseases were found in 4.6% of AIDS patients in Cuiaba, Mato Grosso - *Cryptococcus neoformans* in 50% of these, *Cryptococcus neoformans var. gattii* 1.6%, *Cryptococcus* spp in 6.6%, *Histoplasma capsulatum* 38.3% and *Paracoccidioides brasiliensis* 3.3% (2005 to 2008) [144]

- 21 HIV-positive patients in Rio de Janeiro were treated for sporotrichosis (7 in disseminated form) during 1999 to 2009.

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[145]

- Cryptococcosis accounted for 0.27% of AIDS deaths (Sao Paulo, 1993 to 2000) [146] ; 9.5% of infectious AIDS deaths in Uberaba (1990 to 2000). [147]

84.5% of persons diagnosed with cryptococcosis at a university hospital were HIV-positive; 89.6% were *Cryptococcus neoformans var. neoformans* and 10.4% var. gattii. (Mato Grosso do Sul, 1995 to 2005) [148]

58.7% of patients with cryptococcosis were found to be HIV-positive. *Cryptococcus neoformans* accounted for 86.5% of CSF *Cryptococcus* isolates from HIV-positive patients; while *Cryptococcus gattii* accounted for 80.8% of isolates from HIV-negative patients (Piaue, 2008 to 2010) [149]

Cryptococcus neoformans var. gattii infection has been reported in AIDS patients. (Rio Grande do Sul, 1999 publication) [150]

Cryptococcosis was associated with 50.9% of deaths among AIDS patients, systemic candidiasis 30.2% and histoplasmosis 10.1% (1996 to 2006) [151]

- 83.3% of histoplasmosis patients hospitalized in Mato Grosso do Sul have AIDS. (1998 to 2005) [152]

164 cases of histoplasmosis were reported among HIV-positive patients in Fortaleza (2007 publication) [153]

208 cases of histoplasmosis were reported among HIV-positive patients in Ceara during 2006 to 2010. [154]

11 cases of oral histoplasmosis (5 HIV-positive) were treated in a single institution in Porto Alegre during 1987 to 2008. [155]

30 cases of histoplasmosis were registered at a University Hospital in Mato Grosso do Sul during 1998 to 2005. 97.7% had disseminated infection and 83.3% had AIDS. [156]

- Paracoccioidomycosis was present in 0.09% of AIDS patients (1992). A series of ten coinfected patients was published in 2003. [157]

Paracoccidioidomycosis was found in 12.2% of HIV-positive patients in a region endemic for paracoccidioidomycosis (2011 publication) [158]

Paracoccidioidomycosis was present in 1.4% of HIV-positive patients in Ribeirao Preto, Sao Paulo (2009 publication) [159]

- Vaginal candidiasis is present in 29.7% of HIV-positive women (2008 publication) [160]

Oral candidiasis was present in 37.0% of HIV-positive patients receiving HAART (1997 to 2004) [161]

Miscellaneous:

10% of submandibular and 7% of cases of sublingual lymphadenopathy in advanced AIDS patients were associated with mycobacteria, 13% and 2% CMV and 3% and 4% cryptococcosis (2009 publication) [162]

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Amoeba - free living

Agent	PARASITE - Protozoa. Centramoebida, Acanthamoebidae: Acanthamoeba and Balamuthia Schizopyrenida, Vahkampfidae: Naegleria
Reservoir	Water Soil
Vector	None
Vehicle	Water (diving, swimming)
Incubation Period	5d - 6d (range 2d - 14d)
Diagnostic Tests	Wet preparation. Specialized cultures. Serology available in reference centers.
Typical Adult Therapy	CNS Naegleria: Amphotericin B to 1 mg/kg/d IV + 1.5 mg intrathecal X 8 days; plus Miconazole 350 mg/sq m/d IV + 10 mg intrathecal qod X 8d Acanthamoeba: Sulfonamides + Flucytosine
Typical Pediatric Therapy	CNS Naegleria: Amphotericin B to 1 mg/kg/d IV + 1.5 mg intrathecal X 8 days; plus Miconazole 350 mg/sq m/d IV + 10 mg intrathecal qod X 8d Acanthamoeba: Sulfonamides + Flucytosine
Clinical Hints	Severe, rapidly-progressing meningoencephalitis (*Naegleria*, *Acanthamoeba* or *Balamuthia*) following swimming or diving in fresh water; or keratitis (*Acanthamoeba*), often following use of contaminated solutions to clean contact lenses.
Synonyms	Acanthamoben, Acanthamoeba, Amebic keratitis, Balamuthia, Balmuthia, Dictyostelium, Free-living ameba, Leptomyxid ameba, Naegleria, Paravahlkampfia, Primary amebic meningoencephalitis, Sappinia, Vahlkampfia. ICD9: 136.2 ICD10: B60.1,B60.2

Clinical

Primary amebic meningoencephalitis usually occurs in children and young adults who have been swimming in warm fresh water. [1]

Infection is heralded by abnormal sensations of taste or smell followed by abrupt onset of fever, nausea, and vomiting.
- The majority of patients have headache, meningitis and disorders of mental status changes.
- Coma and death may ensue within one week
- Only three nonfatal infections had been reported to 2003.

Acanthamoeba encephalitis:

Granulomatous amebic encephalitis due to *Acanthamoeba* occurs in immunocompromised and debilitated patients.
- Infection has a gradual onset characterized focal neurological deficits, mental status abnormalities, seizures, fever, headache, hemiparesis and meningismus.
- Visual disturbances and ataxia are often encountered.
- Death may ensue within 7 to as long as 120 days.
- Secondary infection of a cerebral ependymal cyst has been reported. [2]
- Disseminated *Acanthamoeba* infection has been reported in an HIV-positive patient. [3]

Balamuthia encephalitis:

Balamuthia mandrillaris encephalitis may be associated with headache, low-grade fever, vomiting, ataxia, photophobia, cranial nerve palsy, speech disturbances, cerebellar nystagmus, seizures, and altered mental status. [4] [5]
- The case-fatality rate for *Balamuthia* encephalitis is over 90%.

Acanthamoeba keratitis:

Acanthamoeba keratitis is clinically similar to herpetic infection, and presents with a foreign-body sensation followed by severe pain, photophobia, tearing, blepharospasm, conjunctivitis, iritis, anterior uveitis, dendriform keratitis, radial keratoneuritis, ptosis and blurred vision. [6-11]
- In rare instances, the infection is painless. [12] [13]
- Rupture of Descemet's membrane may occur. [14]
- Bilateral infection is common. [15]
- Dacryoadenitis may be present in some cases. [16]
- Ocular discharge and endophthalmitis are very rare. [17]
- Sympathetic ophthalmia of the un-infected eye has been reported. [18]

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• Atypical presentations have been described in patients with keratoconus. [19]

Acanthamoeba infection has also been associated with skin ulcers [20] , pneumonia, adrenalitis, vasculitis, osteomyelitis, and sinusitis.
• Cutaneous acanthamoebiasis has been associated with ulceronecrotic lesions, an infiltrative bluish plaque, or periorbital tumor. [21]
• Fatal disseminated *Acanthamoeba lenticulata* infection has been reported in a heart transplant patient.
• Four cases of disseminated *Acanthamoeba* infection in stem-cell transplant recipients had been reported as of 2008. [22]

This disease is endemic or potentially endemic to all countries.

Amoeba - free living in Brazil

Sporadic cases are reported. [23]

Both *Acanthamoeba* and *Naegleria* spp. have been identified in hospital dust samples. [24] [25]
- Potentially-pathogenic *Acanthamoeba* species were identified in dust samples at a public hospital in Curitiba, Parana State. [26]

Acanthamoeba genotypes T3, T4 and T5 have been identified in swimming pools in southern Brazil (2011 publication) [27]

Naegleria fowleri has been identified in a lake in Rio de Janeiro. [28]
- *Naegleria fowleri* meningoencephalitis was reported in a bovine in northeastern Brazil. [29]

Prevalence surveys:

35% of biofilms sampled from hospital environments; of these, 34% had morphological characteristics of *Acanthamoeba*. (Porto Alegre, 2004 to 2005) [30]

17% and 39% of dust samples from two university campuses in Santos (2010 publication) [31]

20% of water samples from swimming pools in Porto Alegre (*Acanthamoeba* spp., 2006 to 2007) [32]

9.6% of tap water samples in Rio Grande do Sul (*Acanthamoeba* spp., 2009) [33]

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Amoebic abscess

Agent	PARASITE - Protozoa. Sarcomastigota, Entamoebidea: Entamoeba histolytica (must be distinguished from non-invasive, Entamoeba dispar)
Reservoir	Human
Vector	Fly (Musca) - occasionally
Vehicle	Food Water Sexual contact Fly
Incubation Period	2w - 6m (rarely years; 95% within 6m)
Diagnostic Tests	Imaging. Serology. Nucleic acid amplification. Note: Amoebae are usually not present in stool at this stage.
Typical Adult Therapy	Metronidazole 750 mg TID X 10d OR Tinidazole 800 mg TID X 5d
Typical Pediatric Therapy	Metronidazole 15 mg/kg TID X 10d OR Tinidazole 15 to 20 mg/kg TID X 5d
Clinical Hints	Fever, local pain, weight loss. Remember that liver abscess may be bacterial or amoebic - latter most often single and in right hepatic lobe.
Synonyms	Absceso amebiano, Amebic liver abscess. ICD9: 006.3,006.4,006.5,006.6,006.8 ICD10: A06.4,106.5,A06.7,106.8

Clinical

The clinical presentation may be acute or subacute in onset.
- Fever than 50% of patients have fever, hepatomegaly or abdominal pain.
- 30% to 40% have concurrent diarrhea.
- Other findings may include shoulder pain, cough, chest pain, pleural or pericardial effusion. [1] [2]
- The findings of ameboma may mimic those of malignancy. [3]
- A case of Budd-Chiari syndrome complicating amebic abscess has been reported. [4]

Laboratory findings include leukocytosis without eosinophilia in 80%, anemia in over 50%, elevated serum alkaline phosphatase levels in 80%.

Pleuropulmonary amebiasis is the most common complication of amebic liver abscess, usually representing rupture of a superior right lobe abscess through the diaphragm.
- Symptoms include cough, pleuritic pain, and dyspnea.
- Empyema, hepatobronchial fistula or pericarditis (from left lobe abscesses) may follow.
- Although most cases involve the liver, abscesses may occur in virtually any organ. [5] [6]
- *Entamoeba histolytica* encephalitis has been reported. [7]

This disease is endemic or potentially endemic to all countries.

Amoebic abscess in Brazil

Epidemiological data regarding Amebic abscess are included in the notes for Amebic colitis

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Amoebic colitis

Agent	PARASITE - Protozoa. Sarcomastigota, Entamoebidea: Entamoeba histolytica (must be distinguished from non-invasive, Entamoeba dispar)
Reservoir	Human
Vector	Fly (Musca) - occasionally
Vehicle	Food Water Sexual contact Fly
Incubation Period	1w - 3w (range 3d - 90d)
Diagnostic Tests	Fresh stool/aspirate for microscopy. Stool antigen assay. Stool PCR. Note: serological tests usually negative.
Typical Adult Therapy	Metronidazole 750 mg TID X 10d OR Tinidazole 2 g as single dose daily X 5d
Typical Pediatric Therapy	Metronidazole 15 mg/kg TID X 10d OR Tinidazole 50 mg/kg as single dose daily X 5d
Clinical Hints	Dysentery, abdominal pain, tenesmus - without hyperemia of rectal mucosa or fecal pus (i.e., unlike shigellosis); liver abscess and dysentery rarely coexist in a given patient.
Synonyms	Amebiasis, Amebiasis intestinal, Amebic dysentery, Amoebenruhr, Entamoeba bangladeshi, Entamoeba gingivalis, Entamoeba moshkovskii. ICD9: 006.0,006.1,006.2 ICD10: A06.0,A06.1,A06.2

Clinical

Patients with noninvasive infection may present with nonspecific gastrointestinal complaints such as chronic intermittent diarrhea, mucus, abdominal pain, flatulence and weight loss [1] [2]

Infection has been documented in children as young as two weeks of age. [3] [4]

A review of amebiasis among men who have sex with men • see reference [5]

Cases of cutaneous amebiasis of the penis have been acquired through insertive anal intercourse. [6]

Invasive amebiasis:
The onset of invasive infection is usually gradual (over 1 to 3 weeks) and characterized by abdominal pain, tenderness, and bloody stools.
• Fever is present in one third of cases, and the may be enlarged and tender.
• Signs of fluid loss and electrolyte loss may be seen in severe infections.
• In children, colitis can present as rectal bleeding alone without diarrhea.
• Fecal leukocytes may not be present, and are not as numerous as in shigellosis.
• Charcot-Leyden crystals are often seen in the stool.

Fulminant colitis:
Fulminant colitis is rare and carries a very high mortality.
• Predisposing factors include malnourishment, pregnancy and corticosteroid treatment.
• Such patients are severely ill with fever, leukocytosis, profuse bloody and mucoid diarrhea, generalized abdominal pain.
• Hypotension and peritonitis may be evident.
• Intestinal perforation and necrosis, or hepatic abscess may ensue.
• The clinical features of Cytomegalovirus colitis in AIDS patients may mimic those of amebic colitis. [7]

Additional complications:
Additional complications include toxic megacolon (complicates 0.5% of amebic colitis cases); annular ameboma of the colon, which may mimic carcinoma.
• Chronic, irritative bowel syndromes, ulcerative post-dysenteric colitis or perianal amebiasis may also follow acute amebic colitis.
• Extraintestinal amebiasis may involve a wide variety of organs.
• Other forms of amebiasis include colocutaneous fistula [8] or amebiasis cutis [9] , brain abscess, cervicitis [10] , rectovaginal fistulae and penile infection

Liver abscess is discussed separately in this module.

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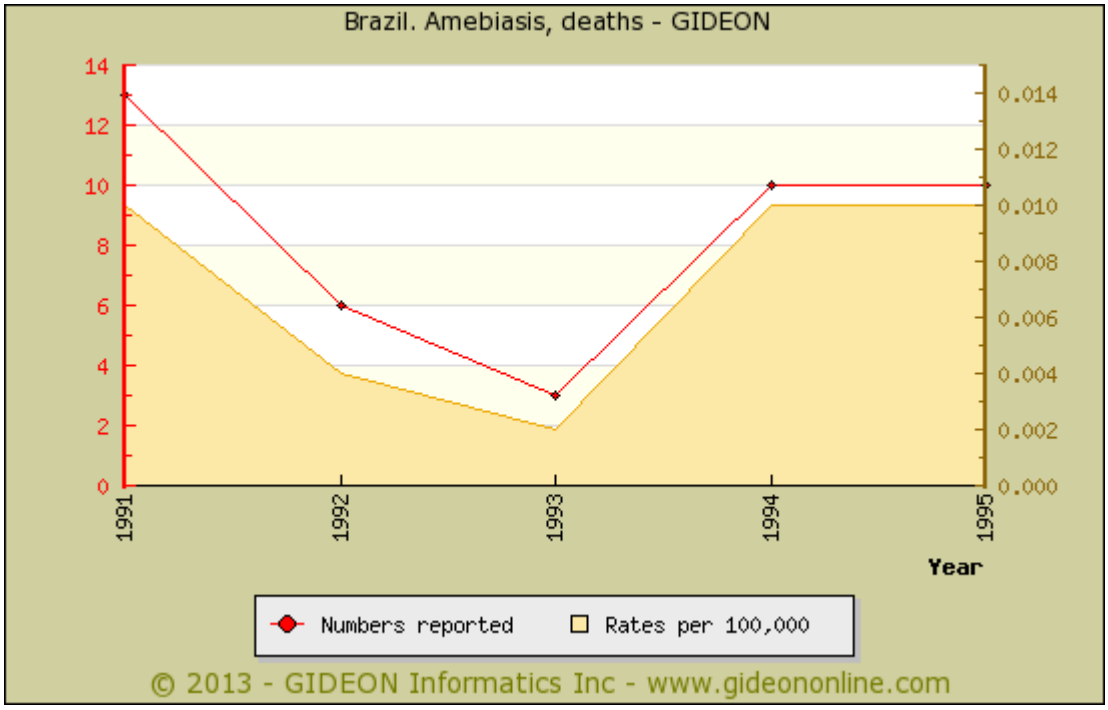
This disease is endemic or potentially endemic to all countries.

Amoebic colitis in Brazil



Graph: Brazil. Amebiasis, deaths

Prevalence surveys:

0.2% of Sao Paulo children below age 5 years (1995 to 1996)
13.7% of children presenting to outpatient clinics in Manaus (2009 publication) [11]
1.8% of urban children with diarrhea in Salvador (2000 to 2002) [12]
5.8% of children with diarrhea in northeastern Brazil (2008 publication) [13]
21.5% of asymptomatic persons in Manaus (2005 publication) [14]
0.2% of persons in Maria Helena, Parana (2004 to 2006) [15]
3.4% of stool samples from clinical laboratories in Salvador City, Bahia State (*E. histolytica/E. dispar*, 2011 publication) [16]
29.35% of diarrhea patients in metropolitan Belem, Para State (2005 publication) [17]
2.5% of HIV-positive patients with diarrhea, and 1.7% of control patients (southeastern Brazil, 2006 to 2007) [18]
0.5% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [19]
20% of asymptomatic persons in slum communities in northeastern Brazil (1998 publication) [20]
9.0% of asymptomatic persons in slum communities in northeastern Brazil (1996 publication) [21]
0.3% of elderly residents at long-term residency institutions in southeastern Brazil (2013 publication) [22]
2.7% of persons in Campos do Jordao (2003 to 2004) [23]
13.3% of persons in Eirunepe, Amazon (2005 publication) [24]
25.3% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [25]
65.0% of the Parakana indigenous community in southwestern Para State (1992) [26]
75% of Hupda and 35.4% of other indigenous peoples in Lauarete (2001) [27]
31.6% of Terena Indians (Mato Grosso do Sul) [28]
10.1% of food handlers in Cascavel, Parana (2009 publication) [29]
0.2% of money bills acquired from shops (Rio Grande do Norte, 2007 publication) [30]

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Anaplasmosis

Agent	BACTERIUM. Anaplasmataceae Anaplasma phagocytophilum. (E. phagocytophila, E. equi "HE agent" have merged into this species) Intracellular Rickettsia-like
Reservoir	Rodent Rabbit Deer Tick
Vector	Ixodes dammini (scapularis); I. pacificus and I. ricinus also implicated
Vehicle	Blood or secretions (rare)
Incubation Period	Unknown; mean 8d
Diagnostic Tests	Intraleucocytic inclusions ('morulae') seen in blood smear. Serology. Nucleic acid amplification/
Typical Adult Therapy	Doxycycline 100 mg PO BID X 7 to 14 days OR Tetracycline 500 mg PO QID X 7 to 14 days
Typical Pediatric Therapy	Above age 8 years: Doxycycline 2 mg/kg PO BID X 7 to 14 days OR Tetracycline 500 mg PO QID X 7 to 14 days OR Rifampin 10 mg/kg/day PO
Clinical Hints	Fever, headache and myalgia following tick bite or exposure; arthralgia or macular rash may be present; leukopenia, thrombocytopenia or hepatic dysfunction common; inclusions in granulocytes; case-fatality rate 5%.
Synonyms	Anaplasma ovis, Anaplasma phagocytophilum, Anaplasmosis - human granulocytic, Ehrlichia equi, Ehrlichia ewingii, Ehrlichia microti, Ehrlichia phagocytophila, Ehrlichiosis - human granulocytic, Human granulocytic anaplasmosis, Human granulocytic ehrlichiosis. ICD9: 082.4 ICD10: B28.8

Clinical

Human Monocytic Ehrlichiosis (HME) and Anaplasmosis (Human Granulocytic Ehrlichiosis, HE) are characterized by fever, headache, myalgia, thrombocytopenia, leukopenia, and elevated liver enzyme levels. [1-3]
• A rash occurs in approximately one third of patients with HME but is less common in patients with HE.
• Rare instances of pneumonia have been reported.
• Most cases of anaplasmosis are mild; however, complications such as adult respiratory distress syndrome, renal failure, neurological disorders, sensorineural hearing loss [4] , and disseminated intravascular coagulation can occur. [5-8]
• Co-infection by *Borrelia burgdorferi* is common. [9]
• Case-fatality ratios in severe cases are as high as 5% for HME and 10% for Anaplasmosis.

The clinical features of anaplasmosis are similar to those of Lyme disease; however, patients with anaplasmosis are more likely to exhibit headache, dizziness, myalgias, abdominal pain, anorexia, leukopenia, lymphopenia, thrombocytopenia, and elevated liver enzymes. [10]

This disease is endemic or potentially endemic to 40 countries. Although Anaplasmosis is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Anaplasmosis in Brazil

Ehrlichia canis is responsible for most cases of ehrlichiosis among dogs in Brazil; however, *Ehrlichia ewingii* infection has been recently suspected in five dogs (2011 publication). [11]

Prevalence surveys:

0% of dogs with evidence of tick-borne infection (*Anaplasma phagocytophilum*, southeastern Brazil, 2007 publication) [12]
7.11% of dogs (*Anaplasma phagocytophilum*, Seropedica and Itagua, Rio de Janeiro State, 2011 publication) [13]
17.2% of Brazilian Brown Brocket Deer (*Mazama gouazoubira*) and Marsh Deer (*Blastocerus dichotomus*) (*Anaplasma* spp, Minas Gerais, 2011 publication) [14]

Anaplasma phagocytophilum has been detected in carnivorous birds. [15]

Seroprevalence surveys:

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20.2% of Brazilian marsh deer (*Blastocerus dichotomus*) prior to flooding of the Parana River (Mato Grosso do Sul, 2012 publication) [16]

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Angiostrongyliasis

Agent	PARASITE - Nematoda. Phasmidea: Angiostrongylus [Parastrongylus] cantonensis
Reservoir	Rat Prawn Frog
Vector	None
Vehicle	Snail Slug Prawn Lettuce
Incubation Period	2w (range 5d - 35d)
Diagnostic Tests	Identification of parasite. Serological tests have limited reliability.
Typical Adult Therapy	Corticosteroids if severe cns disease Mebendazole 100 mg BID X 5d; OR Albendazole (20 mg/kg/day)
Typical Pediatric Therapy	Corticosteroids if severe cns disease. Mebendazole 100 mg BID X 5d (age >2); OR Albendazole (20 mg/kg/day)
Clinical Hints	Eosinophilic meningitis or encephalitis - generally self-limited; absent or low grade fever; cranial nerve involvement (II, VI, V and VII); follows ingestion of slugs, snails, prawns or frogs.
Synonyms	Alicata's disease, Angiostrongylose, Angiostrongylus cantonensis, Bundibugyo, Eosinophilic meningitis, Haemostrongylus ratti, Panstrongyliasis, Parastrongyliasis, Parastrongylus cantonensis, Pulmonema cantonensis. ICD9: 128.8 ICD10: B83.2

Clinical

Angiostrongyliasis is characterized by severe headache, neck and back stiffness and paresthesias. [1-3]
• Bell's palsy occurs in 5 percent of patients; and disturbances of vision or eye movement in 15%.
• Low-grade fever may be present.
• Infection may present as meningitis, encephalitis, neuritis or ventriculitis [4]
• Progression of meningitis to encephalitis is more likely in elderly patients, and is associated with prolonged headache and fever >38 C. [5]
• Communicating hydrocephalus may develop during the course of infection. [6]
• Sudden death has been associated with infection of the fourth ventricle. [7]

The worm has been found in the CSF and the eye. [8-10]
• Eye infection manifests with generalized retinal pigment epithelial alteration, subretinal tracks, retinal edema, macular edema, and a pale disc. Visually-evoked potentials show secondary optic neuritis [11-13]
• Cerebrospinal fluid usually has a pleocytosis with 25 to 100 percent eosinophiles.
• Blood eosinophilia is present in most cases. [14]
• Rare instances of eosinophilic enteritis have been reported. [15]

CT studies may demonstrate pulmonary nodules and sup-pleural ground-glass opacities. [16]

The illness may last from a few days to several months.

Rare instances of *Ascaris suum* infection (discussed under 'Toxocariasis') in humans have been characterized by eosinophilic myelitis. [17]

This disease is endemic or potentially endemic to 43 countries.

Angiostrongyliasis in Brazil

Time and Place:
Angiostrongylus cantonensis appears to have been introduced into Brazil by rats on ships from Africa and Asia. [18]
- Following introduction into Brazil, *Achatina fulica* - the disease reservoir - became widely distributed. [19] [20]
- Brazil's first cases (3) of human infection were confirmed in 2007 in Cariacica, Espirito Santo. [21] [22]
- A fourth case of human infection was reported in Pernambuco in 2009.

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Prevalence surveys:

75% of Norway rats (*Rattus norvegicus*) in Rio de Janeiro (2010) [23]
10% of rats in Belem (2012 publication) [24]

Angiostrongylus cantonensis has been identified in the following snail species: *Sarasinula marginata*, *Subulina octona*, *Achatina fulica* , *Pomacea lineate* [25] [26] and *Bradybaena similaris* [27]
- *Angiostrongylus cantonensis* has been identified in *Achatina fulica* from Rio de Janeiro and Santa Catarina States. [28]

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Angiostrongyliasis - abdominal

Agent	PARASITE - Nematoda. Phasmidea: Parastrongylus [Angiostrongylus, Morerastrongylus] costaricensis
Reservoir	Cotton rat (Sigmodon) Slug
Vector	None
Vehicle	Slug Slug excretions
Incubation Period	10d - 14d
Diagnostic Tests	Identification of ova or adults in surgical material. Serologic tests used in some endemic areas.
Typical Adult Therapy	Mebendazole 200 to 400 mg PO tid X 10 days. OR Thiabendazole 25 mg/kg TID (max 3g/d) X 3d. Surgery for complications
Typical Pediatric Therapy	As for adult
Clinical Hints	Mimics acute appendicitis, including presence of a right lower quadrant mass: eosinophilia (uncommon in appendicitis) is prominent; patient may recall recent ingestion of slugs or vegetation contaminated by slugs.
Synonyms	Angiostrongylus costaricensis, Parastrongylus costaricensis. ICD9: 128.9 ICD10: B81.3

Clinical

Most patients have been children.

Clinical manifestations consist of abdominal pain, vomiting, fever, and a right lower quadrant mass (50% of cases). [1]
• The syndrome suggests appendicitis or heavy infection by Ascaris; however, the former may be discounted by the presence of eosinophilia.
• Radiographic findings are nonspecific and show filling defects and spasticity of the ileum, cecum, or colon.
• Complications include intestinal perforation or obstruction. [2] [3]
• Atopic worms have been recovered from the portal vein, hepatic artery [4] , testis and arteries of the limbs.
• Ova and parasites are not found in feces, and diagnosis is made using serological tests, or identification of parasites in surgical specimens.

This disease is endemic or potentially endemic to 24 countries.

Angiostrongyliasis - abdominal in Brazil

Time and Place:
- Most cases are reported from Sao Paulo, Parana, Santa Catarina [5] , Minas Gerais [6] , Federal District of Brasilia and Rio Grande do Sul [7]
- Seropositivity rates as high as 75% are reported in Coronel Vivida and Ilha Vermelha. [8]
- A single case has also been reported from Espirito Santo. [9]
- A series of 16 cases has been reported from Porto Alegre (2008 publication) [10]

Prevalence surveys:
3.6.6% of port areas (mollusk infection, 2012 publication) [11]
2.8% to 28.2% of the population of Guapore (southern region) (1995 to 1999). [12]

Reservoirs:
Mollusk hosts in southern Brazil include *Phyllocaulis variegatus* [13] , *Bradybaena similaris*, *Belocaulus angustipes*, *Doroceras laeve*, *Subulina octona* , *Sarasinula marginata* [14] , *Sarasinula lingaeformis* [15] and *Phyllocaulis soleiformis*. [16]
- Following introduction into Brazil, *Achatina fulica* - an additional potential disease reservoir - has become widely distributed. [17]

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Animal bite-associated infection

Agent	BACTERIUM. Pasteurella multocida, and other zoonotic bite pathogens
Reservoir	Cat Dog Marsupial (Tasmanian devil) Other mammal Rarely bird
Vector	None
Vehicle	Cat (60%), dog (30%) or other bite. No obvious source in 10%
Incubation Period	3h - 3d
Diagnostic Tests	Gram stain/culture. Hold specimen for 2 weeks to discount Capnocytophaga & other genera.
Typical Adult Therapy	Penicillin, a Tetracycline or Cefuroxime. Dosage and duration appropriate for nature and severity of infection
Typical Pediatric Therapy	Penicillin or Cefuroxime. Dosage and duration appropriate for nature and severity of infection
Clinical Hints	Infection of cat, dog or other bite wound - acquired during the preceding 3 to 72 hours (no history of bite in 10%); systemic infection (meninges, bone, lungs, joints, etc) may occur.
Synonyms	Bacteroides pyogenes, Bacteroides tectus, Bergeyella zoohelcum, Bisgaard's taxon 16, Capnocytophaga canimorsus, Capnocytophaga cynodegmi, CDC EF-4, CDC NO-1, Coryebacterium kutscheri, Corynebacterium canis, Corynebacterium freiburgense, Fusobacterium canifelinum, Halomonas venusta, Kingella potus, Moraxella canis, Neisseria animaloris, Neisseria canis, Neisseria weaveri, Neisseria zoodegmatis, Pasteurella caballi, Pasteurella canis, Pasteurella dagmatis, Pasteurella multocida, Pasteurella stomatis, Psychrobacter immobilis, Staphylococcus intermedius, Vibrio harveyi. ICD9: 027.2 ICD10: A28.0

Clinical

These are typically skin and soft infections which follow the bites of cats, dogs or other animals • usually during the preceding 3 to 72 hours. [1]
• There is no history of bite in ten percent of cases.
• Systemic infection (meninges [2] , bone, lungs [3] , joints, etc) may occur, with rare instance of severe septicemia. [4-7]

See the Microbiology module (Bacteria • Characterize) for a comprehensive discussion of bacterial species associated with bite wound infection in humans.

This disease is endemic or potentially endemic to all countries.

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Anisakiasis

Agent	PARASITE - Nematoda. Phasmidea: Anisakis simplex and Pseudoterranova decipiens
Reservoir	Marine mammals Fish
Vector	None
Vehicle	Undercooked fish
Incubation Period	Hours - 14d
Diagnostic Tests	Endoscopic identification of larvae.
Typical Adult Therapy	Endoscopic removal of larvae; surgery for complications
Typical Pediatric Therapy	As for adult
Clinical Hints	Allergic reactions; or acute and chronic abdominal pain, often with 'peritoneal signs' or hematemesis; follows ingestion of undercooked fish (e.g., sushi), squid or octopus.
Synonyms	Anasakis, Bolbosoma, Cod worm disease, Contracaecum, Eustrongylides, Herring worm disease, Pseudoterranova, Whaleworm. ICD9: 127.1 ICD10: B81.0

Clinical

The location of the worms and presenting features depend somewhat on the genus.
• *Phocanema* more commonly associated with infection of the stomach.
• *Anisakis* is usually associated with intestinal disease. [1]

Invasive anisakiasis:
Symptoms occur within 48 hours after ingestion.
• Gastric anisakiasis is characterized by intense abdominal pain, nausea, and vomiting. [2]
• Small intestinal involvement results in lower abdominal pain and signs of obstruction [3] , and may cause [4] or mimic appendicitis. [5] [6]
• Rare instances of duodenal ulcer have been reported. [7]
• Symptoms may last for months, rarely for years.
• The disease may also suggest tumor, regional enteritis or diverticulitis. [8]
• Rare instances of overt hemorrhage [9] and intussusception are reported. [10] [11]
• Patients may also experience a pharyngeal "tickling sensation", cough or a foreign body in the mouth or throat. [12]

Allergic anisakiasis:
Ingestion of *Anisakis* larvae with seafood is often responsible for acute allergic manifestations such as urticaria and anaphylaxis [13] , with or without accompanying gastrointestinal symptomatology. [14]
• Eosinophilia is usually not present in either gastric or intestinal anisakiasis; however, leukocytosis is noted in two thirds of patients with intestinal involvement.
• Urticaria is present in 20% of cases [15]

This disease is endemic or potentially endemic to all countries.

Anisakiasis in Brazil

Cases of human anisakiasis are reported. [16]

Prevalence surveys:
Nematode larvae of the Anisakidae family were detected in 64% of codfish sampled (Ribeirao Preto, 2007 publication) [17]

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Anthrax

Agent	BACTERIUM. Bacillus anthracis An aerobic gram positive bacillus
Reservoir	Soil Goat Cattle Sheep Water Horse
Vector	Fly (rare)
Vehicle	Hair Wool Hides Bone products Air Meat
Incubation Period	1d-7d; 1-12 cutaneous, 1-7 GI; 1-43 pulmonary
Diagnostic Tests	Bacteriological culture. Alert laboratory that organism may be present. Serology and rapid tests by Ref. Centers.
Typical Adult Therapy	Isolation (secretions). Ciprofloxacin; alt. Doxycycline, Penicillin G . Add Clindamycin + Rifampin for pulmonary infection. Dosage/route/duration as per severity
Typical Pediatric Therapy	Isolation (secretions). Ciprofloxacin (Doxycycline if age >= 8y). Add Clindamycin + Rifampin for pulmonary infection. Dosage/route/duration as per severity
Vaccine	Anthrax
Clinical Hints	Edematous skin ulcer covered by black eschar - satellite vesicles may be present; fulminant gastroenteritis or pneumonia; necrotizing stomatitis; hemorrhagic meningitis. Acquired from contact with large mammals or their products (meat, wool, hides, bone).
Synonyms	Antrace, Antrax, Antraz, Carbunco, Carbunculo, Malcharbon, Malignant pustule, Miltbrann, Miltvuur, Milzbrand, Mjaltbrand, Siberian plague, Siberian ulcer, Splenic fever, Wool-sorter's disease. ICD9: 022 ICD10: A22

Clinical

Most cases of anthrax occur in one of four forms: cutaneous, gastrointestinal, oropharyngeal and inhalational. [1]

CDC case definition for reporting:
As of 1996, the CDC (The United States Centers for Disease Control) case definition for reporting purposes consists of any illness with acute onset characterized by one or more of the following:
• cutaneous (a skin lesion evolving during a period of 2-6 days from a papule, through a vesicle to a depressed black eschar)
• pulmonary (hypoxia, dyspnea and mediastinal widening following a brief 'viral-type' prodrome)
• intestinal (severe abdominal distress followed by fever or signs of septicemia)
• oropharyngeal (mucosal lesion, cervical adenopathy and edema, and fever)
• demonstration of *Bacillus anthracis* by culture, immunofluorescence or serological response.

WHO case definition for surveillance:
The WHO Case definition for surveillance is as follows:
Clinical description:
An illness with acute onset characterized by several clinical forms. These are:
(a) localized form:
• cutaneous: skin lesion evolving over 1 to 6 days from a papular through a vesicular stage, to a depressed black eschar invariably accompanied by edema that may be mild to extensive
• systemic forms:
• gastro-intestinal: abdominal distress characterized by nausea, vomiting, anorexia and followed by fever
• pulmonary (inhalation): brief prodrome resembling acute viral respiratory illness, followed by rapid onset of hypoxia, dyspnea and high temperature, with X-ray evidence of mediastinal widening
• meningeal: acute onset of high fever possibly with convulsions, loss of consciousness, meningeal signs and symptoms; commonly noted in all systemic infections
Laboratory criteria for diagnosis
• isolation of *Bacillus anthracis* from a clinical specimen (e.g., blood, lesions, discharges)
• demonstration of *B. anthracis* in a clinical specimen by microscopic examination of stained smears (vesicular fluid, blood, cerebrospinal fluid, pleural fluid, stools)
• positive serology (ELISA, Western blot, toxin detection, chromatographic assay, fluorescent antibody test (FAT)
• Note: It may not be possible to demonstrate B. anthracis in clinical specimens if the patient has been treated with antimicrobial agents.
Case classification
• Suspected: A case that is compatible with the clinical description and has an epidemiological link to confirmed or suspected

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animal cases or contaminated animal products.
• Probable: A suspected case that has a positive reaction to allergic skin test (in non-vaccinated individuals).
Confirmed: A suspected case that is laboratory-confirmed.

Cutaneous anthrax:
• 95% of anthrax cases (worldwide) are cutaneous.
• The incubation period for cutaneous anthrax ranges from 12 hours to 12 days.
• Cutaneous anthrax begins with pruritus at the affected site, typically followed by a small, painless papule that progresses to a vesicle in 1 to 2 days. [2]
• The lesion erodes, leaving a necrotic ulcer with a characteristic black center.
• Secondary vesicles are sometimes observed.
• Lymphadenopathy may occur, and local edema may be extensive.
• Patients may have fever, malaise, and headache.
• The most common sites of cutaneous anthrax are the hands, forearms, and head.
• Anthrax related to illicit drug injection may present as subcutaneous infection rather than overt skin lesions. [3]
• Rarely infection may involve the genital area [4] , eyelid [5-7] or other areas.
• Cutaneous anthrax is fatal in approximately 20% of cases if left untreated.

Inhalational anthrax: [8] [9]
• Infection may progress to respiratory failure and shock within 1 to 2 days following onset of symptoms.
• The case-fatality rate exceeds 80%, even with appropriate antibiotic therapy. [10]
• Symptoms include pharyngeal pain, cough, fever and myalgia • followed by respiratory distress, cervical edema and venous engorgement suggestive of mediastintis. [11] [12]

Gastrointestinal anthrax: [13]
• Infection is characterized by pharyngeal pain, nausea, vomiting, and bloody diarrhea.
• Intestinal gangrene, obstruction and perforation may ensue. [14]
• The case-fatality rate for intestinal infection ranges from 25% to 60%.
• Ulcerative lesions, usually multiple and superficial, may occur in the stomach, sometimes in association with similar lesions of the esophagus and jejunum.
• Ulcers may bleed, and in severe cases the hemorrhage may be massive and fatal.
• Ascites may be present.
• Lesions in the mid-jejunum, terminal ileum, or cecum tend to develop around a single site or a few sites of ulceration and edema, similar to cutaneous anthrax.

Oropharyngeal anthrax:
• Infection is characterized by painful neck swelling and fever.
• The other common symptoms are sore throat, dysphagia, and hoarseness, enlargement of cervical lymph nodes and soft tissue edema.
• Oral lesions are located on the tonsils, posterior pharyngeal wall, or the hard palate. [15]
• In severe cases, the tonsillar lesions extended to involve the anterior and posterior pillars of fauces, as well as the soft palate and uvula.
• Early lesions are edematous and congested.
• By the end of the first week, central necrosis and ulceration produce a whitish patch, which evolves to a pseudomembrane which covers the ulcer after an additional week.

Meningeal anthrax: [16]
• Infection is characterized by fever, malaise, meningeal signs, hyperreflexia, and delirium, stupor, or coma. [17]
• CSF analyses demonstrated hemorrhagic meningitis, with positive Gram's stains and CSF cultures.
• 75% of patients die within 24 hours of presentation; mortality rates of 100% are reported in some series. [18] [19]
• Pathologic findings include hemorrhagic meningitis, multifocal subarachnoid and intraparenchymal hemorrhages, vasculitis, and cerebral edema. [20]

Published case-fatality rates are as follows: cutaneous <1%, gastrointestinal 25% to 60%, inhalational 46% and injectional 33%. [21]

This disease is endemic or potentially endemic to 147 countries.

Anthrax in Brazil

Anthrax, cases: None reported between 1988 and 1997
Notes:
1. Cases of human anthrax were published during the 1970's. [22]

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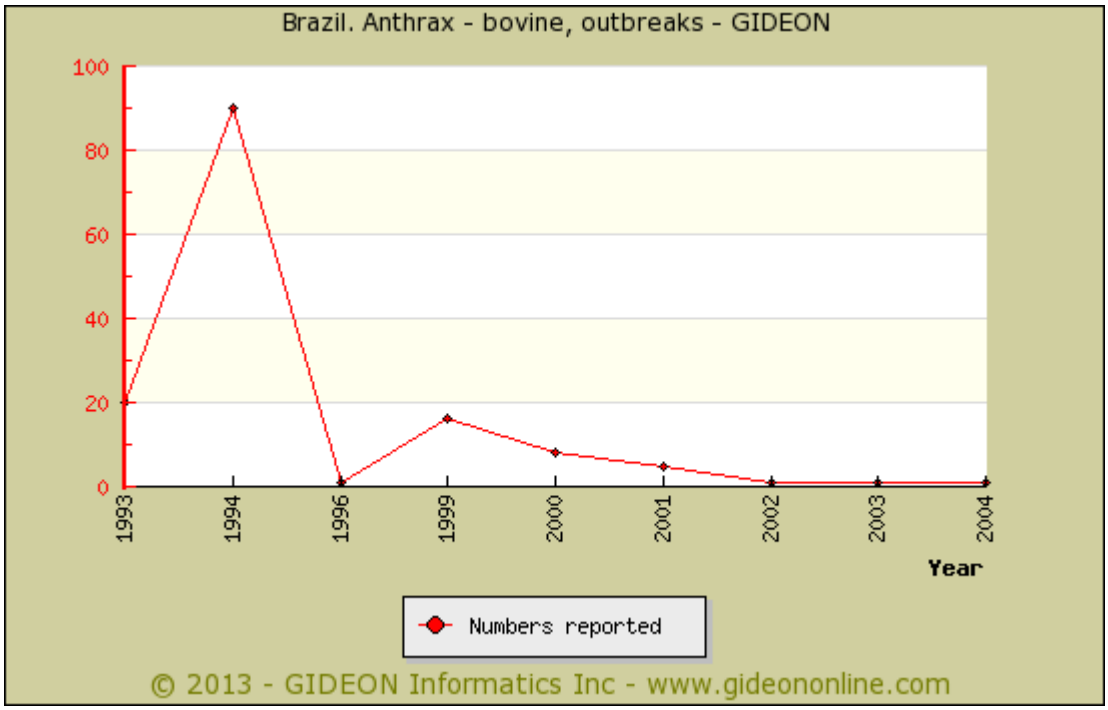
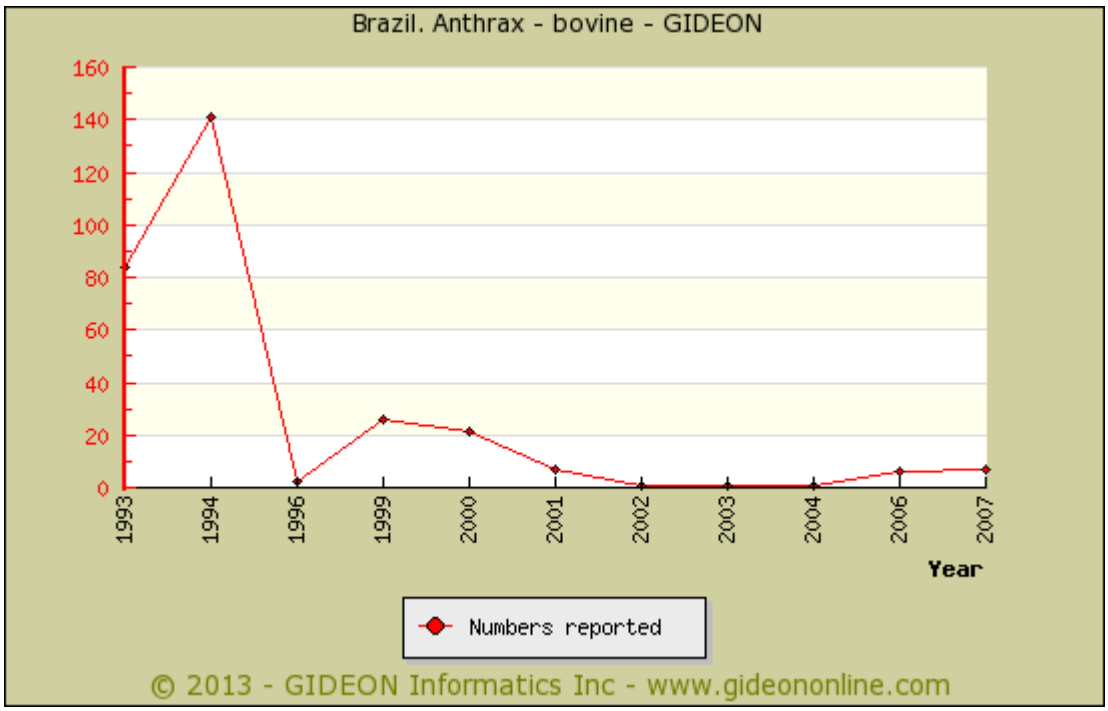


Graph: Brazil. Anthrax - bovine, outbreaks



Graph: Brazil. Anthrax - bovine

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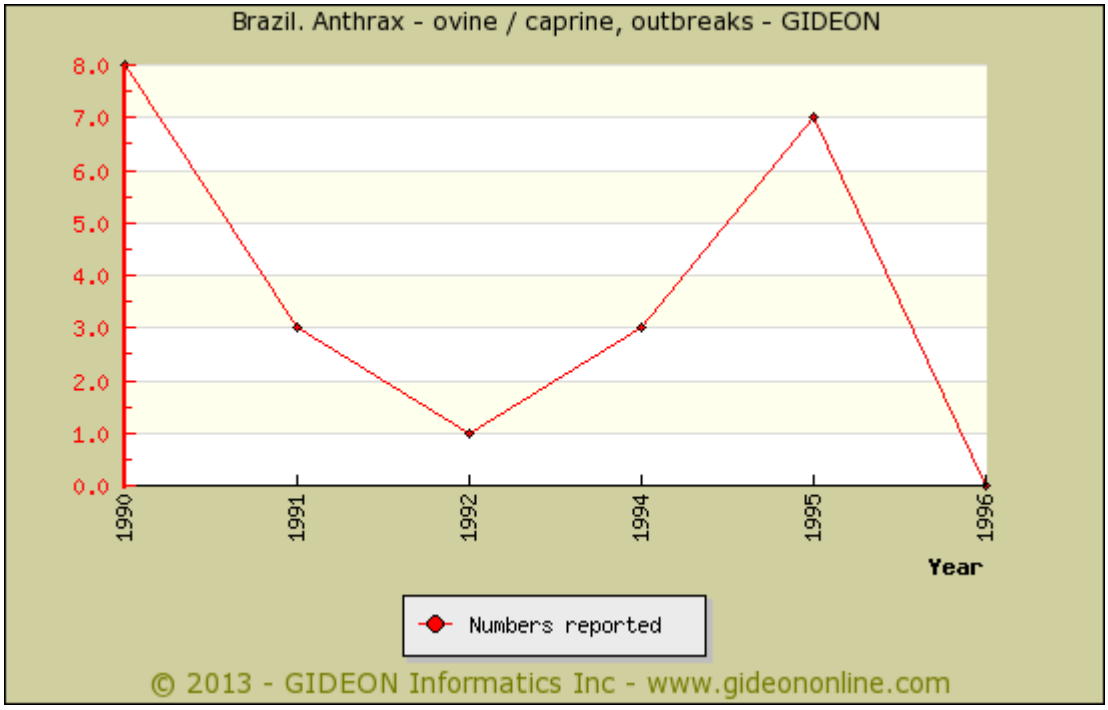
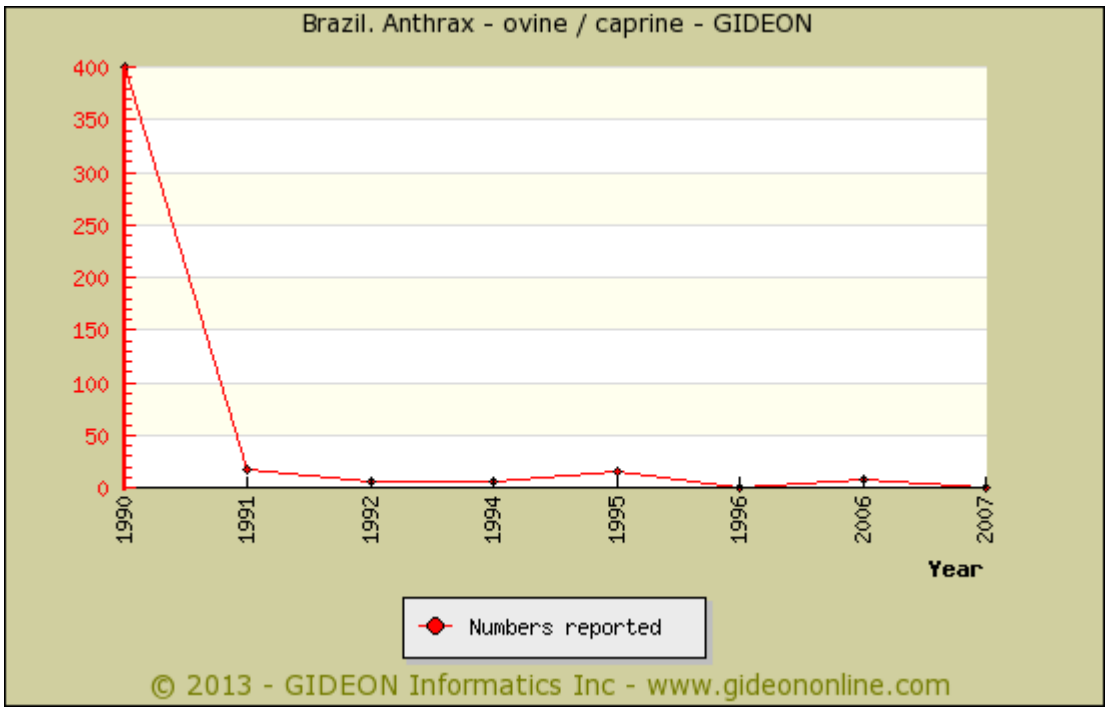


Graph: Brazil. Anthrax - ovine / caprine, outbreaks



Graph: Brazil. Anthrax - ovine / caprine

No cases of swine anthrax were reported in 1996.

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Ascariasis

Agent	PARASITE - Nematoda. Phasmidea: Ascaris lumbricoides
Reservoir	Human ? Dog
Vector	None
Vehicle	Vegetables Fly
Incubation Period	10d - 14d (range 7d - >200d)
Diagnostic Tests	Stool microscopy.
Typical Adult Therapy	Albendazole 400 mg X 1 dose OR Mebendazole 100 mg BID X 3d
Typical Pediatric Therapy	Mebendazole 100 mg BID X 3 d (> age 2).
Clinical Hints	An acute illness characterized by cough, wheezing and eosinophilia; adult worms are associated with abdominal pain (occasionally obstruction), pancreatic or biliary disease; highest rates among children and in areas of crowding and poor sanitation.
Synonyms	Ascaris, Ascaris lumbricoides, Askariasis. ICD9: 127.0 ICD10: B77

Clinical

The pulmonary manifestations of ascariasis occur during the stage of larval migration through the lungs and resemble Loffler's syndrome: cough, wheezing, pulmonary infiltration and eosinophilia. [1] [2]
• Children with heavy *Ascaris* infection experience impaired digestion and absorption of proteins, often with moderate steatorrhea.
• A mass of worms may block the lumen of the small bowel, resulting in acute intestinal obstruction, with vomiting, abdominal distention, cramps [3-6] • and occasionally hemorrhage [7] , gangrene or perforation. [8]
• Ileal volvulus and intussusception are also reported. [9]

Worms may also invade and obstruct the biliary duct (pancreatic-biliary ascariasis) [10-19] , producing abdominal pain, which may be associated with ascending cholangitis, acute or recurrent pancreatitis [20] [21] , pancreatic pseudotumor [22] or obstructive jaundice. [23-26]
• The majority of patients with hepatobiliary and pancreatic ascariasis present with biliary colic. [27]
• Choledocholithiasis, hepatolithiasis, liver abscess and cirrhosis are associated with the presence of dead, rather than viable worms. [28]
• Aberrant worms may appear at umbilical and hernial fistulas, the fallopian tubes, ovaries [29] , lower esophagus [30] , urinary bladder, pleural space [31] , trans-nasal [32] or trans-ostomy [33] feeding tubes, lungs, nose [34] , paranasal sinuses [35] and other sites.

Ascaris suum has been reported to cause rare cases of myelitis, eosinophilic pneumonia and focal liver lesions in humans, and is discussed under 'Toxocariasis.' [36-39]

This disease is endemic or potentially endemic to all countries.

Ascariasis in Brazil

Ova of *Ascaris* have been identified in 18th century human remains the area of Rio de Janeiro. [40]

Prevalence surveys:
31.2% of school children in Salvador (1997)
35% of school children in Lages (2004 publication) [41]
47% of school children in Caxias do Sul, RS (2008 publication) [42]
15.3% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [43]
14.4% of children in Uberlandia, Minas Gerais (2008 publication) [44]

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12.2% of children below age 14 in Caparao and Alto Caparao, Minas Gerais (1998) [45]
3.0% of children in public daycare centers in Belo Horizonte, Minas Gerais (2008 publication) [46]
12.3% of first grade school children in Campinas (1997 publication) [47]
28.7% of primary school children in Aracaju, Sergipe (1999 publication) [48]
71.43% of school children in Americanihnas, Minas Gerais (2008 publication) [49]
4.4% of Sao Paulo children below age 5 years (1995 to 1996) [50]
12.8% of urban children with diarrhea in Salvador (2000 to 2002) [51]
10.5% of children ages 3 to 6 years in day-care centers (Salvador, 2012 publication) [52]
2.5% of children in an urban slum in Sao Paulo (2008 publication) [53]
25.1% of children in northeastern Brazil. (2010 publication) [54]
27.5% of children ages 1 to 9 years in Rio de Janeiro State (2002 publication) [55]
26.1% of children ages 2 to 10 years in Pedregal, Campina Grande (2010 publication) [56]
63% of children in rural Bahia (2006 publication) [57]
24.4% of children ages 1 to 4, in 1998, 12.0% during 2003 to 2004 (Salvador) [58]
13.5% of children presenting to outpatient clinics in Manaus (2009 publication) [59]
4.2% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [60]
48.85% of homeless persons in Rio de Janeiro (2002 publication) [61]
35.6% of persons in Eirunepe, Amazon (2005 publication) [62]
1.4% of persons in Maria Helena, Parana (2004 to 2006) [63]
42.8% of the Parakana indigenous community in southwestern Para State (1992 to 1995) [64]
37.5% of Hupda and 19.3% of other indigenous peoples in Iauarete (2001) [65]
0.6% of Terena Indians (Mato Grosso do Sul) [66]
61.9% of persons in Sao Lourenco da Mata, rural Recife (Pernambuco, 1989) [67]
26% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [68]
48.8% of persons in Americaninhas, Minas Gerais (2004) [69]
50.8% of persons in Americaninhas, Minas Gerais (2004) [70]
14.9% of persons in Campos do Jordao (2003 to 2004) [71]
30.8% of patients with asthma in Bahia (2012 publication) [72]
15.6% of HIV-positive patients before HAART vs. 2% after HAART (Ceara, 2008 publication) [73]
2% of money bills acquired from shops (Rio Grande do Norte, 2007 publication) [74]
6.25% of public restrooms in Uberlandia, Minas Gerais (*Ascaris* and *Enterobius vermicularis*, 2009 publication) [75]
13% of beach sand samples from Alto beach, Pernambuco (2009 publication) [76]
2.1% of beach sand samples from Santos (*Ascaris* sp, 2004 to 2005) [77]

Ascaris spp. found in pigs and humans in Brazil share common haplotypes. [78]

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Aspergillosis

Agent	FUNGUS. Ascomycota, Euascomycetes, Eurotiales: Aspergillus. A hyaline hyphomycete
Reservoir	Compost Hay Cereal Soil
Vector	None
Vehicle	Air
Incubation Period	3d - 21d
Diagnostic Tests	Fungal culture. Biopsy. Nasal culture or serologic testing may be useful in select cases.
Typical Adult Therapy	Voriconazole 6 mg/kg IV Q12h, day 1; follow with 4 mg/kg IV OR Amphotericin B - if invasive, rapidly increase to max dose 0.6 mg/kg/d and to total 2.5g. OR Itraconazole
Typical Pediatric Therapy	Voriconazole 3 to 9 mg/kg IV Q12h OR Amphotericin B - if invasive, rapidly increase to max dose 0.6 mg/kg/d X 6w. OR Itraconazole
Clinical Hints	Pulmonary "fungus ball"; adult-onset asthma; consolidation or infected "pulmonary infarct" in setting of immune suppression (e.g., AIDS, leukemia, etc) leads to widespread hematogenous dissemination if not treated promptly.
Synonyms	Aspergillose, Aspergillus. ICD9: 117.3 ICD10: B44

Clinical

Clinical forms of aspergillosis include: [1] [2]
- allergy (allergic bronchopulmonary aspergillosis)
- colonization of air spaces (otomycosis, fungus ball or mycetoma of the paranasal sinuses [3] or lungs)
- non-pulmonary invasive (eye, sinuses, cardiac valve, skin, gastrointestinal tract) [4] [5]
- pulmonary-invasive

Invasion of the ears and sinuses can cause extensive necrosis in immunocompromised hosts.
- The most common central nervous system manifestations include brain abscess or cerebral infarction
- Meningitis is rare
- Endophthalmitis and keratitis usually occur following injury
- Wound infections and infection of vascular access sites has also been reported. [6]
- Sporadic instances of Isolated invasive *Aspergillus* tracheobronchitis [7] and chronic necrotizing pulmonary aspergillosis are encountered. [8]

Case-fatality rates range from 10% to 90%.
- One series of 289 cases cited a mortality rate of 40.2% (2008 publication) [9]

This disease is endemic or potentially endemic to all countries.

Aspergillosis in Brazil

As of 2005, an estimated 17,852 patients were living with chronic pulmonary aspergillosis secondary to tuberculosis, with 563 new cases occurring each year. [10]

Notable outbreaks:

2007 - An outbreak (6 cases) of invasive pulmonary aspergillosis was reported among patients hospitalized in a bone marrow transplant ward in Sao Paulo. [11]

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Babesiosis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea: Babesia microti, Babesia duncani (U.S.); or B. divergens, Babesia EU1 and B. bigemina (Europe)
Reservoir	Rodent (usually white-footed mouse = Peromyscus leucopus) Rabbit Deer Cattle Tick
Vector	Tick (Ixodes scapularis for Babesia microti; Ixodes ricinus for B. divergens)
Vehicle	Blood
Incubation Period	1w - 2w (range 1w - 9w)
Diagnostic Tests	Microscopy of stained blood smears. Animal inoculation. Serology. Nucleic acid amplification.
Typical Adult Therapy	Atovaquone 750 mg BID + Azithromycin 500 mg daily X 7 to 10 days. OR Clindamycin 600 mg PO TID + Quinine 650 mg PO TID X 7d. Exchange transfusion has been used in some cases
Typical Pediatric Therapy	Atovaquone 10 mg/kg BID + Azithromycin 12 mg/kg daily X 7 to 10 days. OR Clindamycin 13 mg/kg PO TID + Quinine 10 mg/kg TID X 7to 10 days.
Clinical Hints	Fever, rigors, myalgia, hepatomegaly and hemolysis; may relapse repeatedly; mimics malaria; severe disease among asplenic patients - jaundice, renal failure and death; European (*Babesia divergens*) - invariably in splenectomized patients & usually fatal.
Synonyms	Babesia, Babesia bigemina, Babesia bovis, Babesia divergens, Babesia duncani, Babesia EU1, Babesia microti, Babesiose, Colpodella. ICD9: 088.82 ICD10: B60.0

Clinical

Infection presents gradually
• Infection is characterized by malaise, fatigue, anorexia, shaking chills, fever, headache, myalgias, arthralgias, nausea, vomiting, abdominal pain, emotional lability, and dark urine. [1] [2]
• Photophobia, conjunctivitis, sore throat, and cough are also encountered.
• Adult respiratory distress syndrome, shock, petechiae, splinter hemorrhages, and ecchymoses

Hemolytic anemia and elevated reticulocyte counts are noted. [3]
• One to ten percent of erythrocytes are parasitized in most cases, but as many as 85% infection rates have been encountered in severe cases.
• The leukocyte count may be normal or decreased, and thrombocytopenia is common.
• Urinalysis reveals proteinuria and hemoglobinuria
• Blood urea nitrogen and serum creatinine levels may be elevated.
• Mild hepatic dysfunction [4] or splenic infarction have been reported. [5]
• Spontaneous splenic rupture has also been reported. [6] [7]

Immunocompromised patients are at risk for persistent relapsing babesiosis and require prolonged therapy [8]

Infection by a similar parasite in the Apicomplexa, *Colpodella*, has been reported in China. [9]
• Illness was characterized by relapsing fever, hemolytic anemia and the finding of intra-erythrocytic parasites.

This disease is endemic or potentially endemic to 45 countries. Although Babesiosis is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Babesiosis in Brazil

A Polish national acquired babesiosis while traveling in Brazil (1997 publication). [10]

Seroprevalence surveys:
99.2% of cattle in northeastern Para State are seropositive toward *Babesia bigemina* and 98.8% toward *B. bovis* (2008 publication) [11]

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Bacillary angiomatosis

Agent	BACTERIUM. Bartonella henselae or Bartonella quintana. Rickettsia-like bacteria
Reservoir	Human ? Tick ? Cat
Vector	Cat flea Tick (ixodid) - rare
Vehicle	None
Incubation Period	Unknown
Diagnostic Tests	Histology with special stains. Specialized culture techniques. Serology. Nucleic acid amplification.
Typical Adult Therapy	Clarithromycin 500 mg BID X 8 weeks Alternatives Azithromycin 250 mg QD or Ciprofloxacin 500 mg BID
Typical Pediatric Therapy	Clarithromycin 7.5 mg/kg PO BID X 8 months. OR Gentamicin 2 mg/kg IMq12h
Clinical Hints	Hemangiomatous papules and nodules of skin, spleen, liver (peliosis hepatis), bone or other tissues; virtually all in the setting of AIDS or other immune deficiency; rare instances following tick bite in immune-competent individuals.
Synonyms	Bacillary peliosis, Peliosis hepatis. ICD9: 757.32,083.8 ICD10: K76.4,A44.0

Clinical

Bacillary angiomatosis was originally described as involving skin and regional lymph nodes of HIV-infected persons. [1]
• Subsequent infections have involved patients with other forms of immune suppression, and presented in a variety of organs including liver, spleen, bone, brain, lung, bowel, and uterine cervix.

Cutaneous lesions often arise in crops and resemble the lesions of verruga peruana.
• Lesions may present as fixed or mobile subcutaneous or dermal nodules.
• Single or multiple dome-shaped, skin-colored, red or purple papules are also described, which may ulcerate and discharge serosanguinous fluid. [2] [3]
• Lesions can range in diameter from millimeters to centimeters, and may mimic pyogenic granuloma. [4]
• Regional lymph nodes are frequently enlarged in a variety of distributions.
• Involved organs contain multiple blood-filled cystic structures that range from microscopic to several millimeters in size.
• Bone disease may present as multiple osteolytic lesions.

This disease is endemic or potentially endemic to all countries.

Bacillary angiomatosis in Brazil

Sporadic cases are reported. [5]

13 cases were diagnosed in five AIDS centers in Rio de Janeiro during 1990 to 1997. [6]

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Bacillus cereus food poisoning

Agent	BACTERIUM. Bacillus cereus (toxin). An aerobic gram-positive bacillus
Reservoir	Soil Processed & dried foods
Vector	None
Vehicle	Food
Incubation Period	2h - 9h (range 1h - 24h)
Diagnostic Tests	No practical test available. Isolation of organism from suspect food.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Usually follows ingestion of rice or other vegetables; vomiting within 1 to 6 hours and/or diarrhea within 6 to 24 hours; no fecal leucocytes.
Synonyms	Bacillus cytotoxicus. ICD9: 005.89 ICD10: A05.4

Clinical

Two types of illness are caused by two distinct metabolites. [1]
• Diarrhea is caused by a large molecular weight protein.
• Vomiting is caused by a low molecular weight, heat-stable peptide. [2]

Symptoms of *B. cereus* diarrheal food poisoning mimic those of *Clostridium perfringens* food poisoning.
• Symptoms of the emetic form mimic *S. aureus* food poisoning. [3]

Diarrheal form:
The onset of watery diarrhea, abdominal cramps, and pain occurs 6 to 15 hours after consumption of contaminated food. [4]
• Nausea may accompany diarrhea, but vomiting (emesis) rarely occurs.
• Symptoms persist for 24 hours in most instances.

Emetic form:
The emetic type of food poisoning is characterized by nausea and vomiting within 0.5 to 6 h after consumption of contaminated foods.
• Occasionally, abdominal cramps and/or diarrhea may also occur.
• Duration of symptoms is generally less than 24 h.

Only two fatal cases had been reported to 2005. [5] [6] Illness was characterized by rhabdomyolysis and renal failure.
• A case of encephalopathy and hepatic failure • similar to Reye's syndrome • was related to *Bacillus cereus* food poisoning. [7]

• A case report of fatal *Bacillus cereus* food poisoning was published from Belgium in 2011. [8]

This disease is endemic or potentially endemic to all countries.

Bacillus cereus food poisoning in Brazil

Prevalence surveys:

56.7% of ready-to-drink ground roasted coffee (*Bacillus cereus*, 2012 publication) [9]
19% of air samples from restaurants in Vicosa, Minas Gerais State (2010 publication) [10]
38.3% of equipment and utensils used in university cafeterias (2011 publication) [11]

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Bacterial vaginosis

Agent	BACTERIUM. Gardnerella vaginalis (facultative gram-negative bacillus), Mobiluncus curtisii, Mobiluncus mulieris, Prevotella, et al
Reservoir	Human
Vector	None
Vehicle	Sexual contact - normal flora in 14% (girls) to 70% (women)
Incubation Period	Unknown
Diagnostic Tests	Identification of "clue cells" or positive KOH test in vaginal discharge. Culture.
Typical Adult Therapy	Metronidazole 500 mg BID X 7d OR Tinidazole 2 g PO daily X 3d OR Clindamycin 300 mg BID X 7d + intravaginal Clindamycin or Metronidazole ? Also treat sexual partner
Typical Pediatric Therapy	Metronidazole 7.5 mg/kg BID X 7d
Clinical Hints	Thin vaginal discharge - "fishy" odor when mixed with KOH; mild to moderate pruritus; occasionally urethritis in sexual partner.
Synonyms	Gardnerella, Gardnerella vaginalis, Mobiluncus. ICD9: 041.89,616,10,099.8 ICD10: N76.1

Clinical

The diagnosis of bacterial vaginosis required three of the following: [1-3]
1. A white, noninflammatory vaginal discharge or coating
2. The presence of clue cells [4]
3. A vaginal pH above 4.5
4. A fishy odor following addition of 10% KOH to the vaginal discharge (presumably due to liberated trimethylamine).

Note that routine culture is unnecessary.

Associated conditions:
Sequelae of bacterial vaginosis include preterm birth [5-7] and neonatal distress [8], low birth weight [9], chorioamnionitis, cervicitis [10], scalp abscess of the newborn, an increased risk of late miscarriage [11] and maternal infection. [12]
- Some studies have suggested a correlation between bacterial vaginosis and infertility. [13-19]
- Bacterial vaginosis may increase the risk for acquisition of HIV infection.
- Bacterial vaginosis may predispose to urinary tract infection [20] and endometritis. [21]

Gardnerella vaginalis has rarely been associated with balanitis, urethritis, urinary tract infections, asymptomatic bacteremia and infectious endocarditis in adult males. [22]

Cases of osteomyelitis, discitis and septic arthritis due to *Gardnerella vaginalis* have been reported. [23-26]

This disease is endemic or potentially endemic to all countries.

Bacterial vaginosis in Brazil

Prevalence surveys:
15% of women in rural Alagoas (2003 publication) [27]
21.3% of women ages 14 to 49 attending a primary health unit (2008 publication) [28]
6.75% of women in the Brazilian Public Health System, and 3.53% in the private sector (clue cells, 2004 to 2007) [29]
18.7% of women in Serra Pelada, Para State (2004) [30]
20.7% of pregnant women in Botucatu (2006 to 2008) [31]
21.6% of pregnant women in Botucatu (2010 publication) [32]
34.7% of women undergoing pre-term and 28.9% undergoing full-term labor (2012 publication) [33]
15% of female prisoners in Espirito Santo (2000 publication) [34]

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20% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (2007 publication) [35]
20% of sexually-active adolescents in Salvador, Bahia (2012 publication) [36]
51% of female CSW in Sao Paulo (2007 publication) [37]
26.6% of HIV-positive women (2008 publication) [38]

Vaginal *Gardnerella vaginalis* is present in 15.9% of women (Uberaba, 1998). [39]

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Balantidiasis

Agent	PARASITE - Protozoa. Ciliate (Ciliophora), Litostomatea: Balantidium coli
Reservoir	Pig Non-human primate Rodent
Vector	None
Vehicle	Water Food
Incubation Period	1d - 7d (range 1d - 60d)
Diagnostic Tests	Microscopy of stool or colonic aspirates.
Typical Adult Therapy	Tetracycline 500 mg QID X 10d. OR Metronidazole 750 mg TID X 5d. OR Iodoquinol 650 mg TID X 20d
Typical Pediatric Therapy	Age >= 8 years: Tetracycline 10 mg/kg QID (max 2g/d) X 10d. Age <8 yrs, Metronidazole 15 mg/kg TID X 5d; or Iodoquinol 13 mg/kg TID X 20d
Clinical Hints	Dysentery, often with vomiting; mimics intestinal amebiasis. The disease is most common in pig-raising areas. Symptoms last for one to four weeks, and may recur.
Synonyms	Balantidiose, Balantidiosis, Balantidium coli, Balantidosis, Balindosis, Ciliary dysentery. ICD9: 007.0 ICD10: A07.0

Clinical

Most cases are asymptomatic.
• Clinical manifestations, when present, include persistent diarrhea, occasionally dysentery, abdominal pain, and weight loss. [1]

Symptoms can be severe in debilitated individuals.
• *Balantidium* pneumonia has been reported in immune-compromised patients [2] and persons with occupational exposure. [3]

Diagnosis is based on detection of trophozoites in stool specimens or in tissue collected during endoscopy.
• Cysts are less frequently encountered.
• *Balantidium coli* is passed intermittently and once outside the colon is rapidly destroyed. Thus stool specimens should be collected repeatedly, and immediately examined or preserved.
• Rare cases of pulmonary infection [4] and osteomyelitis have been reported. [5]
• *Balantidium coli* has been identified in the urine. [6]

This disease is endemic or potentially endemic to 109 countries.

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Bartonellosis - cat borne

Agent	BACTERIUM. Afipia felis, Bartonella henselae, Bartonella clarridgeiae, et al. A facultative gram-negative coccobacillus
Reservoir	Cat Possibly tick
Vector	Flea (cat flea = Ctenocephalides)
Vehicle	Cat scratch Plant matter (thorn, etc)
Incubation Period	3d - 14d
Diagnostic Tests	Visualization of organisms on Warthin Starry stain. Culture. Serology. Nucleic acid amplification.
Typical Adult Therapy	Aspiration of nodes as necessary. Azithromycin 500 mg day 1, then 250 daily X 4 days Alternatives: Clarithromycin, Ciprofloxacin, Sulfamethoxazole/trimethoprim
Typical Pediatric Therapy	Aspiration of nodes as necessary. Azithromycin 10 mg/kg day 1, then 5 mg/kg daily X 4 days
Clinical Hints	Tender suppurative regional adenopathy following cat scratch (usually kitten); fever present in 25%. systemic infection (liver, brain, endocardium, bone, etc) occasionally encountered; most cases resolve within 6 weeks.
Synonyms	Afipia felis, Bartonella clarridgeiae, Bartonella henselae, Bartonella koehlerae, Cat scratch disease, Debre's syndrome, Foshay-Mollaret cat-scratch fever, Katszenkratz-Krankheit, Petzetakis' syndrome, SENLAT. ICD9: 078.3 ICD10: A28.1

Clinical

Clinical history:.
Approximately 90% of patients have a history of exposure to a cat.
• The disease has also been reported after exposure to squirrels, dogs, goats, thorns and barbed wire. [1]
• 75% of patients report a bite or scratch to the head, neck or upper limbs.
• Subclinical bacteremia is common among immuno-competent persons with animal and arthropod contact.

Symptoms:
Following an incubation period of 3 to 10 days, a small skin lesion appears consisting of a macule, papule, pustule or vesicle.
• Within 1 to 2 weeks, edema and tenderness of the regional lymph nodes appear.
• In some cases, the patient may present with Parinaud oculoglandular syndrome (conjunctival granuloma with suppurative preauricular adenitis), encephalopathy, erythema nodosum, thrombocytopenic purpura, arthritis, synovitis or pneumonia.

Signs:
Physical examination reveals involvement of a single node in 50% of cases.
• 30% have involvement of multiple sites, and 20% involvement of several nodes in the same region.
• Lymph nodes typically measure 1 to 5 cm.
• The majority of lesions regress over 2 to 6 months, but may last for as long as 2 years.
• Suppuration occurs in 10% of cases, and cellulitis is rare.
• Inguinal lymphadenopathy in cat-scratch disease may suggest a diagnosis of lymphogranuloma venereum. [2]

Additional findings:
One third of patients manifest fever, lasting 1 to 7 days; and some cases may present as Fever of Unknown Origin. [3]
• Malaise, fatigue, anorexia, vomiting, weight loss, headache, splenomegaly and pharyngitis are occasionally observed.
• 10.5% of patients have musculoskeletal manifestations [4] , including osteitis [5] and osteomyelitis [6-8]
• Rare features include a transient truncal maculopapular rash, encephalopathy [9] or encephalitis [10] with seizures, lethargy, coma, parotitis [11] , cranial or peripheral nerve involvement, facial nerve paresis, myelitis [12] , uveitis or neuroretinitis [13-25] , optic neuritis [26] with transient blindness, vitreal hemorrhage [27] , polyneuritis, radiculitis, Guillain-Barre syndrome [28] , disseminated visceral infection [29] [30] , osteomyelitis [31] [32] , endocarditis of native or prosthetic valves [33-38] or vascular prostheses [39] , hepatosplenomegaly with hepatic granulomata [40] , renal microabscesses [41] , erythema marginatum, erythema multiforme, erythema nodosum [42] and thrombocytopenic purpura. [43]
• Scalp eschar with neck lymphadenopathy (SENLAT) has been reported in some cases [44] , and could be confused with tularemia or infection by *Rickettsia slovaca* or *Rickettsia raoultii*. [45]

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• *B. henselae* accounts for 6.1% of bacterial species causing uveitis (2001 to 2007) [46]

29 cases of *Bartonella henselae* infection of solid-organ transplant recipients were reported to 2011 • many with disseminated disease. [47]

In one case, *Bartonella koehlerae* infection was associated with depression, anxiety, mood swings, severe headaches, muscle spasms, interphalangeal joint stiffness, decreased peripheral vision, diminished tactile sensation and hallucinations. [48]

This disease is endemic or potentially endemic to all countries.

Bartonellosis - cat borne in Brazil

Prevalence surveys:

4.5% of cats in Lao Luis, Maranhao (2012 publication) [49]
17.02% of cats (*Bartonella henselae*, Vale do Sinos, Rio Grande do Sul, 2010 publication) [50]
4.3% of cats in Jaboticabal, Sao Paulo (2012 publication) [51]

Seroprevalence surveys:

13.7% of healthy persons in Minas Gerais (2005 publication) [52]
38.4% in HIV positive individuals vs. 34% of controls (*Bartonella* species, Rio de Janeiro, 2010 publication) [53]
<2% of sick dogs in Botucatu positive for *Bartonella henselae* and *Bartonella vinsonii* subsp. *berkhoffii* (2007 publication) [54]
3% of sick dogs (Sao Paulo State, 2007 publication) [55]

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Bartonellosis - other systemic

Agent	BACTERIUM. Bartonella quintana, B. koehlerae, B. elizabethae, B. tamiae, B. washoensis, etc A fastidious gram-negative coccobacillus
Reservoir	Human Louse Rat Cat Dog Sheep
Vector	Louse (Pediculus) Flea - rare (Ctenocephalides, Pulex) Mite - rare (Dermanyssus)
Vehicle	Wound or eye contact with secretions/louse feces
Incubation Period	9d - 25d (range 4d - 35d)
Diagnostic Tests	Serology. Culture. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3 to 5 days (if endocarditis, add Gentamicin 3 mg/kg daily X 28 days) Alternatives: Clarithromycin, Azithromycin, Gentamicin, Fluoroquinolone (Levofloxacin, Trovafloxacin, Pefloxacin, Sparfloxacin or Moxifloxacin)
Typical Pediatric Therapy	Erythromycin 10 mg/kg PO QID X 3 to 5 days. OR Gentamicin 2 mg/kg IM q12h. Alternatives: Clarithromycin, Azithromycin
Clinical Hints	Headache, myalgias, shin pain, macular rash, splenomegaly; endocarditis & bacteremia seen; relapse common; often associated with poor hygiene & crowding.
Synonyms	Bartonella alsatica, Bartonella bovis, Bartonella capreoli, Bartonella doshiae, Bartonella elizabethae, Bartonella grahamii, Bartonella quintana, Bartonella rochalimae, Bartonella schoenbuchensis, Bartonella tamiae, Bartonella vinsonii, Bartonella vinsonii berkhoffii, Bartonella volans, Bartonella washoensis, Candidatus Bartonella mayotimonensis, Candidatus Bartonella melophagi, Candidatus Bartonella merieuxii, Candidatus Bartonella rochalimae, Five day fever, His-Werner disease, Meuse fever, Quintan fever, Quintana fever, Shank fever, Shin fever, Shinbone fever, Trench fever, Volhynian fever. ICD9: 083.1 ICD10: A44.0,A44.8,A79.0

Clinical

Infection is characterized by abrupt onset of headache, postorbital pain, conjunctivitis, leg and back pain, relapsing fevers, splenomegaly and an erythematous maculopapular rash on the chest, back and abdomen. [1]
• In 50% of cases, as many as 3 to 8 relapses occur.

Subclinical bacteremia is common among immuno-competent persons with animal and arthropod contact.

No fatalities have been reported in classic trench fever.

Bartonella quintana (formerly *Rochalimaea quintana*) and related bacteria may also produce bacillary angiomatosis (discussed separately in this module), bacteremia, endocarditis [2-5], myocarditis [6], uveitis [7] [8], neuroretinitis [9] or chronic lymphadenopathy.
• *Bartonella* species other than *B. henselae* account for 8.1% of bacterial uveitis (France, 2008 publication) [10]
• A single reported case of *Bartonella rochalimae* infection was characterized by fever, myalgia, headache and splenomegaly. [11]
• *Bartonella vinsonii* subsp *berkhoffii* genotype has been implicated in a case of epithelioid hemangioendothelioma. [12]

This disease is endemic or potentially endemic to all countries.

Bartonellosis - other systemic in Brazil

Seroprevalence surveys:

12.8% of healthy persons in Minas Gerais (*Bartonella quintana*) [13]
38.4% in HIV positive individuals vs. 34% of controls (*Bartonella* species, Rio de Janeiro, 2010 publication) [14]
7.6% of domestic dogs (*Bartonella* spp., 2012 publication) [15]
<2% of sick dogs in Botucatu positive for *Bartonella henselae* and *Bartonella vinsonii* subsp. *berkhoffii* (2007 publication) [16]

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Bertiella and Inermicapsifer

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Anoplocephalidae: Bertiella spp. and Inermicapsifer spp.
Reservoir	Rodent Non-human primate
Vector	None
Vehicle	Mite (ingestion)
Incubation Period	Unknown
Diagnostic Tests	Identification of ova or proglottids in stool.
Typical Adult Therapy	Not established
Typical Pediatric Therapy	As for adult
Clinical Hints	Abdominal pain, vomiting, diarrhea or constipation following contact with primates.
Synonyms	Bertiella, Bertiella, Bertiella mucronata, Bertiella studeri, Bertielliasis, Inermicapsifer. ICD9: 123.8 ICD10: B71.8

Clinical

The few cases reported have ranged from asymptomatic infection to moderate abdominal pain, vomiting and diarrhea. [1]
- Symptoms may be intermittent or continuous. [2]
- Adult tapeworms are know to live for at least two years.
- Diagnosis is based on finding worms, worm segments or ova in stool.

This disease is endemic or potentially endemic to 29 countries.

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Blastocystis hominis infection

Agent	PARASITE - Protozoa. Chromista, Bigyra, Blastocystea: Blastocystis hominis. [taxonomic status remains uncertain]
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Water
Incubation Period	Unknown
Diagnostic Tests	Stool microscopy. Nucleic acid amplification.
Typical Adult Therapy	Nitazoxanide 500 mg BID X 3 d. OR Metronidazole 750 mg TID X 10d. OR Iodoquinol 650 mg TID X 20 d. or Sulfamethoxazole/trimethoprim
Typical Pediatric Therapy	Nitazoxanide - Age 1 to 3 years: 5 ml (100 mg) PO Q12h X 3 days - Age 4 to 11 years: 10 mg (200 mg) PO Q12h X 3 days; OR Metronidazole 15 mg/kg/d X 10d. Sulfamethoxazole/trimethoprim
Clinical Hints	Diarrhea and flatulence; usually no fever; illness similar to giardiasis; increased risk among immune-suppressed patients; the exact role of this organism in disease is controversial.
Synonyms	Apoi, Blastocystiose, Blastocystis hominis, Zierdt-Garavelli disease. ICD9: 007.8 ICD10: A07.8

Clinical

[1] Symptoms ascribed to blastocystosis include leucocyte-negative diarrhea, nausea, pain [2] , flatulence and abdominal distention. [3] [4]

- Some reports suggest an association between urticaria and *Blastocystis* infection. [5-11]
- Symptoms usually last for 3 to 10 days, but may persist for weeks or months.
- *Blastocystis hominis* has also been implicated in the etiology of irritable bowel syndrome and urticaria [12-14] , and may contribute to the development of anemia among infected pregnant women. [15]

A search for alternative etiologies (including other infectious agents) should always be made in such patients. [16] [17]

This disease is endemic or potentially endemic to all countries.

Blastocystis hominis infection in Brazil

Prevalence surveys:

4.6% of the population in Araquara region, Sao Paulo (2008 publication) [18]
2.2% of persons in Campos do Jordao (2003 to 2004) [19]
1.4% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [20]
40.9% of Terena Indians (Mato Grosso do Sul, 2007 publication) [21]
21% of the Tapirape community in Mato Grosso (2010) [22]
57.8% of the Mapuera community in Oriximina, Para State (2009 publication) [23]
18% of hemodialysis patients (Parana, 2006 to 2007) [24]
28.2% of food handlers in Cascavel, Parana (2009 publication) [25]

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Botulism

Agent	BACTERIUM. Clostridium botulinum. An anaerobic gram-positive bacillus
Reservoir	Soil Animal Fish
Vector	None
Vehicle	Food Occasionally soil (wound contamination)
Incubation Period	1d - 2d
Diagnostic Tests	Electrophysiologic (EMG) pattern. Isolation of organism from food (occ. from infant stomach). Mouse toxin assay
Typical Adult Therapy	Heptavalent (types A-G) or trivalent (types A, B, E) antitoxin [following test dose] 10 ml in 100 ml saline over 30 min Additional 10 ml at 2 and 4 hours if necessary. Respiratory support
Typical Pediatric Therapy	As for adult
Vaccine	Botulism antitoxin
Clinical Hints	Clinical manifestations similar to those of atropine poisoning: dysarthria, diplopia, dilated pupils, dry mouth, constipation, flaccid paralysis, etc); onset approximately 36 hrs after ingestion of poorly-preserved food.
Synonyms	Botulisme, Botulismo, Botulismus, Kerner's disease. ICD9: 005.1 ICD10: A05.1

Clinical

For reporting purposes, the CDC (The United States Centers for Disease Control) case definitions for Foodborne, Infant and Wound Botulism are as follows:
• 1) Neurological syndrome (diplopia, blurred vision, bulbar weakness, symmetric paralysis); or
• 2) Infant exhibiting constipation, poor feeding and failure to thrive, followed by progressive weakness, impaired respiration and death. [1]

Symptoms and signs of botulism reflect characteristic electrophysiological abnormalities [2] and include diplopia [3] [4], blurred vision, ptosis, slurred speech, difficulty swallowing, dry mouth [5], and muscle weakness. Infants are lethargic, 'floppy,' constipated and feed poorly• exhibiting a weak cry and poor muscle tone. [6] [7]
• In food-borne botulism, symptoms generally begin 18 to 36 hours after ingestion (range 6 hours to 10 days). [8]
• Type F botulism is characterized by the appearance of respiratory failure within 24 hours, quadriplegia by the fifth day and rapid recovery beginning on the eighth day. [9] [10]
• A case of asymmetric cranial nerve demyelination due to type F botulism has been reported. [11]
• If untreated, these symptoms progress to paralysis of the arms, legs, trunk and respiratory muscles.
• Patients who experience nausea and vomiting, cranial neuropathy or urinary retention are most likely to develop respiratory failure. [12]
• Botulinum toxin may persist in the serum of patients for as long as 12 days. [13]

Infant botulism should be suspected if a previously healthy infant (age <12 months) develops constipation and weakness in sucking, swallowing, or crying; hypotonia; and progressive bulbar and extremity muscle weakness. [14]
• Approximately 50% of patients require mechanical ventilation.
• Lumbar puncture and brain imaging studies are usually normal, in contrast to other causes of flaccid weakness.
• The findings of infant botulism may mimic those of Hirschprung's disease [15] or acute abdomen. [16]

This disease is endemic or potentially endemic to all countries.

Botulism in Brazil

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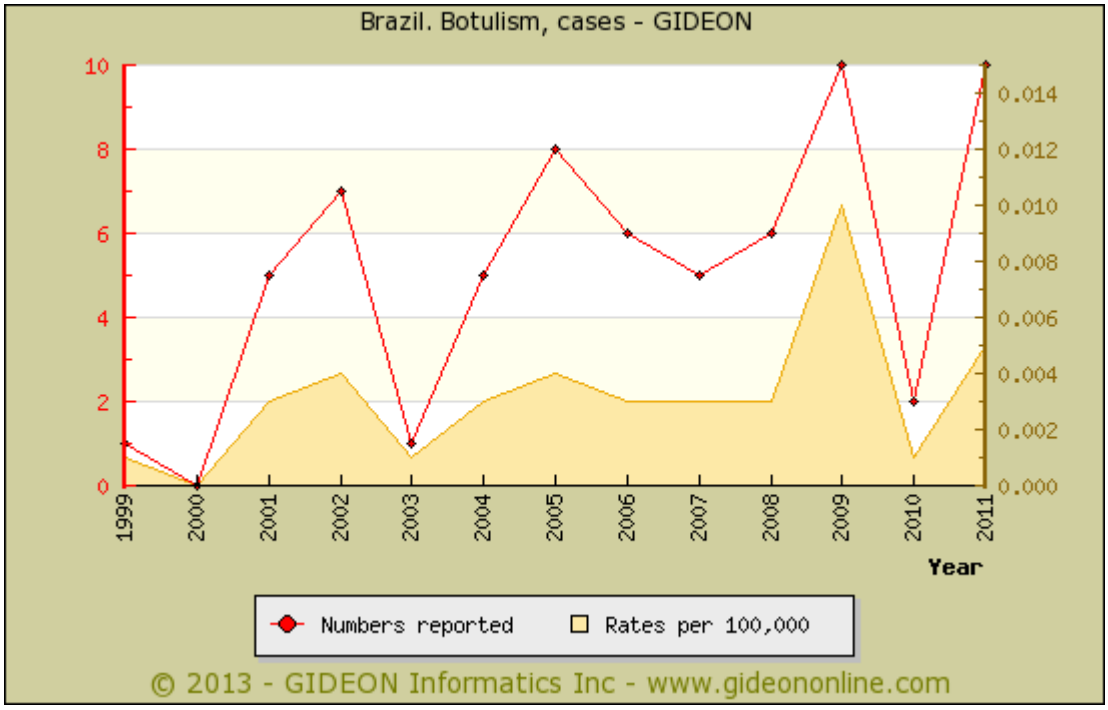
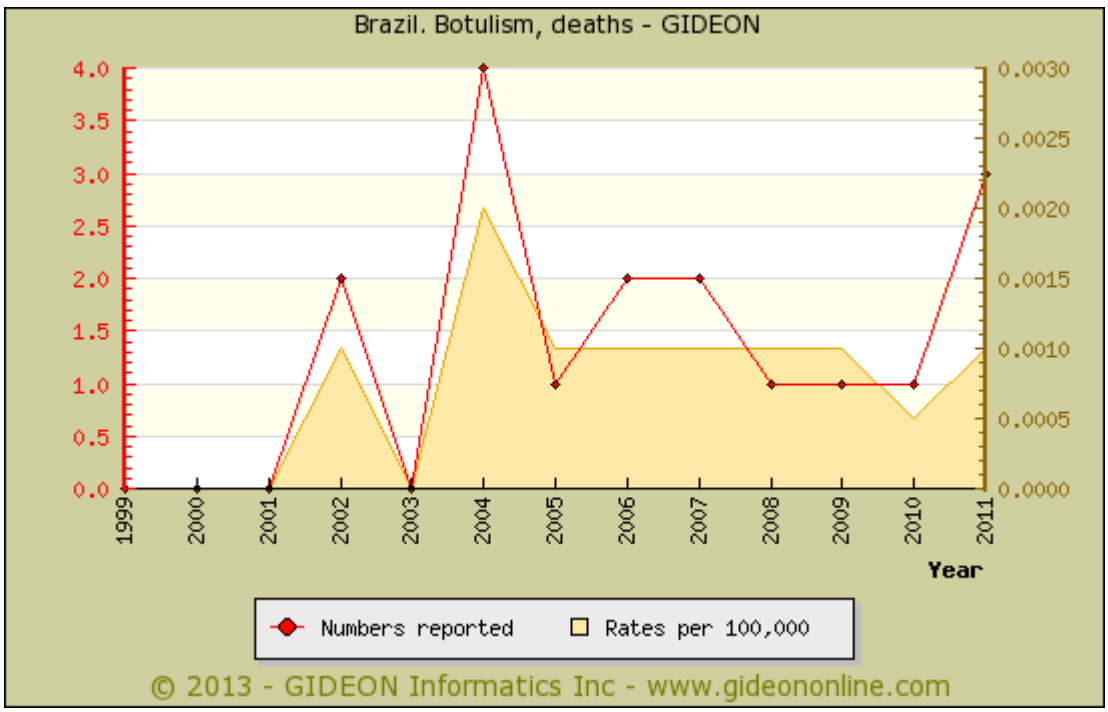


Graph: Brazil. Botulism, cases

117 cases of suspected botulism were reported during January 2000 to October 2008. [17]



Graph: Brazil. Botulism, deaths

Eight outbreaks of botulism were reported in Brazil during 1982 to 2001.
- Seven of these were due to type A botulism. [18]

Notable outbreaks:

1958 - An outbreak (6 fatal cases in a family) was caused by home-canned fish.

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1987 (publication year) - An outbreak (8 cases) was reported in Minas Gerais. [19]

2006 - An outbreak (3 cases, 1 fatal) among members of a family in Ceara was ascribed to contaminated chicken pie. [20] [21]

2009 - An outbreak (2 fatal cases) in Sao Paulo was related to canned jilo fruit. [22] [23]

2011 - An outbreak (7 cases) in Santa Catarina was associated with contaminated bologna sausage. [24]

2012 - An outbreak (4 cases, 2 fatal) in Parana was associated with contaminated sausage. [25]

2012 - An outbreak (4 cases) of suspected botulism in Sao Paulo was associated with mordatella (Italian cured sausage). [26]

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Brain abscess

Agent	BACTERIUM OR FUNGUS. Mixed oral anaerobes / streptococci, Staphylococcus aureus (from endocarditis), etc.
Reservoir	Human
Vector	None
Vehicle	None
Incubation Period	Variable
Diagnostic Tests	Imaging techniques (CT, scan, etc).
Typical Adult Therapy	Antibiotic(s) appropriate to likely pathogens + drainage
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, vomiting and focal neurological signs; often associated with chronic sinusitis or otitis media, pleural or heart valve infection; patients are often afebrile.
Synonyms	Ascesso cerebrale, Cerebral abscess. ICD9: 324.0 ICD10: G06.0

Clinical

The clinical presentation of brain abscess may range from indolent to fulminant. [1]
• Most manifestations are due to the size and location of this space-occupying lesion within the brain and the virulence of the infecting microorganism, and not to infection per se.
• Headache is observed in approximately 70% of patients and may be moderate to severe and unilateral or generalized.
• Sudden worsening of the headache, accompanied by meningismus, may herald rupture of the abscess into the ventricular space.
• Less than 50% of patients present with a classic triad of fever, headache, and focal neurological deficit.
• Mental status changes are seen in 70% of cases, fever in 45 to 50%, seizures in 25 to 35%, vomiting in 25 to 50%, nuchal rigidity in 25% and papilledema in 25%.

Metastatic infections are most often associated with endocarditis, and may present with multiple abscesses.
• Although the distribution of the middle cerebral artery is most often involved, any part of the brain may be infected.
• Common pathogens in this setting reflect the usual flora of endocarditis and bacteremia.

Etiological associations:
• Congenital heart disease: viridans streptococci, *Haemophilus* spp.
• Endocarditis: *Staphylococcus aureus*, streptococci
• Immunodeficiency: Toxoplasmosis, *Nocardia*, fungi
• Otitis: Peptostreptococci, streptococci, Enterobacteriaceae
• Pleuropulmonary infection: anaerobes, *Nocardia*
• Sinusitis: Streptococci, Enterobacteriaceae, *Bacteroides*, *Haemophilus influenzae*
• Traumatic or post-surgical: *Staphylococcus aureus*, streptococci, Enterobacteriaceae

This disease is endemic or potentially endemic to all countries.

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Brazilian hemorrhagic fever

Agent	VIRUS - RNA. Arenaviridae, Tacaribe complex, Arenavirus: Sabia virus
Reservoir	Rodent (presumed)
Vector	None
Vehicle	Excretions
Incubation Period	7d (precise data lacking)
Diagnostic Tests	Viral culture (blood, liver, spleen). Serology. Nucleic acid amplification. Biosafety level 4.
Typical Adult Therapy	Strict isolation; no therapy proven. Suggest IV Ribavirin 2g; then 1g q6h X 4d; then 0.5g q8h X 6d
Typical Pediatric Therapy	As for adult (Ribavirin dosage not established)
Clinical Hints	Fever, headache, myalgia, pharyngeal injection, azotemia and neutropenia; may progress to coma, seizures and gastrointestinal hemorrhage; case-fatality rate probably exceeds 33%.
Synonyms	Sabia. ICD9: 078.7 ICD10: A96.8

Clinical

The signs and symptoms are reminiscent of Argentine hemorrhagic fever (q.v.), with natural infection limited to Brazil. [1]

This disease is endemic or potentially endemic to 1 country.

Brazilian hemorrhagic fever in Brazil

To date, only three cases (one fatal) of Sabia have been described - two of these acquired in laboratories.

The first case (fatal) was reported in Sao Paulo, Brazil in 1990 - infection was thought to have originated in a community called 'Sabia.' [2]

Nonfatal infection occurred among University personnel in the United States who had been working with a patient specimen from Brazil. [3]

Nonfatal secondary infection has occurred among laboratory personnel working with patient specimens. [4]

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Brazilian purpuric fever

Agent	BACTERIUM. Haemophilus influenzae biogroup aegyptius A facultative gram-negative bacillus
Reservoir	Human
Vector	None
Vehicle	? Eye/respiratory secretions
Incubation Period	1w - 2w (range 1d - 60d)
Diagnostic Tests	Culture of blood, conjunctivae and skin lesions for Haemophilus.
Typical Adult Therapy	Isolation; intravenous antibiotic appropriate for Haemophilus influenzae
Typical Pediatric Therapy	Rifampin 10 mg/kg PO BID X 4 days may prevent spread from the conjunctivae
Clinical Hints	Fever, vomiting, abdominal pain, hypotension, purpura and thrombocytopenia one to two weeks following purulent conjunctivitis; case fatality rate over 75% - death often within 24 hours.
Synonyms	Febre purpurica do Brasil. ICD9: 041.5 ICD10: A48.4

Clinical

The CDC (The United States Centers for Disease Control) case-definition for BPF is as follows:
A febrile illness in a child with isolation of *H. aegyptius* from a normally sterile body site (e.g., blood, CSF).
OR

1. An acute illness in a child aged 3 months to 10 years characterized by:
   a. Fever of 38.5 C (101.3 F) or higher.
   b. Abdominal pain and/or vomiting.
   c. Development of petechiae and/or purpura.
   d. No evidence of meningitis.
2. History of conjunctivitis within the 30 days preceding the onset of fever.
3. At least one of the following two tests negative for *Neisseria meningitidis*:
   a. Blood cultures taken before antibiotic administration.
   b. Serum or urine antigen detection.
4. Other laboratory data if obtained:
   a. CSF containing 100 or fewer leukocytes per cu mm and negative culture or antigen detection for pathogenic bacteria other than *H. aegyptius*.
   b. Blood cultures taken before antibiotic administration negative for known pathogenic bacteria other than *H. aegyptius*.
   c. Serologic studies, if obtained, negative for known pathogens other than *H. aegyptius*.

A purulent conjunctivitis precedes BPF in 87% to 91% of cases. [1] [2]

This disease is endemic or potentially endemic to 2 countries.

Brazilian purpuric fever in Brazil

Brazilian purpuric fever (BPF) was recognized during 1984 in the town of Promissao (Sao Paulo State) when 10 children in a town of 20,000 persons died of an acute febrile illness associated with purpura and vascular collapse. [3-6]
- No cases were reported outside of Sao Paulo and Parana during 1984 to 1988.

17 cases were reported nationwide in 1984; 12 in early 1985; 13 in 1986.
- During 1989 to 1990, 10 definite (6 fatal) and 7 possible cases were confirmed in Mato Grosso. [7]
- The overall attack rate for the affected area was six per 100,000 children less than 10 years of age.
- No cases of BPF were reported during 1994 to 2006. [8]

Notable outbreaks:

2007 - An outbreak (7 cases, 5 fatal) of probable BPF was reported in Para State. [9]

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Brucellosis

Agent	BACTERIUM. Brucella abortus, Brucella melitensis, Brucella suis, Brucella canis An aerobic gram-negative bacillus
Reservoir	Pig Cattle Sheep Goat Dog Coyote Caribou
Vector	None
Vehicle	Food Air Dairy products Animal excretions
Incubation Period	10d - 14d (range 5d - 60d)
Diagnostic Tests	Culture of blood or bone marrow. Serology. Note: Alert laboratory to possibility of Brucella.
Typical Adult Therapy	Doxycycline 100 mg BID + Rifampin 600 mg BID X 6 weeks. Alternatives Tetracycline + Gentamicin
Typical Pediatric Therapy	Rifampin 20 mg/kg/day (maximum 600 mg) plus: >age 8 years: Doxycycline 2 mg/kg BID PO X 6w age < 8 years Sulfamethoxazole/trimethoprim 4/20 mg/kg BID X 4 to 6w Add Gentamicin if severe
Clinical Hints	Prolonged fever, hepatosplenomegaly, lymphadenopathy, arthritis, osteomyelitis or chronic multisystem infection following ingestion of unpasteurized dairy products, contact with farm animals or meat processing.
Synonyms	Bang's disease, Bangsche Krankheit, Brucella, Brucellemia, Brucelliasis, Brucellose, Brucellosen, Brucellosi, Brucelose, Brucelosis, Cyprus fever, Febris melitensis, Febris sudoralis, Febris undulans, Fievre caprine, Gibraltar fever, Goat fever, Malta fever, Maltafieber, Melitococcosis, Neapolitan fever, Rock fever, Typhomalarial fever, Undulant fever. ICD9: 023 ICD10: A23

Clinical

For surveillance purposes the CDC (The United States Centers for Disease Control) case definition of brucellosis consists of "an illness characterized by acute or insidious onset of ever, night sweats, undue fatigue, weight loss, headache and arthralgia" associated with epidemiological or laboratory evidence for infection.

WHO Case definition for surveillance:

The WHO Case definition for surveillance is as follows:
Clinical description
• An illness characterized by acute or insidious onset, with continued, intermittent or irregular fever of variable duration, profuse sweating particularly at night, fatigue, anorexia, weight loss, headache, arthralgia and generalized aching. Local infection of various organs may occur
Laboratory criteria for diagnosis
• Isolation of *Brucella* spp. from clinical specimen or
• *Brucella* agglutination titer (e.g., standard tube agglutination tests: SAT>160) in one or more serum specimens obtained after onset of symptoms or
• ELISA (IgA, IgG, IgM), 2-mercaptoethanol test, complement fixation test, Coombs, fluorescent antibody test (FAT), and radioimmunoassay for detecting antilipopolysaccharide antibodies; and counterimmunoelectrophoresis (CIEP)
Case classification
• Suspected: A case that is compatible with the clinical description and is epidemiologically linked to suspected or confirmed animal cases or contaminated animal products.
• Probable: A suspected case that has a positive Rose Bengal test.
• Confirmed: A suspected or probable case that is laboratory-confirmed.

Clinical manifestations: [1]

The clinical picture of brucellosis is nonspecific, and most often consists of fever, sweats, malaise, anorexia, headache, depression and back pain. [2] [3]

• The fever of brucellosis may mimic that of enteric fever [4] ; and an undulant fever pattern is seen in chronic infections.
• Fever may be absent among patients with end-stage renal disease who acquire brucellosis. [5]
• Mild lymphadenopathy is seen in 10 to 20% of patients; and splenomegaly or hepatomegaly in 20 to 30%. Rare instances of splenic rupture have been reported. [6]
• Bone and joint infections are common [7-10] , including a high rate of vertebral osteomyelitis. [11-14] Rare instances of acute or granulomatous myositis [15] , bursitis [16] and soft tissue or muscular abscesses have also been reported. [17-20] Most cases of brucellar monoarthritis represent reactive rather than septic disease. [21] Infection of natural [22] or prosthetic joints [23] [24]

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and soft tissue has been reported. [25]
• Vertebral osteomyelitis is characterized by osteolysis, often associated with paravertebral masses, spondylodiscitis [26] [27] , epidural abscess [28-30] , or psoas abscesses. [31-33]
• Epididymoorchitis is found in 7.6% to 12.7% of male patients with brucellosis. [34-40] Prostatitis has also been reported. [41]
• Endocarditis is well documented [42-51] , including isolated case reports of *Brucella* infection of prosthetic valves [52] [53] and devices such as implantable defibrillators [54] and pacemaker leads. [55] Rare instances of aortitis [56] [57] , arterial thrombosis [58] , myocarditis [59] and pericarditis are also reported. [60-64]
• Pulmonary infiltrates [65-67] , pleural effusion [68] , ileitis [69] , chest wall infection [70] , cholestatic jaundice [71] , acalculous cholecystitis [72] , peritonitis associated with dialysis [73] , and liver abscess have been reported. [74] [75]
• Ocular manifestations include uveitis, visual loss due to suprasellar mass [76] , keratitis, conjunctivitis, papillitis, retinal hemorrhages and third-nerve palsy. [77] [78]
• Neurological manifestations may include encephalitis [79] , meningitis [80-84] , cranial [85] or peripheral neuropathy [86] , progressive paraparesis [87] or Guillain-Barre syndrome [88] , cerebral vasculitis with infarct [89] , intracranial hypertension or hydrocephalus [90] [91] , psychosis [92] , and parenchymal granulomata [93] or abscesses. [94-101]
• Renal infection may present at hematuria, proteinuria, pyuria, overt nephritis or renal failure. [102] Rare instances of glomerulonephritis have also been reported. [103-105]
• Persons working with animals may present with severe pharyngitis as an initial feature of brucellosis. [106]
• Abscesses involving a variety of body areas and solid organs may occur [107-111]
• Various forms of rash occur in 6% to 13% of patients including generalized or localized papules or macules [112] , ulcers, purpura, vasculitis / leukocytoclastic vasculitis [113] , panniculitis [114] and erythema nodosum [115] [116]
• Brucellosis has been implicated in cases of human abortion. [117] [118]

Virtually any organ or body system may be infected during the course of illness [119-130]
• Chronic brucellosis generally represents persistence of local infection in bone, joints, liver, spleen or kidneys.
• Relapses are common, especially following inadequate therapy.
• Pancytopenia is reported in 15% of cases [131] [132]
• Brucellosis has been reported to cause myelofibrosis [133] , and to trigger hemolytic anemia in patients with Glucose-6-Phosphate Dehydrogenase deficiency. [134]
• Isolated thrombocytopenia mimicking ITP is reported in 6% of cases [135-140] ; hepatic dysfunction [141] , colitis [142] Coombs-positive hemolytic anemia [143] [144] , reactive hemophagocytic [145] [146] or myelodysplastic syndrome [147] , pancytopenia [148] , disseminated intravascular coagulation [149] , TTP [150] [151] , Guillain-Barre syndrome [152] and syndrome of inappropriate secretion of antidiuretic hormone (SIADH) have also been documented. [153]

This disease is endemic or potentially endemic to 179 countries.

Brucellosis in Brazil

Brazil reported its first case of human brucellosis in 1993.

Human disease in this country is due to *Brucella abortus*, *B. melitensis* , *B. suis* [154] and *B. canis* [155]

Prevalence surveys:

10.20% of cattle in northern Brazil and 0.06% in southern Brazil (2012 publication) [156]
50.7% of female dogs with reproductive problems (*Brucella canis*, Sao Paulo, 2007 publication) [157]
31% of semen samples from bulls (DNA, 2012 publication) [158]

Seroprevalence surveys:

13% / 4.6% vs. *Brucella abortus* / *Brucella canis*, of persons in a poor urban community in Salvador (2012 publication) [159]

0.66% of slaughterhouse workers in northern Para (2006 publication) [160]
2.5% of cattle and 11.4% of cattle herds in Maranhao (2007 to 2009) [161]
0.7% of goats and sheep in Rio de Janeiro (all positives were goats, 2011 publication) [162]
0% of free-ranging Brazilian tapirs (*Tapirus terrestris*) in Goias state (2010 publication) [163]

Brucella abortus is found in cattle and water buffalo. [164]
- *Brucella suis* infection has been identified in equines [165] and cattle.

Notable outbreaks:

2006 - An outbreak of *Brucella suis* infection among swine in Sao Paulo was associated with one human case. [166]

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Bunyaviridae infections - misc.

Agent	VIRUS - RNA. Bunyaviridae, Orthobunyavirus. Over 30 strains have been associated with human disease (see Synonyms)
Reservoir	Rat Bird Marsupial Chipmunk Cattle Sheep Horse Bat
Vector	Mosquito (exceptions: Shuni is transmitted by culicoid flies; Bhanja, Tamdy, Wanowrie and Zirqa by ticks)
Vehicle	None
Incubation Period	3d - 12d
Diagnostic Tests	Serology and virus isolation. Nucleic acid amplification. Biosafety level 2 or 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Abrupt onset of fever, chills, headache; photophobia, rash arthralgia, myalgia, vomiting, diarrhea or cough may be present; meningitis or myocarditis may occur with Bwamba virus; usual course 2 to 7 days.
Synonyms	Bangui, Batai, Bhanja, Bunyamwera, Bwamba, Cache Valley, Calovo, Catu, Fort Sherman, Garissa, Germiston, Guama, Hartland virus, HNF virus, Huaiyangshan virus, Ilesha, Ingwavuma, Kairi, Lumbo, Ngari, Northway, Nyando, Pongola, Severe fever with thrombocytopenia syndrome, SFTSV, Shokwe, Shuni, Tacaiuma, Tamdy, Tataguine, Tensaw, Wanowrie, Wyeomyia, Zirqa. ICD9: 066.3 ICD10: A93.8

Clinical

As a group, these diseases are characterized by acute febrile illness occurring in persons exposed to wild or forest environments.
• Additional features may include headache, myalgia, arthralgia, rash or aseptic meningitis.

Avalon virus has been implicated in isolated cases of conjunctivitis [1] and polyradiculitis. [2] [3]

Ilesha virus infection may be associated with fever or rash, or hemorrhagic fever.

Huaiyangshan virus (Severe fever with thrombocytopenia syndrome bunyavirus, SFTSV) appears to infect primarily farmers, above age 50. [4-7]
• Contact with the blood of patients appears to facilitate spread of the disease. [8]
• Clinical features include fever, conjunctivitis, diarrhea, abdominal pain, thrombocytopenia, leukopenia, proteinuria and hematuria. [9]
• In one series, 8 of 49 hospitalized infections (16.3%) were fatal. [10]

A novel Bunyavirus ("heartland virus") has been associated with cases of fever, fatigue, diarrhea, thrombocytopenia, and leukopenia • following tick bites. [11] [12]

This disease is endemic or potentially endemic to 90 countries.

Bunyaviridae infections - misc. in Brazil

Seroprevalence surveys:

15.7% of horses in the Brazilian Pantanal (Tacaiuma virus, 1993 publication) [13]

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California encephalitis group

Agent	VIRUS - RNA. Bunyaviridae, Orthobunyavirus: La Crosse, California encephalitis, Jamestown Canyon, et al.
Reservoir	Chipmunk Squirrel Rodent Fox
Vector	Mosquito (Aedes, Anopheles triseriatus, Culex, Psorophora)
Vehicle	None
Incubation Period	5d - 15d
Diagnostic Tests	Viral culture (CSF, brain, blood). Serology. Nucleic acid amplification. Biosafety level 2.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Late summer flu-like illness, meningitis or encephalitis in wooded areas; seizures and paralysis common; polymorphonuclear leucocytosis.
Synonyms	California encephalitis, Guaroa, Inkoo, Jamestown Canyon, Keystone, Khatanga, La Crosse, Snowshoe hare, Tahyna, Trivittatus, Valtice fever. ICD9: 062.5 ICD10: A83.5

Clinical

Infection by the California encephalitis group of viruses is usually subclinical or limited to a mild febrile illness. [1]

Over 90% of overt LaCrosse virus meningoencephalitis occurs in children below the age of 15 years, with a predominance among males.

- Fever, headache, and vomiting progress to lethargy, aphasia, incoordination, focal motor abnormalities or paralysis. [2]
- Seizures occur in 50% of cases.
- The cerebrospinal fluid generally contains fewer than 100 leukocytes per cu mm.
- Peripheral leukocytosis in excess of 15,000 per cu mm is common.

Following recovery, abnormal electroencephalographic findings persist for 1 to 5 years in 75% of cases, and epilepsy develops in 10%.

This disease is endemic or potentially endemic to 16 countries.

California encephalitis group in Brazil

There is evidence for circulation of Guaroa virus in this country. [3]

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Campylobacteriosis

Agent	BACTERIUM. Campylobacter jejuni subsp jejuni, et al A microaerophilic gram-negative bacillus
Reservoir	Human Mammal Bird
Vector	None
Vehicle	Water Food
Incubation Period	2d - 4d (range 1d - 10d)
Diagnostic Tests	Stool (rarely blood, CSF) culture. Nucleic acid amplification. Alert laboratory when these organisms are suspected.
Typical Adult Therapy	Stool precautions. Erythromycin 500 mg QID X 7d. Alternatives Azithromycin, Fluoroquinolone (Levofloxacin, Trovafloxacin, Pefloxacin, Sparfloxacin or Moxifloxacin), Gentamicin
Typical Pediatric Therapy	Stool precautions. Erythromycin 12.5 mg/kg PO QID X 7d. Alternatives - Azithromycin, Gentamicin
Clinical Hints	Febrile diarrhea or dysentery; vomiting or bloody stool often noted; severe abdominal pain may mimic appendicitis; disease is most common among children and lasts one to four days.
Synonyms	Campylobacter. ICD9: 008.43 ICD10: A04.5

Clinical

Following an incubation period of 1 to 10 days, patients develop diarrhea (often bloody) and abdominal pain.
- Initial symptoms of malaise, dizziness, fever, headache and myalgia are common.
- Vomiting is unusual.
- Leucocytes are usually seen on stool smears.

Infection may be complicated by cholecystitis [1] , pseudoappendicitis, peritonitis [2] (including peritonitis associated with dialysis [3] [4]), hemolytic-uremic syndrome, bacteremia [5-7] , myocarditis [8-11] , endocarditis [12] , pericarditis [13] [14] , pleurisy [15] [16] , mycotic iliac [17] and aortic aneurysms [18] , meningitis [19] , encephalopathy [20] , epidural abscess [21] [22] , septic arthritis [23] , cellulitis [24] , Sweet's syndrome [25] , spontaneous abortion, reactive arthritis or Guillain-Barre syndrome.
- Reactive arthritis has been reported in 1% to 13% of cases [26] [27]
- The risk for reactive arthritis following *Campylobacter* infection was 2.1/100,000 cases (United States, 2002 to 2004) [28]
- Elderly patients are at risk for complicated or fatal infection. [29]

Guillain Barre syndrome (GBS) has been estimated to complicate 0.1% of *Campylobacter* infections. [30-34]
- *Campylobacter* infection is implicated in 14% to 40% of GBS episodes. [35-40]
- Risk for GBS continues for up to 2 months following an episode of Campylobacteriosis.
- The rate of GBS is 19.2 per 100,000 episodes of Campylobacteriosis. [41]
- There have been case reports of brain stem encephalitis [42] , cranial neuropathy [43] , acute transverse myelitis [44] and demyelization of the central nervous system or spinal cord following *C. jejuni* infection. [45]
- There is evidence that campylobacteriosis may increase the risk for later development of inflammatory bowel disease. [46]

This disease is endemic or potentially endemic to all countries.

Campylobacteriosis in Brazil

Prevalence surveys:

5.6% of urban children with diarrhea, in Salvador (2000 to 2002) [47]

9.9% of diarrhea among children below age 5 years in Rio de Janeiro (1993 publication) [48]

16.4% (*Campylobacter jejuni*) and 1.45 (*C. coli*) of children ages ? 14 years attending emergency rooms in Northeastern Brazil (2011 publication) [49]

7.5% of childhood diarrheal disease in northeastern Brazil (1983 publication) [50]

9.6% of children with diarrhea and 1.2% without diarrhea, in Northeastern Brazil (*Campylobacter jejuni*) [51]

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2.4% of children with diarrhea in northeastern Brazil (2008 publication) [52]

7.5% of infant diarrhea requiring hospitalization in Sao Paulo (1997 publication) [53]

16.6% of poultry samples at a processing plant (southern Brazil, 2005 publication) [54]

Campylobacter jejuni was found in 50.0% of poultry samples exported to Korea, and *Campylobacter coli* in 8.9% (2004 to 2008) [55]

22% of fecal samples from chicken abattoirs (Sao Paulo, 2006 publication) [56]

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Candidiasis

Agent	FUNGUS - Yeast. Ascomycota, Hemiascomycetes, Saccharomycetales. Candida albicans, and other species.
Reservoir	Human
Vector	None
Vehicle	Contact Catheter
Incubation Period	Variable
Diagnostic Tests	Culture. Serology and assays for cell-specific antigens are performed in some centers,
Typical Adult Therapy	Topical, oral, systemic antifungal agent depending on clinical presentation and species [in Therapy module, scroll through upper left box]
Typical Pediatric Therapy	As for adult
Clinical Hints	Dermal erythema with satellite pustules; "cheesy" mucosal discharge; severe, widespread or intractable disease should suggest the possibility of underlying diabetes, AIDS or other form of immune suppression.
Synonyms	Candida, Candida-Mykosen, Candidiase, Candidiasi, Candidose, Monilia, Moniliasis, Salmonella, Thrush. ICD9: 112 ICD10: B37

Clinical

The clinical features of candidiasis range from localized mucosal or skin inflammation to multi-organ candidal sepsis.

Often infection represents overgrowth of *Candida* species following use of antimicrobial agents, or in the presence of the high mucosal glucose concentrations found in diabetics.
• Other predisposing factors include chronic intertrigo, oral contraceptive use, and cellular immune deficiency.
• Candidiasis is a common initial event in HIV-infected individuals.
• White exudative plaques may occur on the tongue or buccal mucosa (thrush), vaginal or rectal mucosa.
• Fissured, macerated lesions at the corners of the mouth (perleche) are common among individuals with poorly-fitting dentures. In fact, candidal infections have a predilection for sites that are chronically wet and macerated.
• Intertriginous lesions are edematous, erythematous, and scaly; and associated with scattered "satellite pustules." [1]
• The glans penis and scrotum as inner aspect of the thighs are often involved.

Systemic *Candida* infections may involve virtually any organ or organ system, and mimic bacterial sepsis. [2] [3]
• Case fatality rates for infected vascular catheters range from 26% to 38%; 33% for infected prosthetic cardiac valves; 20% to 40% for urinary catheters.

This disease is endemic or potentially endemic to all countries.

Candidiasis in Brazil

The rate of candidemia in Brazil is 2.49 cases per 1,000 hospital admissions (0.37 per 1,000 patient-days), with a case-fatality rate of 54% (2003 to 2004). [4]
- The rate of candidemia in Sao Paulo is 1.66 cases per 1,000 hospital admissions (2007 publication) [5]

Prevalence surveys:

12.5% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (vaginal infection, 2007 publication) [6]
22% of sexually-active adolescents in Salvador, Bahia (vaginal infection, 2012 publication) [7]
9.3% of women ages 14 to 49 attending a primary health unit (vaginal infection, 2008 publication) [8]
47.9% of women with suspected candidiasis and 21.8% of women without suspected candidiasis (Ilheus, 2005 to 2007) [9]
5.7% of women in Serra Pelada, Para State (vaginal infection, 2004) [10]
11.8% of pregnant women in Botucatu (2006 to 2008) [11]
10.2% of pregnant women in Botucatu (2010 publication) [12]

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20.4% of women undergoing pre-term and 28.9% undergoing full-term labor (vaginal infection, 2012 publication) [13]
5.1% of female CSW in Sao Paulo (vaginal infection, 2007 publication) [14]
29.7% of HIV-positive women (vaginal infection, 2008 publication) [15]
37.0% of HIV-positive patients receiving HAART (oral candidiasis, 1997 to 2004) [16]
65.8% of patients with lepromatous leprosy, vs. 47.4% of controls, in Sao Paulo (2009 publication) [17]
30.2% of deaths among AIDS patients (systemic infection, 1996 to 2006) [18]

Notable outbreaks:

1998 (publication year) - An outbreak (6 cases) of *Candida parapsilosis* fungemia was reported among hematology, bone marrow transplant, and oncology patients in Sao Paulo. [19]

2003 (publication year) - An outbreak (6 cases) of *Candida rugosa* fungemia was reported in a tertiary care teaching hospital in Sao Paulo. [20]

2012 (publication year) - An outbreak of *Candida pelliculosa* fungemia was reported in a neonatal intensive care unit. [21]

2012 (publication year) - An outbreak (11 cases) of *Candida parapsilosis* fungemia was reported in a Neonatal Intensive Care Unit in Sao Paulo. [22]

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Capillariasis - extraintestinal

Agent	PARASITE - Nematoda. Adenophorea: Capillaria hepatica (Calodium hepaticum), Capillaria aerophila, Anatrichosoma cutaneum
Reservoir	rat dog cat monkey soil earthworm
Vector	None
Vehicle	Soil Earthworm
Incubation Period	21d -28d
Diagnostic Tests	Visualization of ova or adults in liver, lung or dermal tissue.
Typical Adult Therapy	None available
Typical Pediatric Therapy	As for adult
Clinical Hints	Three infecting species produce: Bronchitis or pneumonia; Acral pruritic rash; or Tender hepatomegaly, abdominal distention, eosinophilia and fever.
Synonyms	Anatrichosoma cutaneum, Calodiasis, Calodium hepaticum, Capillaria aerophila, Capillaria hepatica, Capillariasis - pulmonary, Capillary liver worm, Eucoleus aerophilus, Hepatic capillariasis, Thominx aerophilus. ICD9: 128.8 ICD10: B83.8

Clinical

The spectrum of infestation extends from asymptomatic infestation to fatal disease. [1]
• Symptoms include fever, tender hepatomegaly with hepatic dysfunction, abdominal distention and eosinophilia.
• Splenomegaly and pneumonia have been reported.

Capillaria aerophila infection is characterized by cough, bronchitis, fever and eosinophilia. [2]
• Diagnosis is established by finding ova in sputum.

Anatrichosoma cutaneum infection is characterized by dermal lesions and pruritus of the fingers or feet.

This disease is endemic or potentially endemic to 22 countries.

Capillariasis - extraintestinal in Brazil

Extraintestinal capillariasis was first reported in Brazil in 1963.

Five cases have been reported from Brazil - all from Sao Paulo. [3]

Prevalence surveys:

6.7% of persons in the Amazon region (fecal shedding through spurious infection, 2012 publication) [4]
43% of rats (*Rattus norvegicus*) in Brazil (1981 publication) [5]

Seroprevalence surveys:

0.81% of persons in the Amazon region (2008) [6]

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Chancroid

Agent	BACTERIUM. Haemophilus ducreyi. A facultative gram-negative bacillus
Reservoir	Human
Vector	None
Vehicle	Sexual contact
Incubation Period	3d - 10d (2d - 21d)
Diagnostic Tests	Culture (inform laboratory when this diagnosis is suspected). Fluorescent staining under development
Typical Adult Therapy	Azithromycin 1.0 g PO X 1 dose. OR Ceftriaxone 250 mg IM X 1 dose. OR Ciprofloxacin 500 mg PO BID X 3 days OR Erythromycin 500 mg PO TID X 7d.
Typical Pediatric Therapy	Azithromycin 12 mg/kg PO X 1 dose OR Erythromycin 10 mg/kg PO TID X 7d. OR Ceftriaxone 10 mg/kg IM X 1
Clinical Hints	Soft, painful and tender chancre on erythematous base, with regional lymphadenopathy (generally unilateral and painful); onset 3 to 10 days following sexual exposure.
Synonyms	Blot sjanker, Chancre mou, Chancro blando, Haemophilus ducreyi, Nkumunye, Soft chancre, Ulcera mole, Ulcus molle, Weeke sjanker, Weicher Schanker. ICD9: 099.0 ICD10: A57

Clinical

For surveillance the CDC (The United States Centers for Disease Control) case definition consist of a sexually-transmitted disease characterized by painful genital ulceration and inflammatory inguinal adenopathy; but without evidence for *Treponema pallidum* by dark field and serological examination (after at least 7 days) and without clinical or laboratory evidence for herpes simplex infection.

Infection begins with a papule or pustule which ulcerates and enlarges over a period of 1 to 2 days. [1]
- The lesion is soft, painful and bleeds easily; and the ulcer edges are undermined and irregular. [2]
- Two thirds of patients present with more than one ulcer
- Painful unilateral or bilateral lymphadenopathy is present in 40% of cases.
- Systemic signs are unusual.
- Extragenital skin ulcers are occasionally encountered. [3] [4]
- *Haemophilus ducreyi* has been associated with esophageal ulceration in HIV-positive patients. [5]

This disease is endemic or potentially endemic to all countries.

Chancroid in Brazil

Approximately 9,000 cases were reported during 1987 to 1996.

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Chandipura and Vesicular stomatitis viruses

Agent	VIRUS - RNA. Rhabdoviridae, Vesiculovirus: Chandipura virus Vesicular stomatitis virus
Reservoir	Horse Cattle Pig
Vector	None
Vehicle	Aerosol from animal
Incubation Period	2d - 6d (range 1d - 8d)
Diagnostic Tests	Viral culture (blood). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Myalgia, headache, conjunctivitis, oral and digital vesicles; often follows animal contact; infection resolves within one week; no fatality or residua. Chandipura virus (India) associated with outbreaks of severe encephalitis.
Synonyms	Alagoas, Calchaqui, Chandipura, Cocal, Epidemic stroke, Indiana, Isfahan, LeDantec, Piry, Vesicular stomatitis. ICD9: 066.8 ICD10: A93.8

Clinical

Chandipura virus infection:
Chandipura virus infection is characterized by fever, myalgia, and arthralgia without rash. [1]
• Periodic outbreaks of Chandipura encephalitis, primarily affecting children, are reported in India.
• Infection is characterized by fever, diarrhea, vomiting and encephalitis • with a case-fatality rate of 47%. [2]

Vesicular stomatitis virus infection:
Human infection by Vesicular stomatitis virus is often mild, transitory and nonspecific; however, cases of severe encephalitis (Chandipura and Indiana viruses) have been reported.
• In some cases, a biphasic illness is observed.
• Prominent findings include headache, myalgias, pharyngitis (40% of cases), conjunctivitis and cervical lymphadenopathy.
• Vesicles or ulcers are found in a minority of patients, appearing on the oral mucosa, lips, tongue, fingers or nose.

Piri virus infection is associated with mild illness consisting of headache, myalgia, arthralgia and photophobia.

Rare instances of **Le Dantec virus** infection have been characterized by fever, hepatomegaly, splenomegaly, headache and delirium.

This disease is endemic or potentially endemic to 26 countries.

Chandipura and Vesicular stomatitis viruses in Brazil

Piry virus, a member of the vesicular stomatitis group. was first isolated from a marsupial (*Philander opossum*) in Brazil in 1960. [3]

High levels of seroprevalence are found in the Amazon basin.

8.0 of blood donors in Uberaba, Minas Gerais State are seropositive toward Piry virus. [4]

Accidental infection of laboratory workers has been associated with mild illness characterized by fever, headache, myalgia, arthralgia and photophobia lasting two days.

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Chikungunya

Agent	VIRUS - RNA. Togaviridae, Alphavirus: Chikungunya virus. Related Semliki Forest and Me Tri viruses are found in Africa & Asia
Reservoir	Non-human primate
Vector	Mosquito (Aedes spp. ; Ae. furcifer-taylori group in Africa)
Vehicle	None
Incubation Period	2d - 12d
Diagnostic Tests	Viral culture (blood). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Abrupt fever, leukopenia, myalgia and prominent bilateral joint pain; maculopapular rash appears on 2nd to 5th days in greater than 50% of cases; fever resolves within 7 days, but joint pain may persist for months.
Synonyms	Buggy Creek, Getah, Knuckle fever, Me Tri, Semliki Forest. ICD9: 062.8,066.3 ICD10: A92.1

Clinical

The fever of Chikungunya is characterized by a rapid rise in temperature to as high as 40 C, often accompanied by rigors, myalgia, headache, photophobia, retro-orbital pain, sore throat with objective signs of pharyngitis, nausea, and vomiting. [1]
• Fever may abate after a few days, only to recrudesce ("saddle-back" fever curve").
• Polyarthralgia occurs in 70% of cases, favors small joints and sites of previous injury, and is most intense on arising.
• Joints may swell, but without significant fluid accumulation. [2] [3]
• Joint pain is most severe in adults.
• Symptoms may last for from 1 week to several months. [4]
• Joint involvement may progress to residual chronic pain [5] [6] or destructive arthritis. [7]
• Arthralgia may persist for as long as 24 months. [8-14] In one series, 57% of patients continued to experience rheumatological symptoms for 15 months or more [15] [16] ; and in another, 66.5% reported myalgia, asthenia or arthralgia for more than 12 months. [17]
• Imaging studies may reveal joint effusion, bony erosion, marrow edema, synovial thickening, tendonitis and tenosynovitis. [18]
• Laboratory tests reveal mild leukopenia and relative lymphocytosis; persistent mixed cryoglobulinemia is present in most cases. [19] Cases of hepatic dysfunction have also been reported. [20]

Dermatological manifestations:
A rash characteristically appears on the first day of illness, but may be delayed.
• The patient exhibits erythema of the face and neck, which evolves to a macular or maculopapular exanthem of the trunk, limbs, face, palms, and soles in 50% of cases. [21]
• Common findings also include hyperpigmentation, xerosis, excoriated papules, aphthous-like ulcers, vesiculobullous and lichenoid eruptions, and exacerbation of pre-existing or quiescent dermatoses. [22] [23]
• Pigmentary changes are seen in 42% of cases, intertriginous aphthous-like ulcers in 21.37% and a vesiculbullous eruption in 2.75% (only in infants). [24]
• Morbilliform eruptions are most common, followed by scaling, macular erythema, intertrigo, hypermelanosis, xerosis, excoriated papules, urticaria and petechiae. [25]
• Vesiculo-bullous lesions are most common in children [26] ; and extensive bullous lesions have been reported in infected infants. [27]
• Pruritus is common, and petechiae have been seen in some patients.
• Purpuric macules [28] , genital ulcers, desquamation of the facial skin [29] , erythema multiforme and erythema nodosum have also been reported in patients with Chikungunya. [30]

Complications:
Complications include hemorrhagic syndrome, myopericarditis [31] [32] , hemodynamic disorders [33] and rare instances of renal

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failure. [34]
• Fatal infection [35] [36] and transplacental infections have been reported. [37-39]
• Peritonitis, encephalitis and secondary bacterial infections have been reported among immunocompromised patients with Chikungunya. [40]
• The case fatality rate may be as high as 1 per 1,000 cases. [41] [42]
• Children occasionally present with seizures or convulsions.
• Sudden sensorineural hearing loss has been reported [43]
• Eye involvement may present as transient granulomatous and nongranulomatous anterior uveitis, optic neuritis [44] , retinitis [45] [46] , retrobulbar neuritis [47] , Fuchs' heterochromic iridocyclitis. [48] and dendritic lesions. [49]
• Chikungunya has no observable effect on the outcome of pregnancy [50] ; however, infection of infants during the perinatal period is characterized by fever, rash, peripheral edema, thrombocytopenia, lymphopenia, decreased prothrombin value, and elevation of aspartate aminotransferase levels.
• Neurological complications include altered mental function, seizures [51] , encephalitis [52] [53] , myelopathy [54] [55] , myeloradiculopathy [56] , acute flaccid paralysis [57] , focal neurological deficit [58] with abnormal CT scan of head, Guillain-Barre syndrome [59] , urinary retention [60] and altered CSF biochemistry. [61-63]

In some cases Chikungunya may mimic Kawasaki disease. [64]
• Although the clinical features of dengue and chikungunya are similar, chikungunya patients are more likely to exhibit early myalgia or arthralgia; while sore throat, cough, nausea, vomiting, diarrhea, abdominal pain, anorexia, tachycardia and thrombocytopenia will favor a diagnosis of dengue. [65]

Infection by a related agent, **Semliki Forest virus**, is characterized by fever, myalgia, arthralgia and persistent headache. [66]

This disease is endemic or potentially endemic to 48 countries. Although Chikungunya is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Chikungunya in Brazil

2010 - Two Brazilian travelers acquired Chikungunya in Indonesia, and one in India. [67-69]

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Chlamydia infections, misc.

Agent	BACTERIUM. Chlamydiaceae, Chlamydiae, Chlamydia trachomatis; Simkania negevensis; Waddlia chondrophila
Reservoir	Human
Vector	None
Vehicle	Sexual contact
Incubation Period	5d - 10d
Diagnostic Tests	Microscopy and immunomicroscopy of secretions. Serology. Tissue culture. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg BID X 7d. OR Azithromycin 1g as single dose OR Levofloxacin 500 mg daily X 7 days OR Ofloxacin 300 mg BID X 7 days
Typical Pediatric Therapy	Weight <45 kg: Erythromycin 10 mg/kg QID X 14d Weight >=45 kg, but age <8 years: Azithromycin 1 g as single dose Age >= 8 years: Azithromycin 1 g as single dose OR Doxycycline 100 mg BID X 7 d
Clinical Hints	Thin, scant penile discharge; cervicitis; conjunctivitis; neonatal pneumonia; pelvic inflammatory disease; concurrent gonorrhea may be present.
Synonyms	Bedsonia, Chlamydia trachomatis, Chlamydien-Urethritis, Chlamydien-Zervizitis, Chlamydophila, Inclusion blenorrhea, Non-gonococccal urethritis, Nonspecific urethritis, Parachlamydia, Parachlamydia acanthamoebae, Prachlamydia, Protochlamydia, Protochlamydia naegleriophila, Rhabdochlamydia, Simkania negevensis, Waddlia chondrophila. ICD9: 099.41,099.5 ICD10: A56,A55

Clinical

Genito-urinary Infection with *Chlamydia trachomatis* may result in urethritis, epididymitis [1] , obstructive uropathy [2] , cervicitis, Fitz-Hugh-Curtis syndrome [3-5] , acute salpingitis, tubal scaring, reduced conception rates (even in the absence of scarring) [6] , ectopic pregnancy [7-10] , miscarriage [11] [12] , low birth weight or pre-term delivery. [13-16]
• The rates of orchitis/epididymitis, prostatitis, infertility, and urethral stricture following genital infection in males are 4.28%, 1.41%, 1.27%, and 0.13% • respectively. [17]
• The extent to which *Chlamydia* infection contributes to male and female infertility is unclear. [18-22]
• Levels of serum Prostate-specific Antigen (PSA) may be elevated in patients with *Chlamydia trachomatis* infection. [23]
• Perinatal infections may result in inclusion conjunctivitis or pneumonia in the newborn. [24]
• Asymptomatic pharyngeal infection or acute chlamydial tonsillopharyngitis may follow oro-genital contact. [25]

Chlamydia trachomatis infection is implicated in the etiology of reactive arthritis. [26-45]

Parachlamydiaceae (including *Parachlamydia acanthamoebae*) have been associated with human respiratory infections, conjunctivitis, keratitis and uveitis. [46] [47]
• The signs and symptoms of infection are similar to those of genital *Mycoplasma* infection. [48]
• Recurrent infection may represent either reinfection or treatment failure. [49]

For surveillance purposes, the CDC (The United States Centers for Disease Control) case definition of nongonococcal urethritis requires that gonorrhea has been discounted in the setting of:
• a visible abnormal urethral discharge
• or, a positive leukocyte esterase test from a male aged <60 who does not have a history of kidney disease or bladder infection, prostatic enlargement, anatomical abnormality of the urogenital tract, or recent urinary tract instrumentation
• or microscopic evidence of urethritis (over 5 leukocytes per high-power field) on stain of a urethral smear.

This disease is endemic or potentially endemic to all countries.

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Chlamydia infections, misc. in Brazil

3,481,000 cases of genital *Chlamydia* infection were estimated in 1996.

Prevalence surveys:

21.5% of pregnant women over age 20
41.5% of pregnant adolescents
9% of pregnant women in Sao Paulo (1995 publication) [50]
25.7% of pregnant women in Botacatu, Sao Paulo (2006 to 2008) [51]
9.4% of pregnant women in Manaus, Fortaleza, Goiania, Rio de Janeiro, Sao Paulo and Porto Alegre (2004 to 2005) [52]
4.7% of pregnant women in Pelotas (2005 to 2008) [53] [54]
2.7% of pregnant women in the western Amazon (2008) [55]
8.9% of female adolescents in Vitoria (2004 publication) [56]
19.6% of adolescent and young women in central Brazil (2006 publication) [57]
7.4% of women ages 14 to 49 attending a primary health unit (2008 publication) [58]
9.8% of women ages 15 to 24 years, attending public hospitals (2009) [59]
23.9% of women attending an outpatient clinic in Sao Paulo (2006 to 2008) [60]
8.4% of women ages 18 to 40 years, in Sao Paulo (2011 publication) [61]
13.5% of women ages <24 years attending a family planning clinic (Campinas, 2009 publication) [62]
1.1% of candidates for in-vitro fertilization (Sao Paulo, 2008 to 2009) [63]
8% of women seeking HIV testing in Rio de Janeiro (2004 publication) [64]
4.3% of HIV-positive women in Manaus (2009 to 2010) [65]
11.1% of HIV-positive women in Salvador, Bahia (2012 publication) [66]
31% of sexually-active adolescents in Salvador (2008 to 2010) [67]
4.5% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (2007 publication) [68]
14.5% of sexually-active women ages 15 to 19 (2009 publication) [69]
10.7% of sexually-active urban women ages 16 to 23 (southern Brazil, 2011 publication) [70]
20.7% of female STD patients in Manaus (2003 publication) [71]
13.1% of male STD patients (2005) [72]
6.4% of women in rural Alagoas (1997)
6.7% of gynecological outpatients in Sao Paulo (1987 publication) [73]
58.6% of gynecology outpatients in Recife (PCR, 2008 publication) [74]
52.8% of infertile women in Manaus (2011 publication) [75]
6.3% of cervical specimens from patients attending public health services in Belo Horizonte and Contagem, Minas Gerais (2011 publication) [76]
3.0% of women with systemic lupus erythematosus vs. 5.0% in a control group (Bahia, 2012 publication) [77]
5.0% of asymptomatic military conscripts (2000) [78]
1.4% of men and 4.8% of women in Alto Solimoes, Amazon border region (2012 publication) [79]
11% of Parakana Indians in the Amazon (1993 publication) [80]
1.6% of children below age 5 years hospitalized with pneumonia (*Simkania negevensis*, Salvador, 2009 publication) [81]
9.9% of respiratory infection among infants from low-income families (*Chlamydia trachomatis*, 2012 publication) [82]

Seroprevalence surveys:

16.9% of patients with metabolic syndrome and 5.6% of controls (IgA, 2011 publication) [83]

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Chlamydophila pneumoniae infection

Agent	BACTERIUM. Chlamydiaceae, Chlamydiae, Chlamydophila [Chlamydia] pneumoniae
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	7d - 28d
Diagnostic Tests	Direct fluorescence of sputum. Serology and culture in specialized laboratories. Nucleic acid amplification.
Typical Adult Therapy	Respiratory isolation. Doxycycline 50 mg BID X 10d. Alternatives: Erythromycin 500 mg QID X 10d. Azithromycin 1 g, then 0.5 g daily. Clarithromycin 0.5 g BID
Typical Pediatric Therapy	Respiratory isolation; Erythromycin 10 mg/kg QID X 10d
Clinical Hints	Atypical pneumonia, often associated with pharyngitis and myalgia; consider when Mycoplasma, Legionella and influenza are discounted.
Synonyms	Chlamydia pneumoniae, Chlamydia TWAR, Chlamydophila pneumoniae, TWAR. ICD9: 078.88 ICD10: J16.0

Clinical

Asymptomatic infection is common.
• Pneumonia and bronchitis are the most common clinical syndromes associated with *C. pneumoniae*. [1]
• Sinusitis and pharyngitis may also occur, even in the absence of lower respiratory tract infection.
• Initial symptoms may consist of rhinitis, sore throat, or hoarseness; followed after several days or weeks prominent cough.
• Fever is often absent.
• Cough and malaise may persist for months; and reinfection may occur.

A single, subsegmental, patchy infiltrate may be seen on chest X ray.
• Other findings described include, lobar pulmonary consolidation, interstitial infiltrates, bilateral pneumonia, pleural effusion, acute respiratory distress syndrome [2] , hilar adenopathy [3] and myo-pericarditis. [4]
• The appearance of a miliary infiltrate may suggest a diagnosis of tuberculosis. [5]
• *Chlamydophila pneumoniae* has been identified as an agent of otitis media. [6]
• Rare cases of intra-hepatic cholestasis have been reported. [7]
• The peripheral white blood cell count is usually not elevated.

C. pneumoniae has been identified as a cause of acute respiratory exacerbations in patients with cystic fibrosis and acute respiratory infection in children with sickle cell disease.
• *C. pneumoniae* infection is implicated in the etiology of recurrent tonsillitis. [8]
• The organism has also been implicated in development of asthma [9-12] , chronic rhinosinusitis [13] , otitis media, migraine [14] , endocarditis, lumbosacral meningoradiculitis, erythema nodosum, erythema exsudativum multiforme [15] , nodular vasculitis [16] , Guillain-Barre syndrome, keratoconjunctivitis sicca [17] , hemophagocytic lymphohistiocytosis [18] [19] , reactive arthritis and atherosclerosis. [20]

This disease is endemic or potentially endemic to all countries.

Chlamydophila pneumoniae infection in Brazil

Prevalence surveys:
5.2% of community-acquired pneumonia cases in Porto Alegre - 39.6% of patients showed evidence for past infection (2007 publication) [21]
8.2% of community-acquired pneumonia cases in Sumare microregion (2011 publication) [22]

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Cholecystitis & cholangitis

Agent	BACTERIUM. Escherichia coli, Klebsiella pneumoniae, enterococci, et al.
Reservoir	Human
Vector	None
Vehicle	Endogenous bacteria
Incubation Period	Variable
Diagnostic Tests	Roentgenograms/imaging (cholecystogram, ultrasound, CT, etc).
Typical Adult Therapy	Antibiotics and surgical intervention as required
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, chills and right upper quadrant abdominal pain; often "female, fat and 40"; may be associated with gallstones or pancreatitis, or present as 'fever of unknown origin'.
Synonyms	Acute cholecystitis, Angiocholite, Ascending cholangitis, Cholangitis, Cholecystite, Cholecystitis, Cholezystitis, Colangite, Colangitis, Colecistite, Gall bladder. ICD9: 575.0,576.1 ICD10: K81,K83.0

Clinical

Cholangitis is caused by obstruction of the common bile duct, which subsequently becomes infected. [1]
• Strictures, stenosis, tumors, or endoscopic manipulation of the CBD cause bile stasis.
• The resultant infection ascends into the hepatic ducts, while increased biliary pressure spreads infection into the biliary canaliculi, hepatic veins and perihepatic lymphatics, leading to bacteremia.

Charcot's triad (fever, right upper quadrant pain, and jaundice) is found in 70% of patients.
• Additional findings include right upper quadrant pain, mild hepatomegaly, tachycardia, altered mental status, rigors, fever, hypotension, jaundice, pruritis, acholic stools.
• The case-fatality rate is 7% to 40%, and is highest in patients with hypotension, renal failure, liver abscess, cirrhosis, inflammatory bowel disease, malignant strictures and advanced age, or delays in diagnosis or surgery.

This disease is endemic or potentially endemic to all countries.

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Cholera

Agent	BACTERIUM. Vibrio cholerae A facultative gram-negative bacillus
Reservoir	Human
Vector	None
Vehicle	Water Fecal-oral Seafood (oyster, ceviche) Vegetables Fly
Incubation Period	1d - 5d (range 9h - 6d)
Diagnostic Tests	Stool culture. Advise laboratory when this organism is suspected.
Typical Adult Therapy	Stool precautions. Doxycycline 100 mg BID X 5d, or Fluoroquinolone (Levofloxacin, Trovafloxacin, Pefloxacin, Sparfloxacin or Moxifloxacin)., or Azithromycin Fluids (g/l): NaCl 3.5, NaHCO3 2.5, KCl 1.5, glucose 20
Typical Pediatric Therapy	Stool precautions. Age >=8 years: Doxycycline 2 mg/kg BID X 5d. Age <8 years: Sulfamethoxazole/trimethoprim Fluids (g/l): NaCl 3.5, NaHCO3 2.5, KCl 1.5, glucose 20
Vaccines	Cholera - injectable Cholera - oral
Clinical Hints	Massive, painless diarrhea and dehydration; occasionally vomiting; apathy or altered consciousness common; rapid progression to acidosis, electrolyte imbalance and shock; fever is uncommon.
Synonyms	Colera, Kolera. ICD9: 001 ICD10: A00

Clinical

WHO Case definition for surveillance:
The WHO Case definition for surveillance is as follows:
Clinical case definition
• In an area where the disease is not known to be present: severe dehydration or death from acute watery diarrhea in a patient aged 5 years or more or
• In an area where there is a cholera epidemic: acute watery diarrhea, with or without vomiting in a patient aged 5 years or more
Laboratory criteria for diagnosis
• Isolation of *Vibrio cholerae* O1 or O139 from stools in any patient with diarrhea.
Case classification
• Suspected: A case that meets the clinical case definition.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory-confirmed.
Note: In a cholera-threatened area, when the number of .confirmed cases rises, shift should be made to using primarily the .suspected. case classification.
• Cholera does appear in children under 5 years; however, the inclusion of all cases of acute watery diarrhea in the 2-4 year age group in the reporting of cholera greatly reduces the specificity of reporting.
• For management of cases of acute watery diarrhea in an area where there is a cholera epidemic, cholera should be suspected in all patients.

Symptoms and signs of cholera reflect the degree of fluid loss: thirst, postural hypotension, tachycardia, weakness, fatigue and dryness of the mucous membranes.
• Following an incubation period of 12 hours to 5 days [1] , the patient experiences sudden onset of painless, watery diarrhea, which may later be accompanied by vomiting. [2]
• Abdominal cramps may occur.
• Fever is typically absent in adults, but present in children.
• The diarrhea has a "rice water" appearance and fishy odor.
• In patients with severe disease, stool volume can exceed 250 ml per /kg during the first 24 hours (17.5 liters in a 70 kg adult!).
• Severe cases exhibit sunken eyes (depressed fontanelles in infants), thready pulse, somnolence or coma.
• Without replacement of fluids and electrolytes, hypovolemic shock and death ensue.
• The clinical features of cholera due to *Vibrio cholerae* O139 are indistinguishable from disease due to other strains. [3]
• Rare cases of acalculous [4-6] and infectious cholecystitis have been ascribed to *Vibrio cholerae*. [7]

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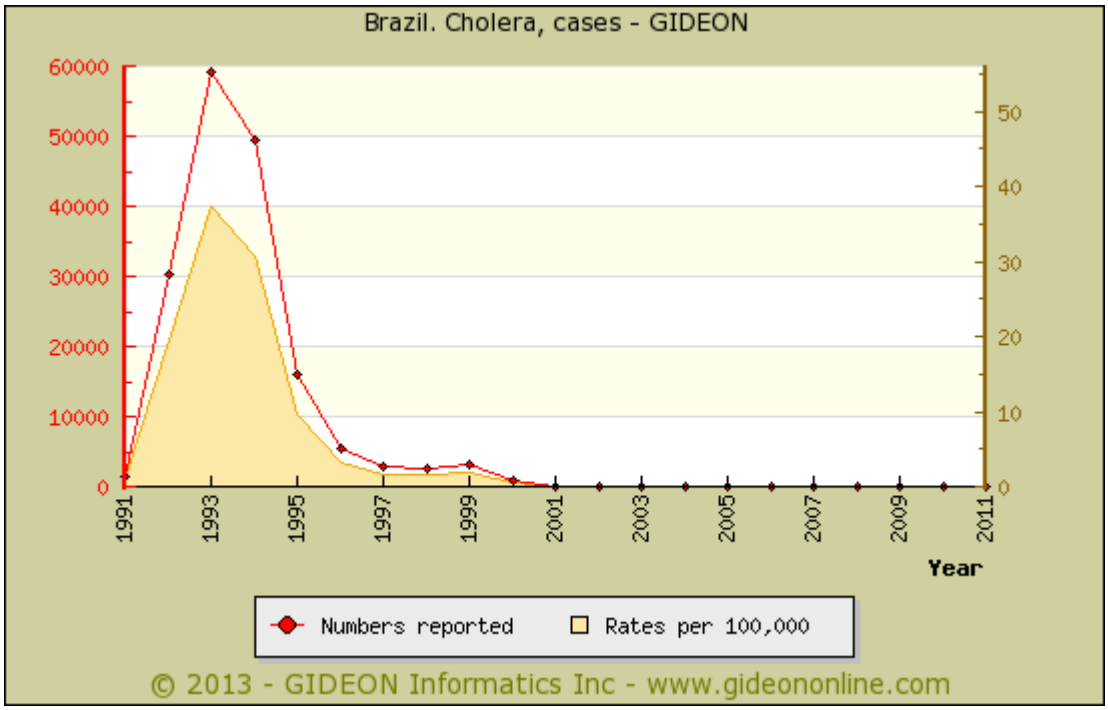
This disease is endemic or potentially endemic to 119 countries.

Cholera in Brazil

Cholera is currently or recently reported from:
Acre State
Alagoas State
Amapa State
Amazonas State
Bahia State
Ceara State
Distrito Federal State
Espirito Santo State
Maranhao State
Mato Grosso State
Minas Gerais State
Para State
Paraiba State
Paranha State
Pernambuco State
Piaui State
Rio de Janeiro State
Rio Grande do Norte State
Rondonia State
Sao Paulo State
Sergipe State

As of December, 1999 the following were removed from the WHO 'Infected Areas list': Acre State, Amapa State, Amazonas State, Distrito Federal, Espirito Santo State, Maranhao State, Mato Grosso State, Para State, Piaui State, Rio de Janeiro State, Rondonia State, Sao Paulo State.
- As of October 2000, the following were removed from the 'Infected Areas List': Bahia State, Ceara State, Minas Gerais State, Parana State, Rio Grande do Norte State.



Graph: Brazil. Cholera, cases

Notes:
1. 166,598 cases were reported during 1980 to 2001.
2. 161,432 cases (1,296 fatal) were reported during 1991 to 1998. [8]
   Individual years:

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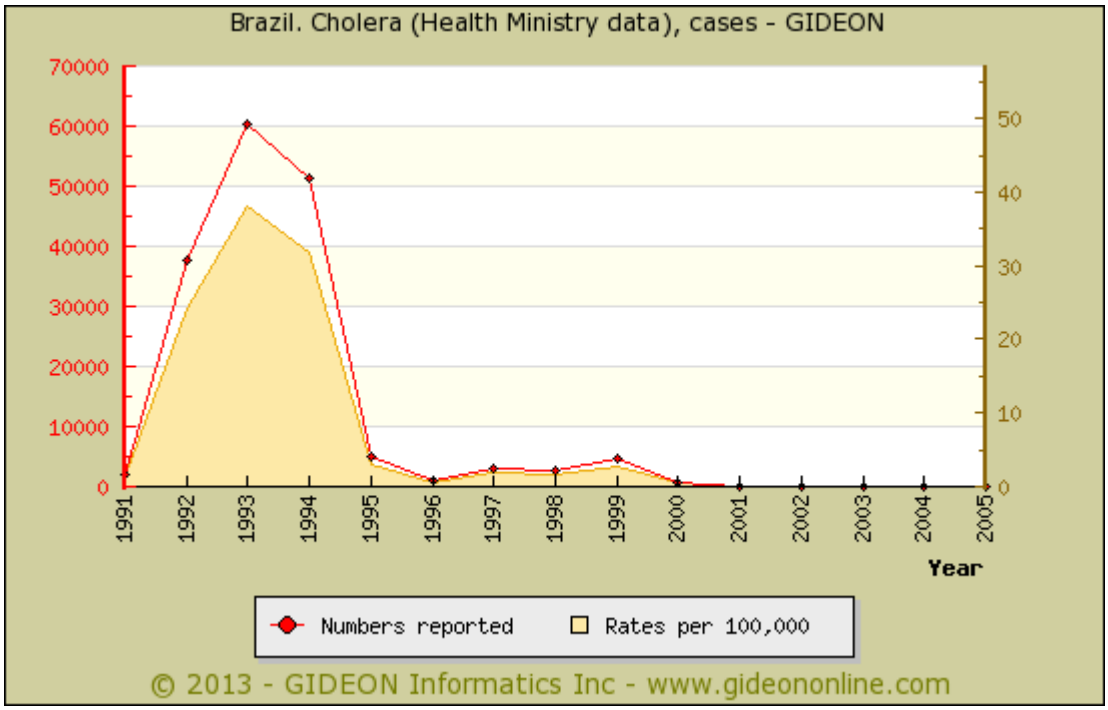
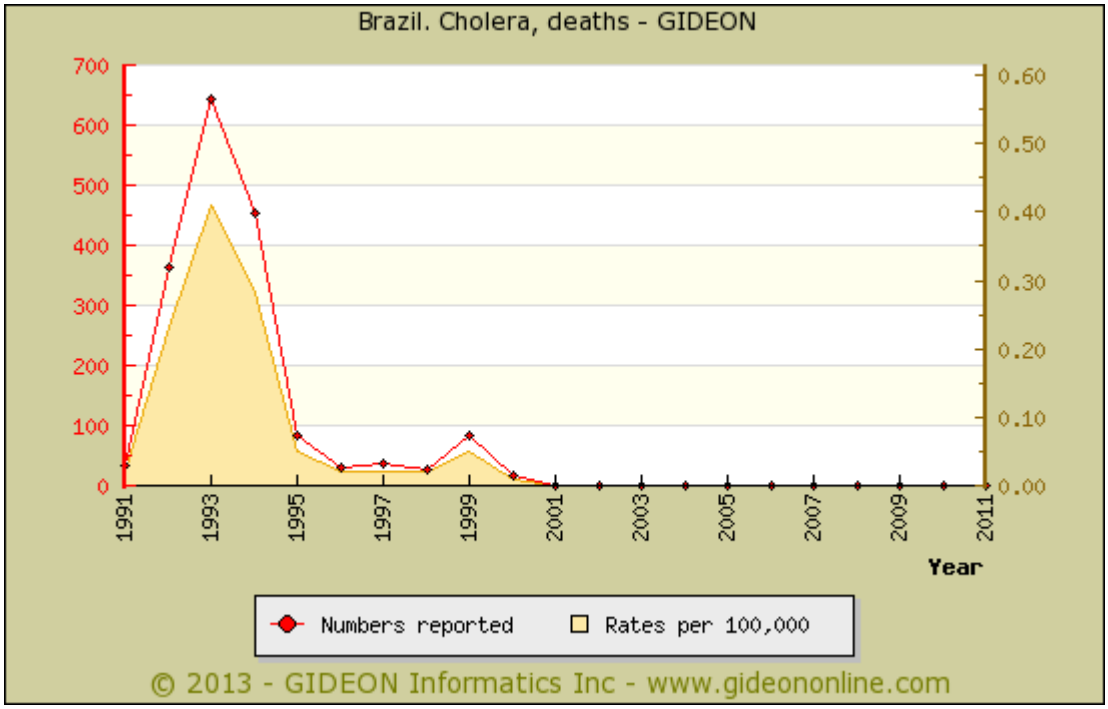
1998 - Notably during an outbreak in Cortez Municipality (Pernambuco).
1999 - Cases reported in Paranagua municipality (Parana State).
2011 - Imported case [9]



Graph: Brazil. Cholera (Health Ministry data), cases



Graph: Brazil. Cholera, deaths

Notable outbreaks:

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1855 to 1856 - An outbreak of cholera was reported in Minas Gerais. [10]

1992 to 1994 - An outbreak (138,976 cases, 1,459 fatal) of cholera was reported. [11-16]

1997 - An outbreak (183 cases, 9 fatal) was reported in Alagoas. [17]

1998 - An outbreak (376 cases, 1 fatal) was reported in Pernambuco. [18] A subsequent outbreak (25 cases) was reported in Bahia. [19]

1999 - An outbreak (155 cases, 2 fatal) was reported in Parana. [20-22]

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Chromomycosis

Agent	FUNGUS. Ascomycota, Euascomycetes, Chaetothyriales. Dematiaceous molds: Phialophora, Cladiophialophora, Fonsecaea, Rhinocladiella
Reservoir	Wood Soil Vegetation
Vector	None
Vehicle	Minor trauma
Incubation Period	14d - 90d
Diagnostic Tests	Biopsy and fungal culture.
Typical Adult Therapy	Itraconazole 100 mg PO QID X (up to) 18 m. OR (for late disease) Flucytosine 25 mg/kg QID X 4m. Terbinafine has been used in some cases. Local heat; excision as necessary
Typical Pediatric Therapy	Itraconazole 1 mg/kg PO BID X (up to) 18 m. OR Ketoconazole (if age >2) 5 mg/kg/d X 3 to 6m. Local heat; excision as necessary
Clinical Hints	Violaceous, verrucous, slowly-growing papule(s) or nodules, most commonly on lower extremities; usually follows direct contact with plant matter in tropical regions.
Synonyms	Chromoblastomycosis, Chromomykose, Verrucous dermatitis. ICD9: 117.2 ICD10: B43.0

Clinical

The lesions of chromomycosis typically progress from a papule to cicatricial fibrosis: nodules, tumors, plaques, warty lesions, and scarring lesions. [1] [2]

- The verrucous form appears at the site of inoculation.
- The primary lesion, a small pink scaly papule, may be pruritic but rarely painful. [3]
- Over time (often months to years), new crops of lesions appear in the same or adjacent areas as warty, purplish, scaly nodules or smooth, firm tumors. [4]
- Peripheral spread may occur with healing in the center, as lesions enlarge and become grouped.
- Older lesions resemble cauliflower, with small ulcerations or "black dots" of hemopurulent material on the surface. [5]
- These lesions can be pruritic and are rarely painful.
- Satellite lesions may develop through autoinoculation or lymphatic spread.
- Coalesced lesions form a large verrucous mass.
- Occasionally, an annular, flattened, papular lesion having a raised border is encountered.
- Keloid formation, fibrosis, lymphostasis and marked edema may follow.
- Fistulae are not seen.
- Malignant transformation has been reported in long-lasting lesions. [6]

Signs of mucosal infection may mimic those of rhinosporidiosis [7] , while those of cutaneous infection may mimic dermal leishmaniasis. [8]

Rare cases of postoperative eye infection have been reported. [9]

Rare cases of hematogenous spread to the brain, lymph nodes, liver, lungs, bones and joints [10] , soft tissues and other organs have been reported. [11]

This disease is endemic or potentially endemic to all countries.

Chromomycosis in Brazil

Sporadic case reports are published. [12-17]

Case series:

- A series of 100 cases was treated at a hospital in Rio Grande do Sul during 1963 to 1998. [18]
- A series of 27 cases was treated at a hospital in Sao Paulo during 1997 to 2007. [19]

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- A series of 18 cases treated at a hospital in Rio de Janeiro was reviewed in 2011. [20]
- A series of 65 cases was treated at a hospital in Para during 2000 to 2007. [21]

Chromomycosis due to *Cladiophialophora carrionii* has been reported in Rio de Janeiro State (2008 publication) [22]

Chromomycosis due to *Rhinocladiella aquaspersa* was reported in a Brazilian immigrant to the Netherlands. [23]

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Chronic meningococcemia

Agent	BACTERIUM. Neisseria meningitidis An aerobic gram-negative coccus
Reservoir	Human
Vector	None
Vehicle	Air Infected secretions
Incubation Period	Unknown
Diagnostic Tests	Blood culture. Test patient for complement component deficiency.
Typical Adult Therapy	Intravenous Penicillin G 20 million units daily X 7 days
Typical Pediatric Therapy	Intravenous Penicillin G 200,000 units daily X 7 days
Clinical Hints	Recurrent episodes of low-grade fever, rash, arthralgia and arthritis - may persist for months; rash is distal, prominent near joints and may be maculopapular, petechial or pustular; may be associated with complement component deficiency.
Synonyms	Meningococcemia, chronic. ICD9: 036.2 ICD10: A39.3

Clinical

Chronic meningococcemia is characterized by persistent meningococcal bacteremia associated with low-grade fever, rash and arthritis.

- The rash is similar to that of gonococcemia. [1] [2]
- The illness may recur over a period of weeks to months.
- Patients (or their contacts) may ultimately present with acute bacterial meningitis or septicemia.

Non-bacteremic cases occur, and may be diagnosed through demonstration of meningococci in skin lesions. [3]

This disease is endemic or potentially endemic to all countries.

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Clostridial food poisoning

Agent	BACTERIUM. Clostridium perfringens An anaerobic gram-positive bacillus
Reservoir	Soil Human Pig Cattle Fish Poultry
Vector	None
Vehicle	Food
Incubation Period	8h - 14h (range 5h - 24h)
Diagnostic Tests	Laboratory diagnosis is usually not practical. Attempt culture of food for C. perfringens.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Abdominal pain; watery diarrhea (usually no fever or vomiting) onset 8 to 14 hours after ingestion of meat, fish or gravy; no fecal leucocytes; usually resolves within 24 hours.
Synonyms	

Clinical

Seven to 15 hours after ingestion of toxin (range 6 to 24), the patient develops watery diarrhea (90%), abdominal cramps (80%); and occasionally nausea (25%), vomiting (9%) or fever (24%). [1]
- Symptoms may persist for 8 to 72 hours (usually one day)
- Fatal cases are rare [2] [3]

This disease is endemic or potentially endemic to all countries.

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Clostridial myonecrosis

Agent	BACTERIUM. Clostridium perfringens An anaerobic gram-positive bacillus
Reservoir	Soil Human
Vector	None
Vehicle	Soil Trauma
Incubation Period	6h - 3d
Diagnostic Tests	Gram stain of exudate. Wound and blood cultures. Presence of gas in tissue (not specific).
Typical Adult Therapy	Prompt, aggressive debridement. Penicillin G 3 million units IV Q3h + Clindamycin 900 mg IV Q8h. Hyperbaric oxygen
Typical Pediatric Therapy	Prompt, aggressive debridement. Penicillin G 50,000 units/kg IV Q3h + Clindamycin 10 mg/kg IV Q6h. Hyperbaric oxygen
Vaccine	Gas gangrene antitoxin
Clinical Hints	Gas gangrene is heralded by rapidly progressive tender and foul smelling infection of muscle associated with local gas (crepitus or seen on X-ray), hypotension, intravascular hemolysis and obtundation.
Synonyms	Anaerobic myonecrosis, Clostridial gangrene, Gas gangrene. ICD9: 040.0 ICD10: A48.0

Clinical

Gas gangrene is a fulminant infection with prominent findings at the infection site and severe systemic disease. [1]

The process may follow trauma (usually of an extremity), surgery (notably intestinal or biliary), septic abortion or delivery, vascular insufficiency or burns, underlying colorectal or pelvic cancer, or neutropenia complicating leukemia or cytotoxic therapy.

Following an incubation period of 1 to 4 days (range 6 hours to 3 weeks) the patient develops severe local pain, heaviness or pressure.
• The infection then progresses within minutes to hours, with localized edema, pallor and tenderness.
• Gas may be noted in the soft tissues by palpation, x-ray or scans, but crepitance is a late finding .
• The skin initially appears pale, and progresses to a magenta or bronze discoloration with hemorrhagic bullae and subcutaneous emphysema.
• A thin, brown, serosanguinous discharge may be present, associated with an offensive odor described as sweetish or "mousey.
• Gram's stain of the discharge shows a large number of gram-positive or gram-variable rods, with few or no white blood cells.

Profound systemic toxicity is also present, diaphoresis, anxiety, and tachycardia disproportionate to fever.
• In fact, fever may be low or absent in the early stages.
• Other complications include intravascular hemolysis, hemoglobinuria, hypotension, renal failure, and metabolic acidosis.
• Central nervous system manifestations are rare and most frequently comprise meningitis with or without pneumencephalon, encephalitis, plexitis, cerebral abscess, or subdural empyema. [2]
• Coma and generalized 'bronze' edema are seen preterminally.

This disease is endemic or potentially endemic to all countries.

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Clostridium difficile colitis

Agent	BACTERIUM. Clostridium difficile An anaerobic gram-positive bacillus
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Assay of stool for C. difficile toxin.
Typical Adult Therapy	Metronidazole 500 mg PO TID X 10d. OR Vancomycin 125 mg [oral preparation] QID X 10d
Typical Pediatric Therapy	Vancomycin 2 mg/kg [oral preparation] QID X 10d
Clinical Hints	Fever, leukocytosis, abdominal pain; mucoid or bloody diarrhea during / following antibiotic therapy; fecal leucocytes present; suspect even when mild diarrhea follows antibiotic intake.
Synonyms	Klebsiella oxytoca colitis, Pseudomembranous colitis. ICD9: 008.45 ICD10: A04.7

Clinical

Symptoms may appear as early as the first or second day of antimicrobial therapy; or as late as 10 weeks after cessation. [1]

• Occasionally, a single dose of an antimicrobial or antineoplastic agent has been implicated. [2]

The frequency of diarrhea ranges from three to as many as 20 stools per day.
• Stools may be soft or watery, but rarely demonstrate overt blood.
• Occult blood in the stool is found in approximately 25% of patients. [3]
• Abdominal pain is present in 22% of patients, fever in 28% and leukocytosis in 50%.
• Reactive polyarthritis and hemolytic-uremic syndrome [4] have been reported in some cases.
• Rare instances of *Clostridium difficile* bacteremia are reported. [5] [6]
• Disease caused by *C. difficile* 027 is relatively severe and carries a higher mortality rate than infection by other strains. [7] [8]

This disease is endemic or potentially endemic to all countries.

Clostridium difficile colitis in Brazil

Prevalence surveys:

5.5% of stool samples from children with acute diarrhea (2003 publication) [9]
6.7% of stool samples from children in Rio de Janeiro (2003 publication) [10]
49% of patients in an intensive care unit, with diarrhea (2006 publication) [11]

Notable outbreaks:

2010 (publication year) - An outbreak of *Clostridium difficile*-associated diarrhea (CDAD) was reported in a hospital intensive care unit in Rio de Janeiro. [12]

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Coccidioidomycosis

Agent	FUNGUS. Ascomycota, Euascomyces, Onygenales: Coccidioides immitis [possibly also Coccidioides posadasii] A dimorphic fungus
Reservoir	Soil
Vector	None
Vehicle	Air
Incubation Period	10d - 14d (range 7d - 28d)
Diagnostic Tests	Culture of sputum, CSF, biopsy etc for fungi. Nucleic acid amplification.
Typical Adult Therapy	(Non-meningitic) Fluconazole 500 mg PO daily. OR Itraconazole 200 mg PO BID X 1y. OR Amphotericin B 0.4 mg/kg/d X 6w, then 0.8 mg/kg qod
Typical Pediatric Therapy	(Non-meningitic) Fluconazole 8 mg/kg/day PO or IV OR Ketoconazole 5 mg/kg/d X 1y, OR Amphotericin B 0.4 mg/kg/d X 6w, then 0.8 mg/kg qod
Clinical Hints	Cough, chest pain, myalgia; often eosinophilia, erythema nodosum, headache; extrapulmonary infection (bone, skin, genitourinary, etc) is occasionally encountered.
Synonyms	California disease, Coccidioides immitis, Coccidioides posadasii, Coccidioidomykose, Desert rheumatism, Posada's disease, Valley fever. ICD9: 114 ICD10: B38

Clinical

It is estimated that 50 to 65% of all infections due to *C. immitis* are subclinical. [1]
- Overt disease is often indistinguishable from other forms of respiratory infection.
- Most cases are self-limited, and without sequelae.

Symptomatic acute infection is characterized by cough, chest pain, shortness of breath, fever, fatigue; often with weight loss. [2] [3]
- A nonpruritic fine papular rash is often present.
- Many patients experience erythema nodosum, erythema multiforme or migratory arthralgias ("desert rheumatism"). [4]

Laboratory findings are usually normal except for an increase in the erythrocyte sedimentation rate, and eosinophilia in some cases. [5]
- Chest X-rays may reveal unilateral infiltrates, hilar adenopathy [6], and pleural effusions (15%) [7]; with pulmonary cavities in 5 to 10%.
- Fulminant bilateral pneumonia has been described rarely, but may herald underlying immune deficiency (eg, AIDS). [8]

A residual pulmonary nodule may be detectable in 5% of cases.
- More overt complications such as fibrotic and cavitary pneumonia are occasionally seen.
- Spontaneous pneumothorax [9] and eosinophilic pneumonia have been reported. [10]
- Failure to respond to therapy may indicate the presence of concurrent tuberculosis. [11]

Systemic dissemination complicates less than 1% of infections in the absence of competent immunity.
- Complications may include fungemia [12], and mediastinal or visceral abscesses [13]
- Severe, disseminated and fatal infections are common in patients with AIDS [14] and other forms of immune deficiency. [15]
- Organ transplant recipients [16] and pregnant women are at risk for disseminated infection. [17]
- Older patients do not appear to be at risk for more severe infection. [18]
- Virtually any organ or system may be infected in this manner [19], including the genital tract [20] [21], bone [22-24] and spine [25-28], native [29] and prosthetic joints [30], eyes [31-34], meninges [35-37], skin [38-40], larynx [41], thyroid [42-44], appendix, liver, pelvic adnexae [45] and pericardium. [46]

Disseminated skeletal infection may suggest a diagnosis of malignancy. [47]

Rare instances of primary cutaneous infection have been acquired through direct inoculation. [48-51]

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This disease is endemic or potentially endemic to 13 countries.

Coccidioidomycosis in Brazil

Twelve autochthonous cases were reported to 1998 - all from the semi-arid portion of northern Brazil. [52]
- 19 cases were reported in Ceara State during a 12-year period - all young males, and most associated with armadillo hunting (2008 publication) [53] [54]

Coccidioides immitis has been found in nine-banded armadillos (*Dasypus novemcinctus*) in Piaui [55] .
- *Coccidioides posadasii* has been recovered from the lungs of bats (*Carollia perspicillata*) in Brazil. [56]

Seroprevalence surveys:

7.4% of volunteers in northeastern Brazil (2008 publication) [57]

Notable outbreaks:

1999 (publication year) - An outbreak (3 humans and 8 dogs) in Piaui was associated with hunting nine-banded armadillos. [58]

2012 (publication year) - An outbreak was reported among armadillo hunters in Ceara. [59]

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Coenurosis

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Taeniidae: Taenia multiceps (Multiceps spp.)
Reservoir	Sheep Wild carnivore, Horse Dog
Vector	None
Vehicle	Water Food Soil (contaminated by dog)
Incubation Period	Unknown
Diagnostic Tests	Identification of parasite in biopsy material.
Typical Adult Therapy	Excision
Typical Pediatric Therapy	As for adult
Clinical Hints	Mass in brain, eye, muscle or subcutaneous tissue; may present months to years after exposure in sheep-raising areas; basilar arachnoiditis with internal hydrocephalus is common.
Synonyms	Multiceps, Taenia multiceps. ICD9: 123.8 ICD10: B71.8

Clinical

Human infection has a predilection for the cysterna magna, and presents as basal arachnoiditis and hydrocephalus. [1]
• Subcutaneous tissue, muscle and eye infections are also reported, and present as a cystic masses (often containing daughter cysts) which may attain the size of a hen's egg.
• The clinical features of coenurosis may mimic those of echinococcosis. [2]

This disease is endemic or potentially endemic to 25 countries.

Coenurosis in Brazil

The first case report (a ewe) of coenurosis in Mato Grosso do Sul was published in 2010. [3]

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Common cold

Agent	VIRUS - RNA. Picornaviridae. Rhinoviruses, Coronavirus, et al.
Reservoir	Human
Vector	None
Vehicle	Droplet Contact
Incubation Period	1d - 3d
Diagnostic Tests	Viral culture and serology are available, but not practical.
Typical Adult Therapy	Supportive; Pleconaril under investigation
Typical Pediatric Therapy	As for adult
Clinical Hints	Nasal obstruction or discharge, cough and sore throat are common; fever >38 C unusual in adults; illness usually lasts one week, occasionally two.
Synonyms	Acute coryza, Raffreddore. ICD9: 079,460 ICD10: J00

Clinical

In young adults, the common cold runs its course in an average of 7 days.

Fever is uncommon, and in most cases, rhinorrhea and nasal obstruction predominate. [1]
- Sore throat, cough and hoarseness are often present.
- The nasal tip is often red, and mucoid secretions and a glistening nasal mucosa are evident.
- The pharynx may be mildly edematous and erythematous, but without exudate.

Complications include bacterial sinusitis, otitis media, exacerbation of chronic bronchitis and precipitation of asthma. [2]
- Rare instances of pneumonia have been attributed to infection by Coronavirus strains OC43 and 229E.
- Severe symptoms, including bronchiolitis are associated with Coronavirus HCoV-NL63 infection in young children.

This disease is endemic or potentially endemic to all countries.

Common cold in Brazil

Prevalence surveys:

16.7% of acute respiratory infections in the age group < 5 years (1984 to 1986). [3]

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Conjunctivitis - inclusion

Agent	BACTERIUM. Chlamydiae, Chlamydia trachomatis
Reservoir	Human
Vector	None
Vehicle	Infected secretions Sexual contact Water (swimming pools)
Incubation Period	5d - 12d
Diagnostic Tests	Demonstration of chlamydiae on direct fluorescence or culture of exudate.
Typical Adult Therapy	Secretion precautions. Topical Erythromycin. Erythromycin 250 mg PO QID. X 14 days OR Doxycycline 100 mg PO BID X 14 days
Typical Pediatric Therapy	Secretion precautions. Topical Erythromycin. Azithromycin 1 g PO as single dose. Alternative If age >8 years, Doxycycline 100 mg PO BID X 7 days.
Clinical Hints	Ocular foreign body sensation, photophobia and discharge which may persist for months to as long as 2 years; keratitis and conjunctival follicles may be evident.
Synonyms	Inclusion conjunctivitis, Paratrachoma. ICD9: 077.0 ICD10: P39.1,A74.0

Clinical

Ophthalmia neonatorum caused by *Chlamydia* is characterized by conjunctival injection without follicles. [1]

Follicular conjunctivitis in adults is most prominent on the lower lid, and the presence of bulbar follicles is highly suggestive of a Chlamydia etiology. [2]
• The infection is usually bilateral and accompanied by profuse discharge.

Parachlamydiaceae (including *Parachlamydia acanthamoebae*) have been associated with conjunctivitis, keratitis and uveitis. [3]

Trachoma may be differentiated from inclusion conjunctivitis by the presence of corneal scarring and a preference of the latter for the upper tarsal conjunctivae.

This disease is endemic or potentially endemic to all countries.

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Conjunctivitis - viral

Agent	VIRUS. Picornavirus, Adenovirus
Reservoir	Human
Vector	None
Vehicle	Contact
Incubation Period	1d - 3d
Diagnostic Tests	Viral isolation is available but rarely practical.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Watery discharge, generalized conjunctival injection and mild pruritus; may be associated with an upper respiratory infection.
Synonyms	Apollo conjunctivitis, Apollo eye, Congiuntivite virale, Hemorrhagic conjunctivitis, Viral conjunctivitis. ICD9: 077.1,077.2,077.3,077.4,077.8,372.0 ICD10: B30,B30.3,H10

Clinical

The symptoms of viral conjunctivitis include erythema, itching and lacrimation.
• The presence of large quantities of pus may suggest a bacterial etiology. [1] [2]

Hemorrhagic conjunctivitis is characterized by sudden onset of painful, swollen, red eyes with subconjunctival hemorrhaging, palpebral follicles, photophobia, foreign body sensation, eyelid edema, punctate keratitis, and excessive tearing. [3] [4]
• Symptoms usually persist for 3 to 5 days.

This disease is endemic or potentially endemic to all countries.

Conjunctivitis - viral in Brazil

Notable outbreaks:

1976 - An outbreak of pharyngoconjunctival fever caused by Adenovirus was reported in Belem. [5]

1983 - An outbreak of acute hemorrhagic conjunctivitis was reported in Cuiaba, Mato Grosso. [6]

1983 to 1984 - An outbreak of acute hemorrhagic conjunctivitis in Sao Paulo was caused by Enterovirus 70. [7] [8]

1987 - An outbreak of acute hemorrhagic conjunctivitis in Belem, Para State was caused by Coxsackievirus A24. [9]

2003 - An outbreak of acute hemorrhagic conjunctivitis caused by Coxsackievirus A24 began in South America in the spring, affecting an estimated 200,000 persons in Brazil. [10-12]

2004 to 2005 - An outbreak (60,000 cases or more) of acute hemorrhagic conjunctivitis due to Coxsackievirus A24 was reported in Rio de Janeiro and Espirito Santo. [13]

2009 - An outbreak of acute hemorrhagic conjunctivitis due to Coxsackievirus A24 was reported in Pernambuco State. [14]

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Cryptococcosis

Agent	FUNGUS - Yeast. Basidiomycota, Hymenomycetes, Sporidiales: Cryptococcus neoformans
Reservoir	Pigeon Soil
Vector	None
Vehicle	Air
Incubation Period	Variable
Diagnostic Tests	Fungal culture and stains. Latex test for fungal antigen in CSF and serum. Nucleic acid amplification.
Typical Adult Therapy	Amphotericin B 0.3 mg/kg/d X 6w (+/- Flucytosine); then 0.8 mg/kg qod X 8w. OR Fluconazole 200 mg/d
Typical Pediatric Therapy	Amphotericin B 0.3 mg/kg/d X 6w (+/- Flucytosine); then 0.8 mg/kg qod X 8w. OR Fluconazole 3 mg/kg/d
Clinical Hints	Chronic lymphocytic meningitis or pneumonia in an immune-suppressed patient; meningitis may be subclinical, or "wax and wane" - nuchal rigidity absent or minimal; bone, skin, adrenals, liver, prostate and other sites may be infected.
Synonyms	Busse-Buschke disease, Cryptococcus, European blastomycosis, Torulosis. ICD9: 117.5,321.0 ICD10: B45

Clinical

Central nervous system infection:
Central nervous system infection may be acute or gradual in onset, with acute manifestations most common in immunosuppressed patients (eg, with AIDS). [1]
• Often, the onset is characterized by waxing and waning manifestations over weeks to months, interspersed by asymptomatic periods.
• Complaints may be mild and nonspecific, and consist of headache, nausea, dizziness, irritability, somnolence, confusion, or obtundation. [2]
• Decreased visual acuity, diplopia, and facial weakness may be evident.
• Fever is often absent, and patients have minimal or no nuchal rigidity.
• Papilledema is noted as many as one third of cases, and cranial nerve palsies in 20%. Bilateral amaurosis has been reported as a sequela of infection. [3] [4]
• Hyperreflexia, choreoathetoid movements or myoclonic jerks may be present.
• Elevated CSF protein concentrations are present in 50%, hypoglycorrhachia in 33% and pleocytosis above 20 cells per cu. Mm. In 20%.
• Peripheral blood eosinophilia may be present. [5] [6]

Respiratory tract infection:
Respiratory tract cryptococcosis may be asymptomatic, or limited to a mild productive cough with blood-streaked sputum and minor ache in the chest. [7] [8]
• Pulmonary infection may present as a single rounded lesion, lobar pneumonia, bronchiolitis obliterans [9] or miliary disease.
• Rales or pleural friction rub are unusual, and pleural effusions are uncommon.
• Pulmonary infection in immunocompetent patients may progress or regress spontaneously over long periods.
• Cryptococcosis among patients with AIDS often presents as a solitary cavitary pulmonary nodule. [10]
• Concurrent CNS infection may be evident in some cases.

One-half of AIDS patients with cryptococcal meningitis have concurrent pulmonary involvement, and two-thirds are fungemic. [11]
• Initial cough and dyspnea are found in 5 to 25% of HIV-positive patients with cryptococcosis.
• Cryptococcal immune reconstitution inflammatory syndrome may present as a clinical worsening of cryptococcal disease after initiation of antiretroviral therapy. [12]
• Case-fatality rates for treated cryptococcosis in AIDS patients are 10% to 25%.

The clinical features of *Cryptococcus neoformans var. gattii* infection are similar to those of *C. neoformans* infection. [13]
• *C. neoformans var. gattii* infections usually involve the lungs (75 percent), although neurological (8 percent) and combined (9 percent) infections are seen. [14]

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• Blindness due to high cerebrospinal fluid pressure, optic neuropathy or endophthalmitis, is relatively common among immunocompetent individuals infected with *C. gattii*. [15]

Cryptococcosis may involve a variety of other sites including skin [16-26] and soft tissues [27-30], blood stream [31] [32], mucosa, colon or intestine [33-35], gall bladder and bile ducts [36], liver, peritoneum [37-39], lymph nodes [40-42], bones and joints [43-49], breasts, pericardium, genital tract [50-52], prostate [53], placenta (without neonatal involvement) [54], adrenals [55], eyes [56] [57], parotid glands [58], tongue [59] [60], retropharyngeal space [61], etc.

The cutaneous features of cryptococcosis include papules, pustules, nodules, subcutaneous swelling, abscesses, molluscum contagiosum-like or tumor-like lesions, cellulitis, blisters, ulcers and very rarely, necrotizing fasciitis [62]
• Primary cutaneous cryptococcosis may occur in persons working with birds. [63]

Note: *Cryptococcus neoformans* is one of at least a dozen *Cryptococcus* species. See the Microbiology • Yeasts module.

This disease is endemic or potentially endemic to all countries.

Cryptococcosis in Brazil

Human infections are caused by both *Cryptococcus neoformans var. neoformans* [64] and *Cryptococcus neoformans var. gattii*. [65] [66]

Prevalence surveys:

0.4% of solid organ transplant recipients (2001 to 2006) [67]

0.62% of autopsies performed in Rio Preto, Sao Paulo (2000 to 2009) [68]

0.27% of AIDS deaths (Sao Paulo, 1993 to 2000) [69]

9.5% of infectious AIDS deaths in Uberaba (1990 to 2000) [70]

50.9% of deaths among AIDS patients (1996 to 2006) [71]

Systemic fungal diseases were found in 4.6% of AIDS patients in Cuiaba, Mato Grosso - *Cryptococcus neoformans* in 50% of these, *Cryptococcus neoformans var. gattii* 1.6%, *Cryptococcus* spp in 6.6%, *Histoplasma capsulatum* 38.3% and *Paracoccidioides brasiliensis* 3.3% (2005 to 2008) [72]

3% of submandibular and 4% of sublingual lymphadenopathy in advanced AIDS patients (2009 publication) [73]

4% of scrapings from tree hollows (*Caesalpinia peltophoroides* and *Anadenanthera peregrine*) (Alfenas, 2007 publication) [74]

25.53% (*Cryptococcus neoformans var. grubii*) of Passerine and Psittacine excreta samples (Parana, 2008 publication) [75]

21.3% of pigeon (*Columba livia*) droppings (*Cryptococcus neoformans var. neoformans*, Forteleza, Ceara, 2009 publication)

26.9% of pigeon excreta in Pelotas, Rio Grande do Sul (2010 publication) [76]

21.4% of dust samples from books in libraries (Cuiaba, Mato Grosso, 2012 publication) [77]

84.5% of persons diagnosed with cryptococcosis at a university hospital were HIV-positive; 89.6% were *Cryptococcus neoformans var. neoformans* and 10.4% *Cryptococcus neoformans var. gattii* (Mato Grosso do Sul, 1995 to 2005) [78]
- 58.7% of patients with cryptococcosis were found to be HIV-positive. *Cryptococcus neoformans* accounted for 86.5% of CSF *Cryptococcus* isolates from HIV-positive patients; while *Cryptococcus gattii* accounted for 80.8% of isolates from HIV-negative patients (Piaue, 2008 to 2010) [79]

35 cases were reported from a single institution in Minas Gerais, 11.4% of which were var. gattii (2008 publication) [80]

Cryptococcosis was the cause of death in 5% of autopsied AIDS patients (Amazonas, 1996 to 2003) [81]

Review of Brazilian publications on cryptococcosis - see reference [82]

***Cryptococcus* strains:**
- *Cryptococcus neoformans var. neoformans* accounted for 83.3% of clinical isolates and *C. neoformans var. gattii* for 15.8% (2000 publication) [83]
- *C. neoformans var. gattii* accounted for 4.8% of clinical isolates and *C. neoformans* var. *grubii* accounted for 95.2% of clinical isolates and 100% of environmental isolates (Goiania, 2009 publication) [84]
- *C. neoformans* accounted for 76% of clinical isolates during 2006 to 2008; *C. gattii* for 23% (Amazonas) [85]
- *C. neoformans var. gattii* infection has been reported in AIDS patients. (Rio Grande do Sul, 1999 publication) [86]
- An outbreak of *Cryptococcus neoformans var. gattii* infection has been reported among psittacine birds in Sao Paulo. [87]
- *C. neoformans var. gattii* has been identified in trees in Espirito Santo (2008 publication) [88]

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Cryptosporidiosis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Eimeriida: Cryptosporidium hominis and C. parvum (rarely C. muris, felis, meleagridis, et al).
Reservoir	Mammal (over 150 species)
Vector	None
Vehicle	Water Feces Oysters Fly
Incubation Period	5d - 10d (range 2d - 14d)
Diagnostic Tests	Stool/duodenal aspirate for acid-fast, direct fluorescence staining, or antigen assay. Nucleic acid amplification
Typical Adult Therapy	Stool precautions. Nitazoxanide 500 mg PO BID X 3 days
Typical Pediatric Therapy	Stool precautions. Nitazoxanide: 1 to 3 years: 100 mg PO BID X 3 days 4 to 11 years: 200 mg PO BID X 3 days >12 years: 500 mg PO BID X 3 days
Clinical Hints	Watery diarrhea, vomiting, abdominal pain; although self-limited in healthy subjects, this is a chronic and wasting illness and may be associated with pulmonary disease among immunosuppressed (e.g., AIDS) patients.
Synonyms	Cryptosporidium, Cryptosporidium andersoni, Cryptosporidium fayeri, Cryptosporidium felis, Cryptosporidium hominis, Cryptosporidium parvum, Cryptosporidium tyzzeri, Cryptosporidium ubiquitum, Cryptosporidium viatorum, Kryptosporidiose. ICD9: 007.4 ICD10: A07.2

Clinical

Cryptosporidiosis affects the gastrointestinal tract and may be asymptomatic or associated with watery diarrhea and abdominal cramps.
• Fever and anorexia are uncommon, and fecal leukocytes are not seen.
• Although vomiting is not common among adults, it is often encountered in children. [1]

Rare instances of pulmonary infection have been reported. [2] [3]

There is some evidence that *Cryptosporidium hominis* infection in children is associated with diarrhea, nausea, vomiting, general malaise, and increased oocyst shedding intensity and duration.
• In contrast, infections caused by *C. parvum*, *C. meleagridis*, *C. canis*, and *C. felis* are associated with diarrhea only.

Illness persists for 1 to 20 days (mean 10) in immunocompetent individuals
• Protracted, severe diarrhea leading to malabsorption, dehydration, extraintestinal (ie, biliary or pulmonary [4-6]) and fatal infection may develop in immunocompromised individuals. [7] [8]

This disease is endemic or potentially endemic to all countries.

Cryptosporidiosis in Brazil

Prevalence surveys:

3.6% of the Mapuera community in Oriximina, Para State (2009 publication) [9]
8.1% of persons in Campos do Jordao (2003 to 2004) [10]
5% to 17% of childhood diarrhea; however, 100% of children are seropositive by age 12 months
18.7% of childhood diarrhea in Goiania, Goias (1998 to 1999)
3.2% of urban children with diarrhea in Salvador (2000 to 2002) [11]
20.3% of asymptomatic children ages 0 to 6 years in a day care center in Sao Paulo (2006 publication) [12]
15.5% (2002) and 3.7% (2003) of children at 5 municipal daycare centers (Botacatu, Sao Paulo, 2002 to 2003) [13]
32.4% of children (ages 1 to 14 years) attending a public day-care center (Recife, 2006 to 2007) [14]
3.7% of children in day care centers in Sao Paulo (2003) [15]
9.3% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [16]

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1.0% of elderly residents at long-term residency institutions in southeastern Brazil (2013 publication) [17]
4% of hemodialysis patients (Parana, 2006 to 2007) [18]
8.8% to 9.5% of institutionalized dogs in Sao Paulo (2006 publication) [19]
15% of HIV-positive patients with diarrhea, and 5% of control patients (southeastern Brazil, 2006 to 2007) [20]
9.4% of fecal samples from HIV/AIDS patients in the Triangulo Mineiro region (1993 to 2003) [21]
8.1% of HIV-positive patients before HAART vs. 0% after HAART (Ceara, 2008 publication) [22]
0.3% of HIV-positive patients in northeastern Sao Paulo (1998 to 2008) [23]
3% of cattle, 10.7% of dogs, 0% of horses, sheep and pigs (Teodoro Sampaio municipality, 2010 publication) [24]
10.7% of cattle in Sao Paulo State (2011 publication) [25]
21.4% of foals and 18.4% of mares in Aracatuba, Birigui, Guararapes and Santo Antonio do Aracangua, Sao Paulo (2010 to 2011) [26]
8% of cats (2012 publication) [27]
52.2% of fecal samples from capybara (*Hydrochoerus hydrochaeris*) (Sao Paulo, 2007 publication) [28]
6.8% of exotic birds sold in markets, commercial aviaries, and pet shops in Rio de Janeiro (2011 publication) [29]
50.0% of gill pools of cockles (*Tivela mactroides*) and 10.0% of gill pools of oysters (*Crassostrea rhizophorae*) in coastal Sao Paulo (2005 to 2006) [30]
14.3% of river water in the Ivai Indigenous Land (2008 publication) [31]
7.6% of water samples from watershed catchments and treated water sources (2010 publication) [32]
2.5% of water sources in Sao Paulo (2004 publication) [33]
30% of water samples in Sao Paulo (2005 to 2006) [34]
9.2% of surface water samples in Sao Paulo (2012 publication) [35]
62.5% of surface water samples in Campinas (2005 to 2006) [36]

The following species of *Cryptosporidium* had been reported in humans and/or animals in Brazil as of 2010: *C. bovis*, *C. canis*, *C. felis*, *C. baileyi*, *C. galli*, *C. meleagridis*, *C. parvum* and, *C. hominis*. [37]

36.9% of slum children in northeastern Brazil acquire Cryptosporidia in stool during the first 5 years of life - *C. hominis* more often than *C. parvum*. [38]

Cryptosporidiosis is the most common cause of diarrhea among HIV-positive children in Southeastern Brazil (Sao Paulo State) [39]

Notable outbreaks:

2006 (publication year) - An outbreak (13 cases) of cryptosporidiosis was reported in a day care center in Sao Paulo. [40]

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Cutaneous larva migrans

Agent	PARASITE - Nematoda. Phasmidea: Ancylostoma braziliense, A. caninum, Bunostomum phlebotomum, Strongyloides myopotami
Reservoir	Cat Dog Cattle
Vector	None
Vehicle	Soil Contact
Incubation Period	2d - 3d (range 1d - 30d)
Diagnostic Tests	Biopsy is usually not helpful.
Typical Adult Therapy	Albendazole 200 mg BID X 3d OR Ivermectin 200 micrograms/kg as single dose. OR Thiabendazole topical, and oral 25 mg/kg BID X 5d (max 3g).
Typical Pediatric Therapy	Albendazole 2.5 mg/kg BID X 3d OR Ivermectin 200 micrograms/kg once OR Thiabendazole topical, and oral 25 mg/kg BID X 5d (max 3g).
Clinical Hints	Erythematous, serpiginous, pruritic advancing lesion(s) or bullae - usually on feet; follows contact with moist sand or beach front; may recur or persist for months.
Synonyms	Creeping eruption, Pelodera, Plumber's itch. ICD9: 126.2,126.8,126.9 ICD10: B76.9

Clinical

Cutaneous larva migrans is characterized by one or more erythematous linear, vesicular or bullous [1] lesions which tend to be raised and palpable. [2-4]

- The lesions are intensely pruritic and extend in length from day to day. [5]
- The site of the lesions reflects contact with sand / soil, as from walking barefoot or lying on a beach. [6]
- Infection may persist for over one year. [7]

This disease is endemic or potentially endemic to all countries.

Cutaneous larva migrans in Brazil

Prevalence surveys:

1.5% of a representative sample of the poor urban population (incidence 18,410 per 100, 000 per year). Prevalence varied from 3.1% in the rainy season to 0.2% in the dry season. (Fortaleza, northeast Brazil, 2003 publication) [8]

1.7% of persons in a rural region of northeastern Brazil during the rainy season, and 4.4% in the dry season (2008 publication) [9]

0.8% of European travelers exiting northern Brazil (2007 publication) [10]

23.6% of dogs in Sao Paulo state are infested by *Ancylostoma* spp. (2002 publication) [11]

19.4% of dogs in Minas Gerais State (*Ancylostoma caninum*, 2012 publication) [12]

87.8% of dogs and 94.2% of cats in Adradina Municipality, Sao Paulo (*A. caninum* and *A. braziliense*, 2011 publication) [13]

95.7% of stray dogs in Fortaleza (2010 publication) [14]

70.9% of fecal samples of stray dogs from Itapema City, Santa Catarina (2005 publication) [15]

71.3% of dog feces from central area of Cassino beach, Rio Grande do Sul (2003 publication) [16]

47.9% of fecal samples from dogs in public squares in Itabuna, Bahia (2008 publication) [17]

65.9% of cats of the metropolitan region of Rio de Janeiro (2004 publication) [18]

45.9% of soil samples from seafront gardens in Sao Paulo State (2005 publication) [19]

30% of beach sand samples from Alto beach, Pernambuco (2009 publication) [20]

15.0% of beach sand samples from Santos (*Ancylostoma* sp, 2004 to 2005) [21]

Children in Taciba (Sao Paulo) have been found to acquire cutaneous larva migrans from sand in public squares. [22]

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Hookworm-related cutaneous larva migrans was detected in 4.4% of village dwellers during the rainy season, and 1.7% during the dry season; prevalence was highest (14.9%) among children below 5 years of age. (North-Eastern Brazil, 2008 publication) [23]

Notable outbreaks:

1984 (publication year) - An outbreak was reported at a nursery school in Belo Horizonte, Minas Gerais. [24]

2000 (publication year) - An outbreak (6 cases) was reported among school children in Campo Grande, Mato Grosso do Sul. [25]

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Cyclosporiasis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Eimeriida: Cyclospora cayetanensis
Reservoir	Human ? Non-human primate
Vector	None
Vehicle	Water Vegetables
Incubation Period	1d - 11d
Diagnostic Tests	Identification of organism in stool smear. Cold acid fast stains and ultraviolet microscopy may be helpful.
Typical Adult Therapy	Sulfamethoxazole/trimethoprim 800/160 mg BID X 7d Ciprofloxacin 500 mg PO BID X 7 d (followed by 200 mg TIW X 2 w) has been used in sulfa-allergic patients
Typical Pediatric Therapy	Sulfamethoxazole/trimethoprim 10/2 mg/kg BID X 7d
Clinical Hints	Watery diarrhea (average 6 stools daily), abdominal pain, nausea, anorexia and fatigue lasting up to 6 weeks (longer in AIDS patients); most cases follow ingestion of contaminated water in underdeveloped countries.
Synonyms	Cryptosporidium muris, Cyanobacterium-like agent, Cyclospora. ICD9: 007.5 ICD10: A07.8

Clinical

Symptoms appear abruptly in 68% of cases
• Patients usually present with intermittent watery diarrhea, with up to eight or more stools per day. [1] [2]
• Other symptoms may include anorexia, nausea, abdominal cramps, bloating, flatulence, mild to moderate weight loss, fatigue, and myalgia.
• Fever is rare.

In the immunocompetent patient, the diarrhea may last from a few days to up to three months, with the organism detectable in the stool for up to two months.
- In immune compromised individual, particularly AIDS patients, the disease can persist for weeks to several months.

Reactive arthritis syndrome (Reiter's syndrome) has been associated with progression of the disease. [3]

Acalculous *Cyclospora* cholecystitis has been demonstrated in a patient with AIDS.

This disease is endemic or potentially endemic to all countries.

Cyclosporiasis in Brazil

Prevalence surveys:

0.7% of stools examined in a university parasitology laboratory. (Sao Paulo, 2007 publication) [4]
10.8% of the Mapuera community in Oriximina, Para State (2009 publication) [5]

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Cysticercosis

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Taeniidae: Taenia solium
Reservoir	Pig Human
Vector	None
Vehicle	Soil (contaminated by pigs) Fecal-oral Fly
Incubation Period	3m - 3y
Diagnostic Tests	Serology (blood or CSF) and identification of parasite in biopsy material.
Typical Adult Therapy	Albendazole 400 mg PO BID X 30d. OR Praziquantel 30 mg/kg TID X 14d (15 to 30d for neurocysticercosis). Surgery as indicated Add corticosteroids if brain involved.
Typical Pediatric Therapy	Albendazole 15 mg/kg PO BID X 30d. OR Praziquantel 30 mg/kg TID X 14d (15 to 30d for neurocysticercosis). Surgery as indicated Add corticosteroids if brain involved.
Clinical Hints	Cerebral, ocular or subcutaneous mass; usually no eosinophilia; calcifications noted on X-ray examination; lives in area where pork is eaten; 25% to 50% of patients have concurrent *Taenia* infestation.
Synonyms	Taenia crassiceps. ICD9: 123.1 ICD10: B69

Clinical

Cysticercosis is manifest as painless, rubbery (average 2 cm) nodules in skin and soft tissues, or other body sites. [1-3]
• "Rice grain" calcifications are often visible on routine roentgenograms of soft tissue, notably the pelvis and upper legs.
• Virtually any area of the body may be affected. [4-24]
• Cysticercosis involving the subcutaneous tissues may mimic malignancy or tuberculous lymphadenitis. [25] [26]
• Rare instances of cysticercosis are reported in infants and young children. [27] [28]

Central nervous system infection may present as seizures, increased intracranial pressure or hydrocephalus [29-31], altered mental status, eosinophilic meningitis [32], ventriculitis [33], focal neurological defects, stroke [34], intramedullary [35-38] or extramedullary spinal mass [39-42], quadriplegia [43], spinal subarachnoid infection [44] or encephalitis. [45-47]
• In humans, cysticerci are more frequently located in the ventricles and subarachnoid space at the base of the brain, while in pigs, cysticerci are more frequently found in the parenchyma. [48]
• Parenchymal infestation and epilepsy are most common among children, while ventricular cysts with blockage of cerebrospinal fluid predominates among adults. [49]

The eyes are infested in 15% to 45% of patients [50] [51], usually presenting as a cyst in the vitreous cavity. [52] [53], less commonly the anterior chamber. [54]
• The first ophthalmologic signs of cysticercosis are papilledema, pupillary abnormalities, or nystagmus. [55]
• Cysticercosis of the extraocular muscles is associated with limitation of eye movement, ptosis, proptosis and local mass. [56-61]

This disease is endemic or potentially endemic to all countries.

Cysticercosis in Brazil

Neurocysticercosis accounts for approximately 30% of epilepsy in Brazil.

811 cases were reported in Ribeirao Preto, in 1995 (54,5 per 100,000) - one of the highest rates in the world.

52 fatal cases of cysticercosis were reported from a hospital in Curitiba during 1977 to 1994, accounting for 0.8% of autopsies. [62]

Travel and Cysticercosis:

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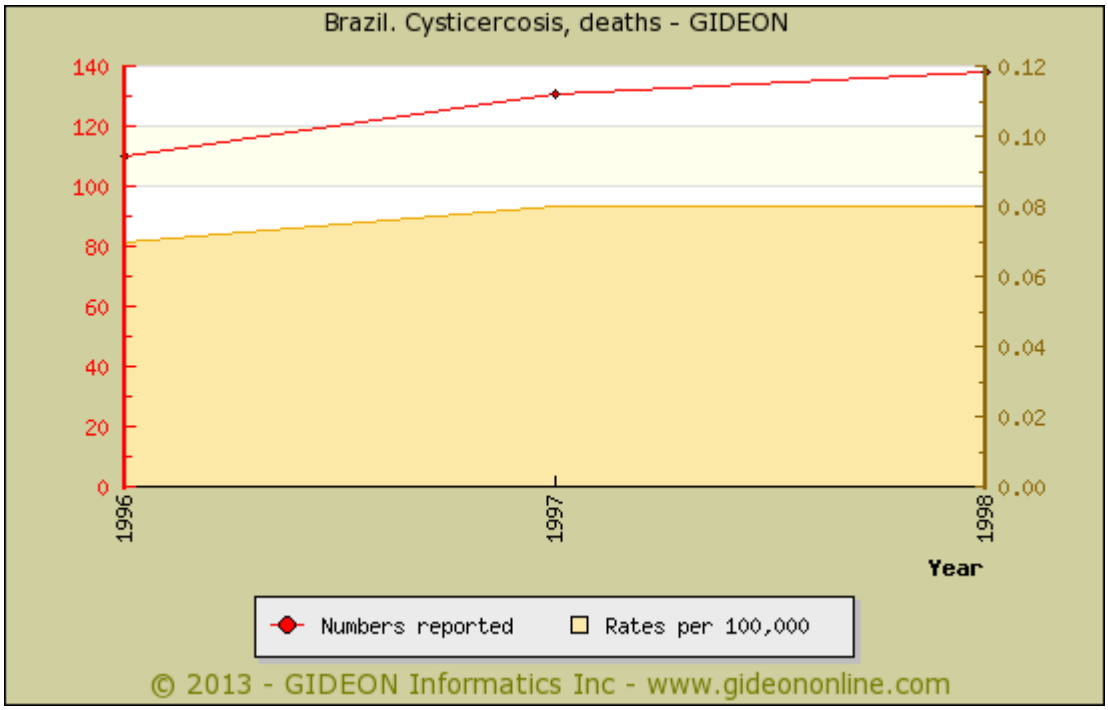
This list does not include cases reported among immigrants.
1995 (publication year) - Two Dutch travelers acquired neurocysticercosis - one during travel in Kenya, Thailand and Brazil; and one during travel in Mozambique and Tanzania. [63]
2006 (publication year) - A German traveler acquired neurocysticercosis during travel in Mexico and Brazil. [64]

Prevalence surveys:

4.8% of autopsies performed in Uberaba, Minas Gerais (1970 to 2004) [65]
0.12% to 9% of autopsies, 0.3% to 7.5% of clinical series, and 0.68% to 5.2% of serosurveys

Seroprevalence surveys:

21.9% of periurban and rural locations of Lages, Santa Catarina (2004 to 2005) [66]
16% of epileptic patients and 22% of epileptic patients with suggestive neuroimaging signs in Sao Paulo and Santa Catarina (2006 publication) [67]
0.6% of persons in urban Sao Paulo (2009 publication) [68]



Graph: Brazil. Cysticercosis, deaths

Notes:
1. Approximately 100 cases of fatal neurocysticercosis are reported from the south and southeast each year.
2. 1,131 fatal cases were reported during 1985 to 2004 [69]

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Cytomegalovirus infection

Agent	VIRUS - DNA. Herpesviridae, Betaherpesvirinae: Human herpesvirus 5 (Cytomegalovirus)
Reservoir	Human
Vector	None
Vehicle	Droplet (respiratory) Urine Dairy products Tears Stool Sexual contact (rare) Transplacental
Incubation Period	3w - 5w (range 2w - 12w)
Diagnostic Tests	Viral culture (blood, CSF, urine, tissue). Serology. Direct viral microscopy. Nucleic acid amplification
Typical Adult Therapy	[Most cases self-limited]. Ganciclovir 5 mg/kg q12h IV X 2 to 3w. OR Foscarnet 90 mg/kg Q12h IV
Typical Pediatric Therapy	[Most cases self-limited] Ganciclovir 5 mg/kg q12h IV X 2 to 3w
Vaccine	Cytomegalovirus immunoglobulin
Clinical Hints	Heterophile-negative "mononucleosis"; mild pharyngitis (without exudate); variable lymphadenopathy and splenomegaly; retinitis in AIDS patients; pneumonia in setting of immune suppression.
Synonyms	Cytomegalovirus, Zytomegalie. ICD9: 078.5 ICD10: B25

Clinical

Acute Cytomegalovirus infection is clinically similar to infectious mononucleosis (IM), and characterized by fever, generalized lymphadenopathy and hepatosplenomegaly. [1]
• In contrast to IM, pharyngitis is uncommon in Cytomegalovirus infection.
• Cytomegalovirus infection is often identified in cases of fatal myocarditis in immunocompetent patients. [2]
• Primary CMV infection may be associated with uveitis [3] , retinitis or pneumonia [4] • even in immunocompetent patients [5] [6]
• Additional manifestations of CMV infection may include prostatitis [7] , adrenal failure [8] , protracted diarrhea [9] , esophagitis [10] , gastritis [11] , duodenitis [12] , colitis with megacolon [13] [14] , appendicitis [15] , colonic pseudotumor [16] or colonic polyposis [17] , pancreatitis [18] , myocarditis [19] and protein-loosing gastropathy (Menterier's disease). [20]
• Sexually-acquired Cytomegalovirus proctitis is characterized by rectal bleeding associated with a mononucleosis-like syndrome. [21]
• The clinical features of Cytomegalovirus colitis in AIDS patients may mimic those of amebic colitis [22] [23] or Crohn's disease. [24]
• Cases of pruritic maculo-papular exanthem due to CMV infection are reported among patients with AIDS. [25]
• Evidence for primary CMV infection is often present among infants hospitalized for wheezing. [26]
• Ocular infection may present as inflammatory ocular hypertensive syndrome (IOHS), corneal endothelitis, or retinitis with retinal necrosis. [27] [28]
• Rare instances of erythema multiforme complicating Cytomegalovirus infection have been reported. [29] [30]
• CMV / EBV co-infection may be associated with prolonged illness. [31]

Severe or fatal multisystem disease occurs is encountered in congenital infection [32-37] and infection of immune-suppressed individuals. [38-41]
• Instances of pure red-cell aplasia [42] and hemophagocytic syndrome have been reported. [43]
• Sensorineural hearing loss detected in 21% of asymptomatic and 33% of symptomatic congenital infections [44-49]
• Residual neurological damage including epilepsy is common among infants with congenital infection. [50]
• Rare instances of persistent pulmonary hypertension have been reported in infants with congenital infection. [51]

Immunocompetent persons may also develop major complications [52] , including cerebral sinus thrombosis [53] ; peripheral venous [54-62] , mesenteric [63-67] or portal vein thrombosis [68-78] , colitis [79] , transverse myelitis [80] , hemolytic anemia [81] , rhabdomyolysis [82] [83] , prostatitis [84] and cholecystitis. [85]

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This disease is endemic or potentially endemic to all countries.

Cytomegalovirus infection in Brazil

The nationwide rate for congenital infection is 100 per 100,000 live births. [86]

Prevalence surveys:

6% of presumed viral CNS infections in Ribeirao Preto (2007 publication) [87]
10% of meningitis cases in a dengue-endemic area (2011 publication) [88]
27% of vitreous viral cultures (Sao Paulo, 1987 to 2001) [89]
68.3% of transplant recipients during the first year post-transplantation (2007 publication) [90]
28.5% of adult liver transplant recipients, within 6 months of transplantation (antigenemia, 2011 publication) [91]
9.8% of liver transplant recipients (pre-transplant biopsy, 2011 publication) [92]
9.25% of liver transplant recipients during the first six months post-transplantation (CMV antigenemia, 2008 to 2009) [93]
43.3% of liver transplant recipients, within 6 months to one year (Sao Paulo, 2011 publication) [94]
51.1% of liver transplant recipients, within 6 months post-transplantation (CMV antigenemia, 2012 publication) [95]
13% of submandibular and 2% of sublingual lymphadenopathy in advanced AIDS patients (2009 publication) [96]
24.4% of HIV-positive patients with diarrhea (2011 publication) [97]
1.08% of infants in Ribeirao Preto City (congenital infection, 2009 publication) [98]

Seroprevalence surveys:

76.6% of pregnant women (Sergipe, 2009 publication) [99]
95% of postpartum women (Sao Paulo, 1999 publication) [100]
97% of pregnant women (Sao Paulo, 2012 publication) [101]
89.3% of potential solid organ donors (Santa Catarina, 2006 to 2007) [102]
96.4% of blood donors in Lages, Santa Catarina (2010 publication) [103]
94.7% of liver transplant recipients (1997 to 2007) [104]
89.4% of patients with hematologic disorders (Bahia, 2010 publication) [105]

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Dengue

Agent	VIRUS - RNA. Flaviviridae, Flavivirus: Dengue virus
Reservoir	Human Mosquito ? Monkey (in Malaysia and Africa)
Vector	Mosquito - Stegomyia (Aedes) aegypti, S. albopictus, S. polynesiensis, S. scutellaris
Vehicle	Blood (rare)
Incubation Period	5d - 8d (range 2d - 15d)
Diagnostic Tests	Viral isolation (blood). Serology. Nucleic acid amplification. Biosafety level 2.
Typical Adult Therapy	Supportive; IV fluids to maintain blood pressure and reverse hemoconcentration
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, myalgia, arthralgia, relative bradycardia, leukopenia and macular rash; dengue hemorrhagic (DHF) = dengue + thrombocytopenia and hemoconcentration; dengue shock = DHF + hypotension.
Synonyms	Bouquet fever, Break-bone fever, Dandy fever, Date fever, Dengue Fieber, Duengero, Giraffe fever, Petechial fever, Polka fever. ICD9: 061 ICD10: A90,A91

Clinical

WHO Case definitions for surveillance:
1. DENGUE FEVER:
Clinical description
• An acute febrile illness of 2-7 days duration with 2 or more of the following: headache, retro-orbital pain, myalgia, arthralgia (as many as 41% of cases [1] , rash, hemorrhagic manifestations, leucopenia.
Laboratory criteria for diagnosis • One or more of the following:
• Isolation of the dengue virus from serum, plasma, leukocytes, or autopsy samples
• Demonstration of a fourfold or greater change in reciprocal IgG or IgM antibody titers to one or more dengue virus antigens in paired serum samples
• Demonstration of dengue virus antigen in autopsy tissue by immunohistochemistry or immunofluorescence or in serum samples by EIA
• Detection of viral genomic sequences in autopsy tissue, serum or CSF samples by polymerase chain reaction (PCR)
Case classification
• Suspected: A case compatible with the clinical description.
• Probable: A case compatible with the clinical description with one or more of the following:
• Supportive serology (reciprocal hemagglutination-inhibition antibody titer >1280, comparable IgG EIA titer or positive IgM antibody test in late acute or convalescent-phase serum specimen).
• Occurrence at same location and time as other confirmed cases of dengue fever.
• Confirmed: A case compatible with the clinical description, laboratory confirmed.

2. DENGUE HEMORRHAGIC FEVER:
A probable or confirmed case of dengue and hemorrhagic tendencies evidenced by one or more of the following:
• Positive tourniquet test (sensitivity questioned • see reference [2])
• Petechiae, ecchymoses or purpura
• Bleeding: mucosa, gastrointestinal tract, injection sites or other
• Hematemesis or melena
• And thrombocytopenia (100 000 cells or less per mm3)
• And evidence of plasma leakage due to increased vascular permeability, manifested by one or more of the following:
• 20% rise in average hematocrit for age and sex
• 20% drop in hematocrit following volume replacement treatment compared to baseline
• signs of plasma leakage (pleural effusion, ascites, hypoproteinemia)

3. DENGUE SHOCK SYNDROME:
All the above criteria, plus evidence of circulatory failure manifested by rapid and weak pulse, and narrow pulse pressure (<=20 mm Hg) or hypotension for age, cold, clammy skin and altered mental status.

CDC case definition:

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For surveillance purposes, the U.S. Centers for Disease Control (CDC) case definition of dengue fever consists of "acute febrile illness characterized by frontal headache, retro-ocular pain, muscle and joint pain, and rash."
• The initial fever rises rapidly and lasts for two to seven days.
• Occasionally "saddleback" fever pattern is evident, with a drop after a few days and rebound within 24 hours. [3] Relative bradycardia is common. [4]
• Conjunctival injection and pharyngeal inflammation may occur as well as lymphadenopathy.
• Rash occurs in up to 50 percent of patients, either early in the illness with flushing or mottling, or between the 2nd to the 6th day as a scarlatiniform or maculopapular rash that usually spreads centrifugally.
• The later rash usually lasts for two to three days.
• Diffuse erythema and late desquamation of hands and feet may be confused with toxic shock syndrome.
• As fever drops, petechiae may be seen.
• Additional manifestations of dengue may include post-dengue depression, acalculous cholecystitis, uveitis, retinitis and psychological depression.

Additional clinical features:
• The likelihood of encountering classic clinical findings of dengue fever increases with patient age. [5]
• The rash of dengue may be mistaken for measles or rubella. [6]
• A long time interval between attacks of dengue may actually increase the risk of dengue hemorrhagic fever. [7]
• Rare instances of encephalopathy [8] [9] , seizures, splenic rupture [10] [11] ,pancreatitis [12] myocarditis, pericarditis [13] and aplastic anemia complicating dengue are reported. [14]
• Hepatic dysfunction is often encountered [15-17] ; however, overt hepatitis is less common. [18]
• Retinal involvement may manifest as foveolitis, which can be diagnosed by funduscopy and optical coherence tomography [19]
• Prolonged post-dengue fatigue is common. [20]
• Renal failure is associated with increased mortality rates in dengue. [21]
• Risk factors for fatal dengue hemorrhagic fever among elderly patients in include male sex, chronic obstructive pulmonary disease, dengue shock syndrome and acute renal failure. [22]

The diagnosis of Dengue Hemorrhagic Fever (DHF) is defined by [23]
• thrombocytopenia (<100,000/mm3)
• evidence of plasma leakage (hematocrit increased by at least 20%) or other objective evidence of increased capillary permeability
• Dengue Shock Syndrome (DSS) consists of DHF in addition to hypotension or narrow pulse pressure (less than 21 mm Hg). [24]

Note that Leptospirosis [25] , Zika, Crimean-Congo hemorrhagic fever [26] and dengue are clinically similar, and may coexist in a given country.
• Although the clinical features of dengue and chikungunya are similar, chikungunya patients are more likely to exhibit early myalgia or arthralgia; while sore throat, cough, nausea, vomiting, diarrhea, abdominal pain, anorexia, tachycardia and thrombocytopenia will favor a diagnosis of dengue. [27]

This disease is endemic or potentially endemic to 144 countries.

Dengue in Brazil

Time and Place:
- The first epidemic of dengue in Brazil was reported in 1845.
- Dengue is most common in Alagoas, Bahia, Ceara, Minas Gerais, Pernambuco, Rio de Janeiro and Sao Paulo.

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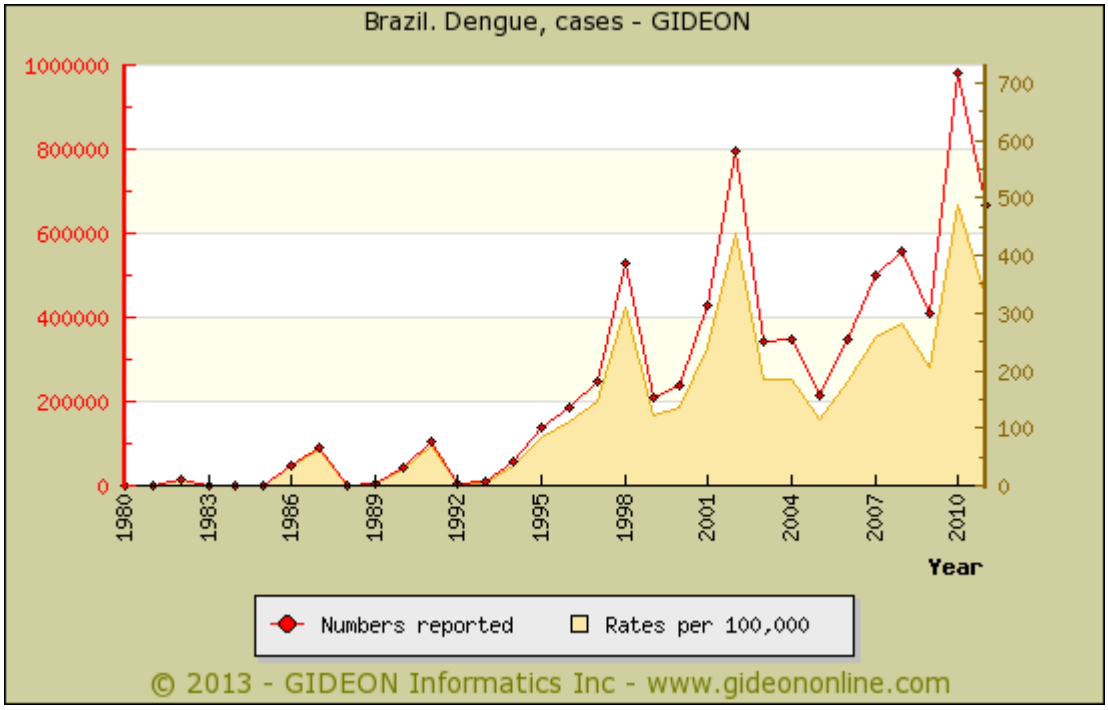

Graph: Brazil. Dengue, cases

Notes:
1. 4,243,049 cases of dengue were reported in Brazil during 1981 to 2006 - including 5,817 DHF and 338 fatal cases. [28]
2. 2,336,751 cases were reported during 1980 to 2001; 294,419 during 1986 to 1993; 2,826,948 during 1994 to 2002. [29]
3. Review of dengue in Rio de Janeiro State during 1986 to 1998 - see reference [30]
   Individual years:
   1986 - 1,000,000 cases were estimated for Rio de Janeiro alone that year.
   1995 - 57% from Rio de Janeiro and Bahia States.
   1997 - 7.4% of cases reported from the northeast.
   1998 - Brazil accounted for 72.2% of all cases in the Latin American region.
   1999 - The disease rate in Roraima was 1,418.39 per 100,000.

Disease rates per 100,000 in the Amazon region: 283.8 in 2001; 173.0 in 2002; 199.6 in 2003; 106.6 in 2004; 164.4 in 2005; 147.9 in 2006. [31]

Geographical notes:
- Alagoas State reported 54,200 cases in 2009 [32] ; 56,384 cases (19 fatal) during January to November, 2010 [33-36] ; 10,647 (10 fatal) during January to December 2011 [37-47] ; 23,000 (12 fatal) during January to July, 2012. [48-55]
- Amazonas State - outbreaks were reported during 1998 to 1999 [56] ; 208 to 2009 [57] , 2010 (3,200 cases to November) [58] ; 56,552 cases (12 fatal) during January to October. [59-68]
- Bahia reported outbreaks in 1987 [69] ; 1994 [70] and 1997 ; 39,972 cases (14 fatal) in 2008 - including 3,600 (1 fatal) in Salvador [71] [72] ; 103,788 (66 fatal) during January to August, 2009. [73-81] ; outbreak activity in 2010 [82] ; 50,000 during January to December, 2011 [83-86] ; 68,414 (27 fatal) during January to December 2012. [87-91]
- Brasilia reported 274 locally-acquired cases in 2008; 271 during January to October 2009 [92] ; 1,956 (2 fatal) during January to March, 2010 [93] [94] ; 638 cases during January to February, 2013. [95]
- Ceara State reported 47,798 cases in 1986 [96] ; 47,889 cases (25 DHF, 12 fatal) in 1994 [97] [98] ; 172 DHF (15 fatal) in 2006 [99] ; 24,793 (298 DHF, 10 fatal) in 2007. [100] ; 44,508 (436 DHF, 31 fatal) in 2008 - including 30,622 cases in Fortaleza [101-105] ; 4,513 (17 fatal) during January to October, 2009 [106-108] ; 13,196 (21 fatal) in 2010 [109] [110] ; 50,593 (55 fatal) during January to September, 2011 [111-127] ; 46,489 (26 fatal) during January to August, 2012. [128-136]
- Espirito Santo reported 43,536 cases (133 fatal) during January to May, 2009 [137-139] ; 32,913 cases (14 fatal) during January to September 2010 [140-142] ; 51,087 (25 fatal) during January to November 2011 [143-146] ; 1,614 cases in January 2013. [147]
- Goias reported 115,000 cases in 2009 [148] [149] ; 102,726 cases (87 fatal) in 2010 [150-155] ; 41,00 (24 fatal) during January to December 2011 [156] [157] ; 20,793 cases during January to February, 2013. [158]

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- Mato Grosso State reported 10,489 cases during January to August 2008; 58,410 (57 fatal) in 2009 [159-168] ; 44,762 (65 fatal) during January to December, 2010. [169-181] ; 40,391 (6 fatal) during January to July, 2011 [182] ; 43,158 (22 fatal) during January to December 2012. [183-198]
- Mato Grosso do Sul reported 1,800 cases during January to November 2008; 2,500 (17 DHF, 6 fatal) during January to December 2009 [199-201] ; 81,863 (47 fatal) during January to December, 2010 [202-206] ; 12,840 (3 fatal) during January to July, 2011 [207-210] ; 16,506 in 2012 [211-220] ; 7,000 cases in January 2013. [221]
- Minas Gerais reported 7,875 cases (5 DHF) in 2007 [222] ; 42,880 (104 DHF, 11 fatal) in 2009 [223-228] ; 220,121 (66 fatal) during January to July, 2010 [229-239] ; 54,588 during January to August 2011 [240-244] ; 4,000 during January to February 2012 [245] ; 29,517 cases during January to Febuary, 2013. [246]
- Para State: 17,440 cases were reported in Belem during 1996 to 1997 [247] ; 925 during January 2011 [248-250] ; 9,419 (3 fatal) during January to July, 2012. [251-253]
- Paraiba State reported 709 cases (1 fatal) were reported in 2009; 4,391 (3 fatal) during January to October 2010 [254] ; 7,230 (7 fatal) during January to September 2011 [255] ; 9,799 (8 fatal) during January to November 2012. [256-263]
- Parana State reported 59,195 cases were reported during January to September, 2010 [264-266] ; 25,099 (14 fatal) during January to June 2011. [267-280] ; 1,913 (1 fatal) during January to August 2012. [281-284] ; 3,439 during January to February, 2013. [285] [286]
- Pernambuco State reported 33,247 clinical cases (10,092 confirmed, 13 fatal) in 2007 [287] ; 39,332 (249 DHF, 59 fatal) during January to July 2010 [288-292] ; an outbreak in 2012. [293]
- Piaui State reported 7,719 cases (6 fatal) during January to November 2010 [294] [295] ; 5,542 during January to March 2011; 13,992 (5 fatal) during January to August 2012. [296-298]
- Rio de Janeiro State reported 92,000 cases during 1986 to 1987 [299-302] ; 85,891 in 1991 [303] ; 51,465 during 1995 to 1996; 155,242 during 2001 to 2002 [304-307] ; 31,054 in 2006; 66,553 (31 fatal) in 2007 [308-310] - including 25,000 (27 fatal) in Rio de Janeiro City [311] ; 248,609 (1,761 DHF, 181 fatal) in 2008 - including 124,203 (105 fatal) in Rio de Janeiro City [312-319] ; 27,885 (39 fatal) during January to December, 2010 [320-324] ; 165,764 (137 fatal) during January to December 2011. [325-352] ; 112,143 (22 fatal) during January to May, 2012. [353-365]
- Rio Grande do Norte reported an epidemic in 1997 [366] ; 37,147 cases (1,161 DHF, 63 fatal) during January to July 2008 [367] ; 25,131 (6 fatal) during January to May 2011. [368-371] ; 24,165 (5 fatal) during January to July 2012. [372-374]
- Rio Grande do Sul reported an outbreak in 1916 [375] ; 3,128 cases during January to March 2010 [376] [377] ; 483 during January to March 2011. [378-382]
- Rondonia reported 22,689 cases (15 fatal) in 2009 [383] [384] ; outbreaks in 2010 [385] ; 3,099 cases in January 2013. [386]
- Roraima experienced an outbreak during the 1980's [387] [388] ; 3,120 cases (61 DHF) in 2009 [389] ; 6,383 cases, 77 DHF (8 fatal) during January to September 2010 [390-394] ; 1,876 in 2012. [395] [396]
- Sao Paulo reported 21,891 cases during 1986 to 1996 [397] [398] ; 847 in 1997; 4,134 in 1998; 4,567 during the first half of 1999; 51,700 in 2001 [399] ; 50,027 cases (6 fatal) in 2006 [400] ; 82,912 in 2007 [401] ; 185,966 (120 fatal) during January to June 2010. [402-423] ; 55,100 (21 fatal) during January to June, 2011. [424-446] ; 19,929 during January to July 2012. [447-456]
- Sergipe reported 861 cases in 2007 [457] ; 1,130 (287 confirmed) during January to August 2009 [458] ; 1,063 (2 fatal) during January to July 2011 [459] [460] ; 5,977 (0 fatal) during January to April 2012. [461-463]

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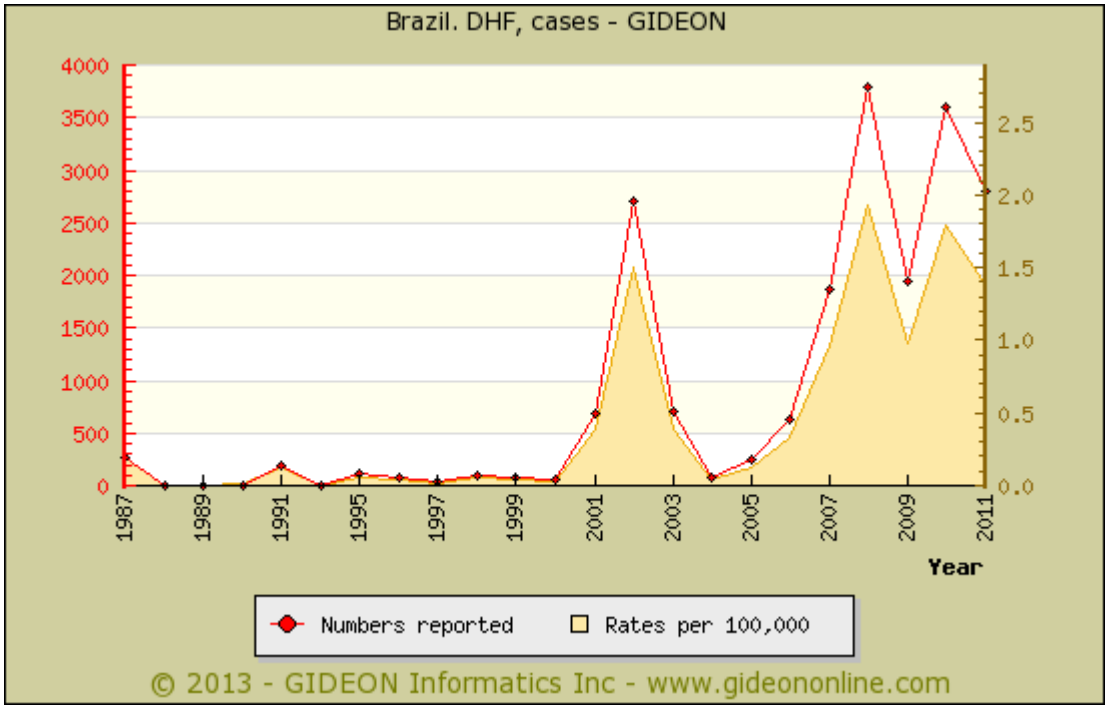
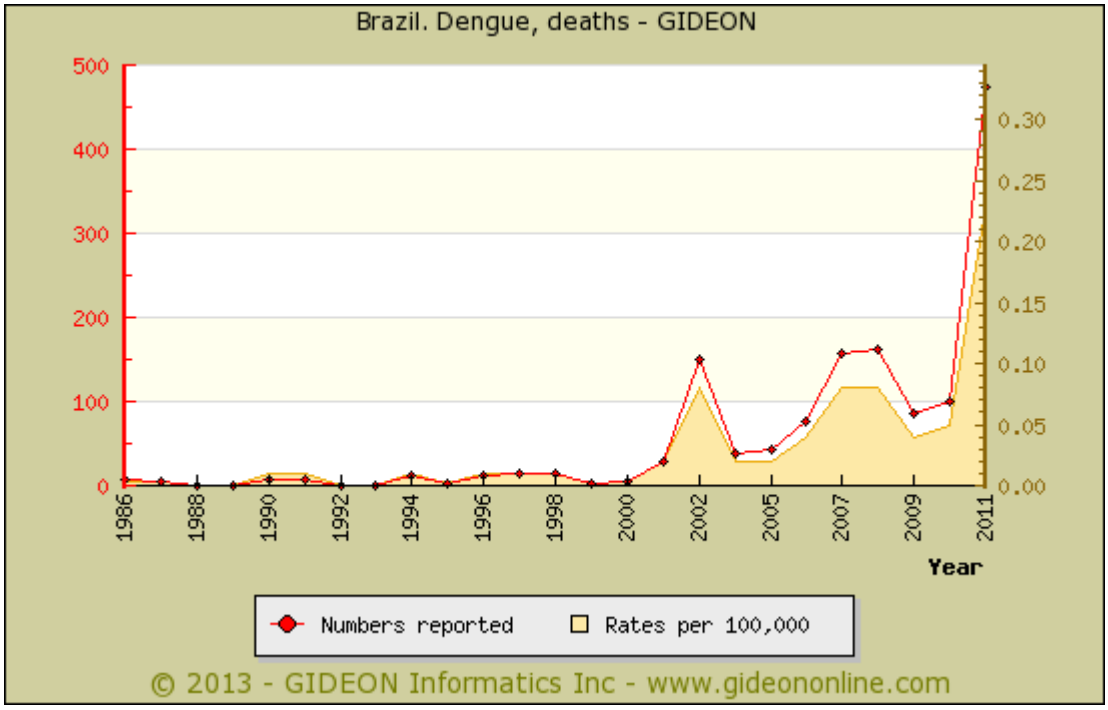


Graph: Brazil. DHF, cases

Notes:
1. A total of 696 cases of DHF (26 fatal) were reported during April 1990 to March 1997; 893 (44 fatal) during 1991 to 2000.
Individual years:
2007 - 86 in the states of Ceara, Rio, Maranhao, Pernambuco, Amazonas, Mato Grosso do Sul, Piaui, Goias, Alagoas, Paraiba, and Rio Grande do Norte. [464]



Graph: Brazil. Dengue, deaths

Notes:
Individual years:
2007 - Included 29 deaths in Rio.

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Prevalence surveys:

0.06% of donated blood units (2003) [465]
0.4% of healthy blood donors in Ribeirao Preto, Sao Paulo (2012 publication) [466]
16.9% of patients suspected to have measles and/or rubella (Pernambuco, 2001 to 2004) [467]
33.0% of patients with maculopapular rash (Niteroi, Rio de Janeiro State, 1994 to 1998) [468]
47% of encephalitis cases and 10% of meningitis cases in a dengue-endemic area (2011 publication) [469]
3.8% of patients with suspected viral meningitis/meningoencephalitis in a dengue-endemic area (2005 to 2008) [470]

Seroprevalence surveys:

5.4% of persons in Ribeirao Preto (1992) [471]
11.9% of persons in Belo Horizonte (2006 to 2007) [472]
26.6% of children ages 0 to 3 years, in Salvador (1998 to 2000) [473]

Vectors:

- *Stegomyia (Aedes) aegypti* was identified in 1,787 cities in 1995.
- As of 2003, *S. albopictus* is present in 10 American countries: Brazil, the Cayman Islands, the Dominican Republic, El Salvador, Guatemala, Mexico, Nicaragua, Panama, Trinidad and the United States.

Notable outbreaks:

2010 - An outbreak (936,260 cases, 592 fatal - during January to November) was reported. [474-478]
2011 - An outbreak (716,000 cases during January to October) was reported. [479-487]
2012 - An outbreak (565,510 cases, 3,774 DHF, 247 fatal - to November) was reported. [488-493]
2013 - Outbreaks were reported. [494-497]

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Dermatophytosis

Agent	FUNGUS. Ascomycota, Euascomyces, Onygenales: Epidermophyton, Microsporum, Trichophyton, Trichosporon spp., Arthroderma, et al
Reservoir	Human Dog Cat Rabbit Marsupial Other mammal
Vector	None
Vehicle	Contaminated soil/flooring Animal contact
Incubation Period	2w - 38w
Diagnostic Tests	Fungal culture and microscopy of skin, hair or nails. Nucleic acid amplification.
Typical Adult Therapy	Skin - topical Clotrimazole, Miconazole, etc. Hair/nails - Terbinafine, Griseofulvin, Itraconazole or Fluconazole PO
Typical Pediatric Therapy	As for adult
Clinical Hints	Erythematous, circinate, scaling or dyschromic lesions of skin, hair or nails; pruritus, secondary infection and regional lymphadenopathy may be present.
Synonyms	Arthroderma, Dermatomicose, Dermatomycose, Dermatomycosis, Dermatomykose, Dermatomykosen, Emericella, Favus, Granuloma trichophyticum, Gruby's disease, Kodamaea, Leukonychia trichophytica, Microsporum, Nattrassia, Onychocola, Onychomycosis, Pityriasis versicolor, Ringworm, Saint Aignan's disease, Scopulariopsis, Scytalidium, Tinea, Tinea barbae, Tinea capitis, Tinea corporis, Tinea cruris, Tinea favosa, Tinea imbricata, Tinea manum, Tinea pedis, Tinea unguinum, Tokelau ringworm, Triadelphia pulvinata, Trichomycosis, Trichophytosis, Trichophytosis gladiatorum. ICD9: 110,111 ICD10: B35,B36

Clinical

Dermatophytosis is characterized by indolent infection of skin, hair or nails. [1] [2]

Common findings include scaling, pruritis and discoloration • usually without overt signs of inflammation.

Tinea imbricata, a superficial mycosis caused by *Trichophyton concentricum*, an anthropophilic dermatophyte.
• The skin lesions are characteristically concentric and lamellar (imbricata: in Latin, tiled) plaques of scale. [3]
• Predisposing conditions include humidity, inheritance, and immunologic factors. [4]

Rare instances of mycetoma of the scalp due to *Microsporum canis* have been reported. [5]

This disease is endemic or potentially endemic to all countries.

Dermatophytosis in Brazil

Notable outbreaks:
1992 (publication year) - An outbreak (18 cases) of tinea capitis due to *Trichophyton tonsurans* and *Microsporum canis* was reported in an orphanage in Niteroi. [6]

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Dicrocoeliasis

Agent	PARASITE - Platyhelminthes, Trematoda. Plagiorchiida, Dicrocoeliidae: Dicrocoelium dendriticum and D. hospes
Reservoir	Sheep Cattle Pig Goat Snail Ant
Vector	None
Vehicle	Ingested ant
Incubation Period	Unknown
Diagnostic Tests	Identification of ova in stool, bile or duodenal aspirate.
Typical Adult Therapy	Praziquantel 25 mg/kg PO TID X 1d (investigational)
Typical Pediatric Therapy	As for adult
Clinical Hints	Abdominal pain, often accompanied by eosinophilia; follows inadvertent ingestion of ants (with raw vegetables or fruit) in sheep-raising areas.
Synonyms	Dicrocoelium dendriticum, Dicrocoelium hospes, Halzoun, Lancet liver fluke. ICD9: 121.8 ICD10: B66.2

Clinical

Human infection occurs after accidental ingestion of infected ants.
• Spurious infections are more frequently observed and are the consequence of the ingestion of raw or undercooked animal liver. In Lebanon, this syndrome presents as severe allergic pharyngitis reported in Lebanon (Halzoun). [1]
• Symptoms and signs of hepato-biliary involvement are usually mild and limited to hepatomegaly, bloating and abdominal discomfort. [2] [3]
• Eosinophilia is present during the early stages of infection.

This disease is endemic or potentially endemic to 39 countries.

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Dientamoeba fragilis infection

Agent	PARASITE - Protozoa. Archezoa, Parabasala, Trichomonadea. Flagellate: Dientamoeba fragilis
Reservoir	Human Gorilla Pig
Vector	None
Vehicle	Fecal-oral (? on pinworm ova)
Incubation Period	8d - 25d
Diagnostic Tests	Identification of trophozoites in stool. Nucleic acid amplification. Alert laboratory if this diagnosis is suspected.
Typical Adult Therapy	Stool precautions. Iodoquinol 650 mg PO TID X 20d. OR Tetracycline 500 mg QID X 10d. OR Paromomycin 10 mg/kg TID X 7d OR Metronidazole 750 mg PO TID X 10d
Typical Pediatric Therapy	Stool precautions. Iodoquinol 13 mg/kg PO TID X 20d. OR (age >8) Tetracycline 10 mg/kg QID X 10d OR Paromomycin 10 mg/kg TID X 7d OR Metronidazole 15 mg/kg PO TID X 10d
Clinical Hints	Abdominal pain with watery or mucous diarrhea; eosinophilia may be present; infestation may persist for more than one year.
Synonyms	

Clinical

Most infections are asymptomatic.
• Symptoms may include diarrhea, flatulence, abdominal pain, fatigue and anorexia; and may rarely mimic acute appendicitis. [1-3]
• Clinical features are similar to those of giardiasis; however, vomiting, anorexia and weight loss are less common in *Dientamoeba* infection. [4]
• The presence of abdominal pain or diarrhea in a patient with enterobiasis should suggest the diagnosis of concurrent *Dientamoeba* infection. [5]

This disease is endemic or potentially endemic to all countries.

Dientamoeba fragilis infection in Brazil

Dientamoeba fragilis infection has been identified in patients with HIV/AIDS. [6] [7]

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Dioctophyme renalis infection

Agent	PARASITE - Nematoda. Phasmidea: Dioctophyme renalis
Reservoir	Dog Mink
Vector	None
Vehicle	Food Fish which have ingested aquatic worm
Incubation Period	3m - 6m
Diagnostic Tests	Identification of ova in urine or adults in tissue.
Typical Adult Therapy	Excision as required
Typical Pediatric Therapy	As for adult
Clinical Hints	Flank pain and hematuria beginning 3 to 6 months after eating raw fish or frog flesh; subcutaneous infection has also been described.
Synonyms	Giant kidney worm. ICD9: 128.8 ICD10: B83.8

Clinical

The few reported instances of human infection have been characterized by the presence of a subcutaneous mass, or progressive destruction of the kidney.

This disease is endemic or potentially endemic to 11 countries.

Dioctophyme renalis infection in Brazil

A report of two cases of canine infection was published in 1976. [1]

Dioctophyme renalis has been identified a little grison (*Galictis cuja*) in Parana State (1990 publication) [2] ; a cat in Rio de Janeiro (2009 publication) [3] ; and in a crab-eating fox (*Cerdocyon thous*, 2008 publication) [4]

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Diphtheria

Agent	BACTERIUM. Corynebacterium diphtheriae A facultative gram-positive bacillus
Reservoir	Human
Vector	None
Vehicle	Droplet Contact Dairy products Clothing
Incubation Period	2d - 5d (range 1d - 10d)
Diagnostic Tests	Culture on special media. Advise laboratory when this diagnosis is suspected.
Typical Adult Therapy	Respiratory isolation. Equine antitoxin 20,000 to 80,000 units IM. Erythromycin 500 mg QID X 10d
Typical Pediatric Therapy	Respiratory isolation. Equine antitoxin 1,000 units/kg IM. Erythromycin 10 mg/kg QID X 10d
Vaccines	Diphtheria antitoxin Diphtheria DTP DT DTaP Td
Clinical Hints	Pharyngeal membrane with cervical edema and lymphadenopathy; or punched out skin ulcers with membrane; myocarditis or neuropathy (foot/wrist drop) appears weeks later.
Synonyms	Corynebacterium diphtheriae, Difteri, Difteria, Difterie, Difterite, Diphterie. ICD9: 032 ICD10: A36

Clinical

WHO Case definition for surveillance:
Clinical description
• An illness of the upper respiratory tract characterized by laryngitis or pharyngitis or tonsillitis, and adherent membranes of tonsils, pharynx and/or nose
Laboratory criteria for diagnosis
• Isolation of *Corynebacterium diphtheriae* from a clinical specimen.
• Note: A rise in serum antibody (fourfold or greater) is of interest only if both serum samples were obtained before administration of diphtheria toxoid or antitoxin. This is not usually the case in surveillance, where serological diagnosis of diphtheria is thus unlikely to be an issue.
Case classification
• Suspected: Not applicable.
• Probable: A case that meets the clinical description.
• Confirmed: A probable case that is laboratory confirmed or linked epidemiologically to a laboratory confirmed case.
Note: Persons with positive *C. diphtheriae* cultures who do not meet the clinical description (i.e. asymptomatic carriers) should not be reported as probable or confirmed diphtheria cases.

Faucal diphtheria:

Following an incubation period of 2 to 5 days (7 days after primary skin infection for cutaneous diphtheria), the patient presents with nonspecific symptom which may include fever and chills, malaise, sore throat, hoarseness or dysphagia, cervical edema and lymphadenopathy, rhinorrhea (mucopurulent or blood-tinged), cough, stridor, wheezing, nausea and vomiting and headache. [1]
• Respiratory diphtheria may progress rapidly to respiratory arrest from airway obstruction by a tracheobronchial pseudomembrane.
• Tachycardia, pallor, and foul breath may be present.
• The pseudomembrane is generally firm, adherent, thick, fibrinous and of a gray-brown color.
• It may occur over the palate, pharynx, epiglottis, larynx, or trachea • occasionally extending into the tracheobronchial tree.
• The area may bleed if disturbed.
• Marked edema of the tonsils, uvula, submandibular region and anterior neck ("bull neck) may be observed and may be associated with thick speech, stridor, anterior cervical lymphadenopathy, and petechial hemorrhages.

Cutaneous diphtheria:

Cutaneous diphtheria is associated with a history of a break in the skin, followed by pain, tenderness, erythema, or exudate.

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- Lesions appear as punched-out ulcers with dirty gray membranes at their margins.
- Genital ulcers may be misdiagnosed as venereal disease. [2]

Cardiac complications:
Cardiovascular signs ensue 1 to 2 weeks following the initial illness.
- Myocarditis occurs in as many as two thirds of patients, and approximately 20% develop cardiac dysfunction.
- Circulatory collapse, heart failure, atrioventricular blocks and arrhythmias may occur.
- Endocarditis and mycotic aneurysms also have been reported, typically in intravenous drug users.

Neurological complications:
Approximately 70% of patients with severe infection develop neuropathy, neuritis or motor paralysis 2 to 8 weeks following initial illness.
- Clinical and cerebrospinal fluid findings at this stage are indistinguishable from those Guillain-Barre syndrome.
- Potentially fatal paralysis of the diaphragm may ensue.
- Paralysis typically resolves completely with resolution of infection.

The neurological manifestations of diphtheria include:
- hypesthesia and paralysis of the soft palate
- weakness of the posterior pharyngeal, laryngeal, and facial nerves, resulting in a "nasal tone" to the voice, difficulty in swallowing, and occasionally aspiration
- cranial neuropathies, typically during the fifth week, leading to oculomotor and ciliary paralysis (strabismus, blurred vision, and loss of accommodation)
- symmetric polyneuropathy beginning within 10 days to 3 months after infection, and manifest as motor deficit with diminished deep tendon reflexes
- proximal muscle weakness of the extremities progressing distally (or distal weakness progressing proximally).

Other forms of diphtheria:
Other less common manifestations include infection of the genitourinary tract, gastrointestinal tract, vagina, external ear, and conjunctiva.
- Hemorrhagic conjunctivitis and dissolution of the cornea may occur. [3]
- Focal necrosis of the kidneys [4] , liver, and adrenal glands may be observed.
- Cases of septic arthritis, osteomyelitis, splenic abscesses, and bacteremia have been reported.

A rare case of diphtherial urethritis was acquired through orogenital contact. [5]

This disease is endemic or potentially endemic to all countries.

Diphtheria in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

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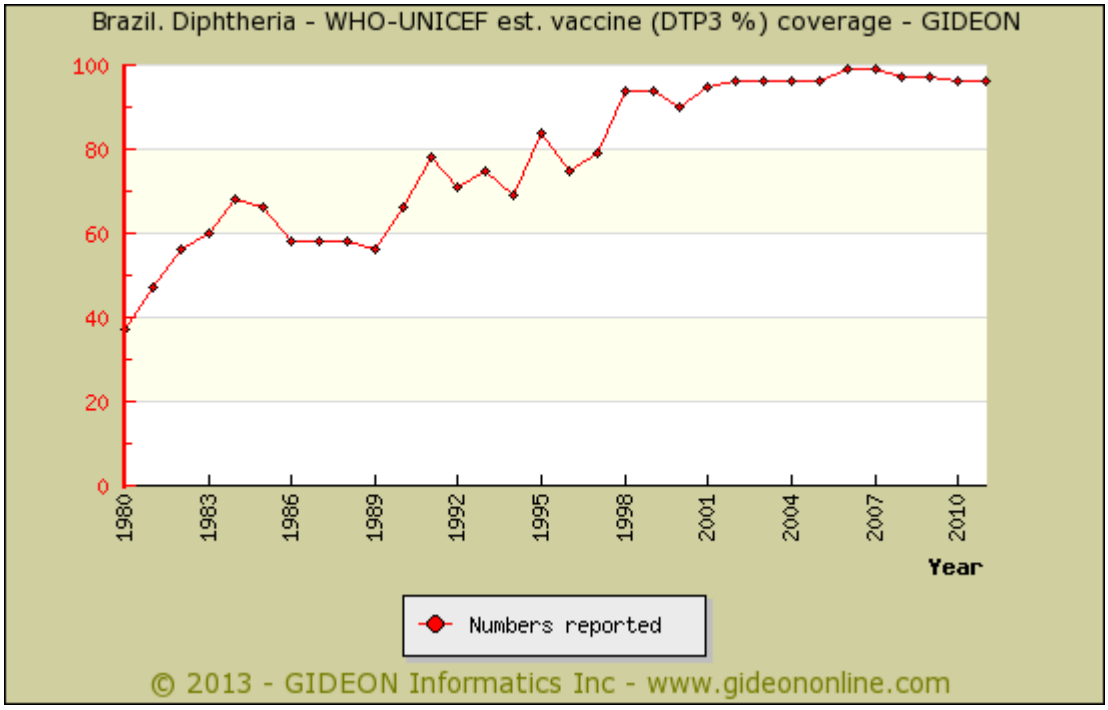


Graph: Brazil. Diphtheria - WHO-UNICEF est. vaccine (DTP3 %) coverage

Seroprevalence surveys:

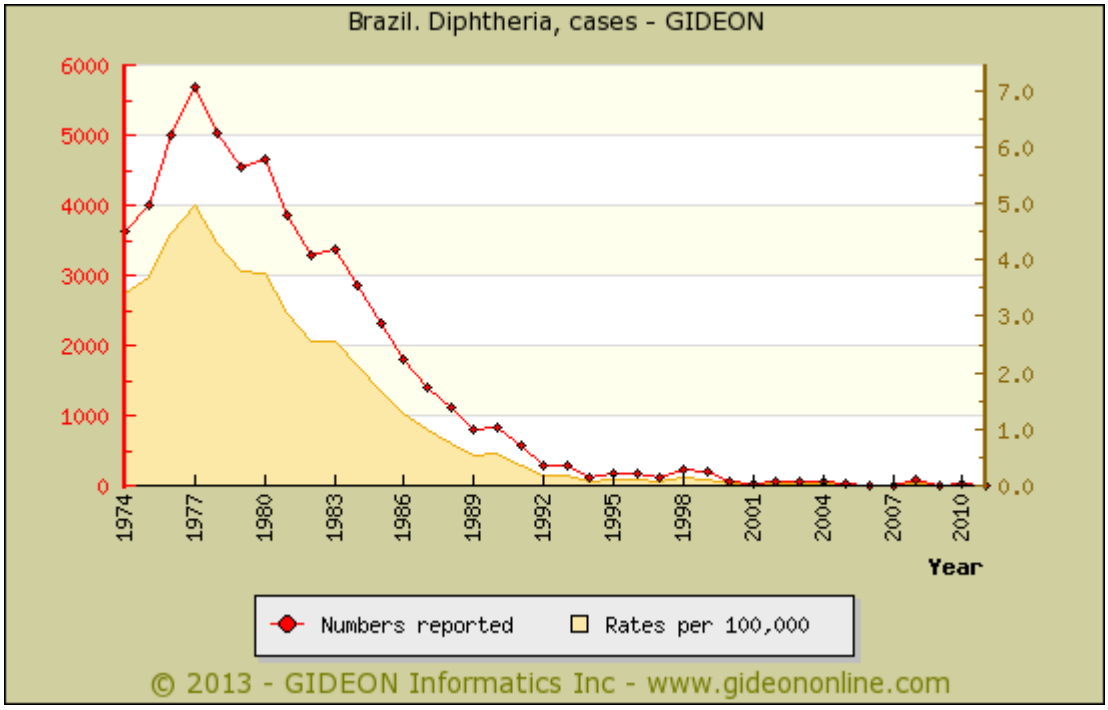
30.4% of blood donors (Rio de Janeiro, 2005 publication) [6]

69.3% of healthy adults in Rio de Janeiro (2006 publication) [7]

99.5% of adolescents. 23.1% of adolescents had not received a booster dose of diphtheria or tetanus vaccine in the previous 10 years (Sao Paulo, 2001) [8]

84% of population (Sao Paulo, 2001 to 2003) [9]

93.0% of health care workers in neonatal units (Sao Paulo, 2002) [10]



Graph: Brazil. Diphtheria, cases

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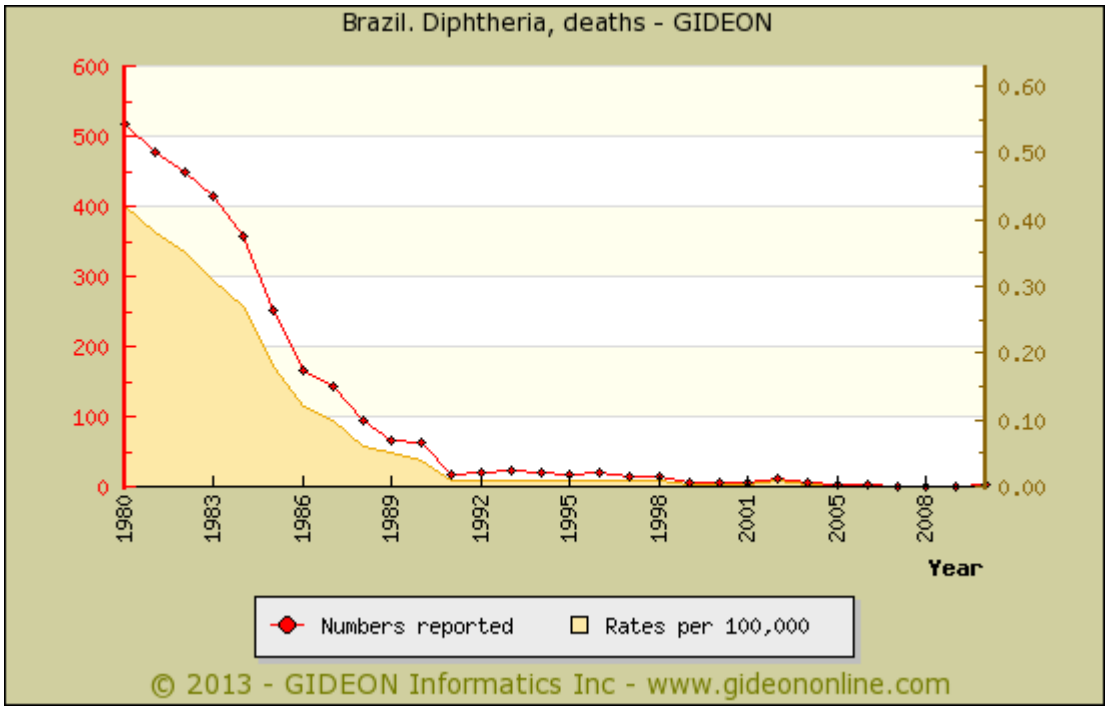
Notes:
1. 27,333 cases were reported during 1980 to 2001.
Individual years:
1995 - Highest rates in the south.



Graph: Brazil. Diphtheria, deaths

A strain of toxigenic *Corynebacterium diphtheriae* was identified in skin ulcer (2008 publication). [11]

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Diphyllobothriasis

Agent	PARASITE - Platyhelminthes, Cestoda. Pseudophyllidea, Diphyllobothriidae: Diphyllobothrium latum, et al
Reservoir	Human Dog Bear Fish-eating mammal
Vector	None
Vehicle	Fresh-water fish - notably (for D. latum) perch, burbot and pike
Incubation Period	4w - 6w (range 2w - 2y)
Diagnostic Tests	Identification of ova or proglottids in feces.
Typical Adult Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 2 g PO once
Typical Pediatric Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 50 mg/kg PO once
Clinical Hints	Abdominal pain, diarrhea and flatulence; vitamin B12 deficiency is noted in 0.02% of patients; rare instances of intestinal obstruction have been described; worm may survive for decades in human intestine.
Synonyms	Bandwurmer [Diphyllobothrium], Bothriocephalus latus, Broad fish tapeworm, Diphyllobothrium latum, Diplogonoporiasis, Fish tapeworm. ICD9: 123.4 ICD10: B70.0

Clinical

Patients may experience abdominal pain, diarrhea, weight loss, asthenia or vertigo. [1]
• Vitamin B-12 deficiency is described in cases of prolonged infestation by *Diphyllobothrium latum* [2-11] (but not other *Diphyllobothrium* species [12] [13]).

This disease is endemic or potentially endemic to all countries.

Diphyllobothriasis in Brazil

Increasing incidence in recent years may be related to ingestion of sushi or sashimi. [14] [15]

The first confirmed case report of *Diphyllobothrium latum* infection in Brazil was published in 2005 - related to ingestion of sashimi in Salvador. [16]
- A second case report was published in 2006. [17]

Notable outbreaks:

2005 - An outbreak (28 cases in Sao Paulo and 13 in Rio de Janeiro) of diphyllobothriasis was reported. [18]

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Dipylidiasis

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Dipylidiidae: Dipylidium caninum
Reservoir	Dog Cat
Vector	None
Vehicle	Flea = Ctenocephalides spp. (by ingestion)
Incubation Period	21d - 28d
Diagnostic Tests	Identification of proglottids in feces.
Typical Adult Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 2 g PO once
Typical Pediatric Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 50 mg/kg PO once
Clinical Hints	Diarrhea, abdominal distention and restlessness (in children); eosinophilia may be observed; proglottids may migrate out of anus.
Synonyms	Cucumber tapeworm, Dipylidium caninum, Dog tapeworm, Double-pored dog tapeworm. ICD9: 123.8 ICD10: B71.1

Clinical

Most infections with *Dipylidium caninum* are asymptomatic.
• Severe diarrhea, urticaria, fever and eosinophilia are occasionally encountered. [1]
• The principal sign (in animals and children) consists of the passage of proglottids on the perianal region, feces, diapers, or occasionally on floor covering and furniture.
• Infection has been reported in patients as young as four months [2] to two years. [3]
• Proglottids are motile when freshly passed and may be mistaken for maggots or fly larvae.

This disease is endemic or potentially endemic to all countries.

Dipylidiasis in Brazil

Prevalence surveys:

0.7% of dogs in Sao Paulo State (2002 publication) [4]
52.6% of cats of the metropolitan region of Rio de Janeiro (2004 publication) [5]
21.6% of cats in Andradina City, Sao Paulo (2007) [6]
1.9% of fecal samples of stray dogs from Itapema City, Santa Catarina (2005 publication) [7]
2.4% of dogs in Botacatu, Sao Paulo (2008 publication) [8]
36.8% of dogs in Minas Gerais State (2012 publication) [9]
45.7% of stray dogs in Fortaleza (2010 publication) [10]

Dipylidium caninum has been identified in a crab-eating fox (*Cerdocyon thous*) in Brazil. [11]

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Dirofilariasis

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Dirofilaria (Nochtiella) immitis (pulmonary); D. tenuis & D. repens (subcutaneous infection) & D. ursi
Reservoir	Mammal Dog Wild carnivore (D. tenuis in raccoons; D. ursi in Bears)
Vector	Mosquito
Vehicle	None
Incubation Period	60d - 90d
Diagnostic Tests	Identification of parasite in tissue (ie, lung biopsy). Serologic tests available in some centers.
Typical Adult Therapy	Not available; excision is often diagnostic and curative
Typical Pediatric Therapy	As for adult
Clinical Hints	Most patients are asymptomatic; occasional instances of cough and chest pain, with solitary pulmonary coin lesion; or multiple tender subcutaneous nodules; eosinophilia usually not present.
Synonyms	Candidatus Dirofilaria hongkongensis, Dirofilariosis, Dirofiliaria, Dog heartworm, Filaria conjunctivae, Loaina, Pelecitus. ICD9: 125.6 ICD10: B74.8

Clinical

Pulmonary infections usually present as a well-circumscribed coin lesion. [1]
• Occasionally the lesions are transient or multiple. [2]
• Symptoms such as chest pain, dyspnea, fever, cough and eosinophilia are present in only 50% of cases.
• Isolated infections have been reported in the mesentery, spermatic cord, epididymis [3] , peritoneal cavity [4] , anterior chamber of the eye [5] , orbital muscles [6] and liver.
• Lesions may suggest malignancy [7] , and coexistence of dirofilariasis and lung cancer has been reported. [8]
• In rare cases pulmonary cavitation may occur [9]

Skin and subcutaneous infections are caused by *D. tenuis*, *D. repens* [10] , *D. ursi*, *D. immitis* and *D. striata*.
• Clinical manifestations are limited to a small (0.5 to 1.5 cm) discrete nodule which may appear on any area of the body. [11-13]
• Local pain, inflammation, eosinophilia and a sensation of motion may be present in some cases.
• Rare instances of scrotal pseudotumor are reported. [14]

A novel *Dirofilaria* species ("*Candidatus Dirofilaria hongkongensis*") has been identified as a cause of human (cervical lymphadenopathy, abdominal subcutaneous mass and subconjunctival nodule) and canine infection in Hong Kong. [15]

This disease is endemic or potentially endemic to 228 countries.

Dirofilariasis in Brazil

Cases of human infection are reported. [16]
- 17 cases were published from Brazil to 1995 [17] ; however, 24 cases were documented at a single center in Sao Paulo during 1982 to 1996. [18] [19]

Prevalence surveys:

12.8% of dogs on Maranhao Island (State of Maranhao) (1991 to 1994) [20]
15% of dogs on Santa Catarina Island (2003 publication) [21]
58% of dogs below age 2 and 100% of older dogs on Marajo Island, Para (2004) [22]
12% of dogs on the southern coast of Alagoas (1995 to 1999) [23]
5% of dogs and 0.8% to 5% of cats in metropolitan Rio de Janeiro (1997 publication) [24]
1% of dogs in Recife (1999 publication) [25]

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5.47% of dogs on the coast of Parana State (1998 to 1999) [26]
7.9% of dogs, nationwide in 1988; 2% in 2001 [27]
32.45% of domestic dogs on Marajo Island (2009 publication) [28]
0% of dogs in Engenho Novo, Rio de Janeiro (2009 publication) [29]

Aedes taeniorhynchus, *Ae. scapularis*, *Culex quinquefasciatus*, *Cx. declarator* and *Cx. nigripalpus* are identified as enzootic vectors. [30-32]

A case of ocular infection by *Pelecitus*, an avian nematode, has been reported. [33]
- A case of ocular infection by a zoonotic *Dirofilaria* species has been reported. [34]

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Eastern equine encephalitis

Agent	VIRUS - RNA. Togaviridae, Alphavirus: Eastern equine encephalitis virus
Reservoir	Wild bird Horse Cattle Pig
Vector	Mosquito (Aedes, Culiseta)
Vehicle	None
Incubation Period	7d - 10d (range 5d - 15d)
Diagnostic Tests	Viral culture (brain tissue, CSF, serum). Serology. Nucleic acid amplification. Biosafety level 2.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Vaccine	Eastern equine encephalitis
Clinical Hints	Headache, fever, seizures, coma and leukocytosis; neurological sequelae in 40%. Case-fatality rates may approach 70%. Infection is most common during summer in temperate areas.
Synonyms	EEE. ICD9: 062.2 ICD10: A83.2

Clinical

Clinical features of eastern encephalitis (EEE) [1] , western equine encephalitis (WEE) and Venezuelan equine encephalitis (VEE) are similar, and will be discussed as a group. [2] [3]

EEE and **WEE** infections begin with headache, high fever, chills and vomiting.
- Vertigo, sore throat and respiratory symptoms are common in WEE infection.
- CNS involvement is heralded by confusion and somnolence which may progress to coma.
- Focal or generalized seizures are most common in younger patients.
- Physical examination reveals nuchal rigidity, depressed or hyperactive reflexes, tremors, or spastic paralysis.

The peripheral blood reveals a leukocytosis, more prominent in EEE [4] than in WEE.
- Cerebrospinal fluid protein levels are elevated.
- WEE tends to produce a lymphocytic pleocytosis of 50 to 500/mm3; and EEE of 600 to 2000/mm3 .

Sequelae are most severe following EEE infection, and include mental retardation, behavioral changes, seizure disorders and paralysis.
- The case-fatality rate for EEE is 42%. [5]
- Sequelae occur in 30% of infants recovering from WEE, and 70% of infants recovering from EEE.
- Parkinsonism may occur in adults following WEE.

WEE is characterized by a case-to-infection ratio of approximately 1/1,000 for adults, 1/60 for children and 1/1 for infants. [6]
- Respiratory symptoms and overt pneumonia are common. [7]
- Younger patients tend to suffer more severe disease, with rapid onset of symptoms, presence of seizures and permanent neurological residua. [8]

VEE is often characterized by a mild flu-like illness in endemic countries.
- Overt infection is heralded by fever, chills, myalgia, headache with or without photophobia, hyperesthesia, and vomiting.
- Sore throat is noted in some cases.
- 4% of children and less than 1% of adults progress to overt encephalitis, a few days to a week following the prodrome.
- Encephalitis is characterized by nuchal rigidity, ataxia, convulsions, coma, and paralysis.
- Lymphopenia, is common, often associated with neutropenia and mild thrombocytopenia.
- Hepatic dysfunction is common, and CSF examination reveals a few hundred lymphocytes.
- The case-fatality rate is less than 1%, but increases to 20% in cases of encephalitis.

This disease is endemic or potentially endemic to 20 countries.

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Eastern equine encephalitis in Brazil

Time and Place:
- Eastern equine encephalitis is reported from the area of Belem.
- As of 2007, only two fatal human cases (Trinidad and Brazil) had been reported in Latin America. [9]
- Viral activity was detected among horses in Parana State during 1996 to 1999. [10]

Vectors:
- The principal vector is *Aedes (Ochlerotatus) taeniorhynchus*).
- Additional vectors include *Culex nigripalpus*, *C. caudelli*, *C. spissipes* and *C. taeniopus*.

Seroprevalence surveys:

6.7% of horses in the Brazilian Pantanal (1993 publication) [11]

47.7% of horses in the Nhecolandia sub-region of South Pantanal (2010 publication) [12]

Seropositive birds have been identified in Sao Paulo State (1978 to 1990) [13]

Notable outbreaks:

1960 - An outbreak was reported among horses in Braganca (Para State). At the time, 61% of horses in the area were seropositive and 8.2% died. [14]

2008 to 2009 - Outbreaks (229 cases on 93 farms) were reported among equids in Pernambuco, Ceara and Para?b. [15]

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Echinococcosis - American polycystic

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Taeniidae: Echinococcus vogeli and E. oligarthrus
Reservoir	Bush dog Paca Rodent
Vector	None
Vehicle	Carnivore feces Soil
Incubation Period	3y - 30y
Diagnostic Tests	Serology (may cross react with E. granulosus). Identification of parasite in surgical specimens.
Typical Adult Therapy	Albendazole 400 mg PO BID X 28d (not of proven benefit) followed by surgery as indicated
Typical Pediatric Therapy	Albendazole 10 mg/kg/day PO X 28d (not of proven benefit) followed by surgery as indicated
Clinical Hints	Right upper quadrant pain and hepatic mass appearing years after acquisition in endemic area - jaundice may be present.
Synonyms	American polycystic echinococcosis, Echinococcus oligarthus, Echinococcus vogeli, Human polycystic hydatid disease, Neotropical echinococcosis, Neotropical polycystic echinococcosis, Neotropical unicystic echinococcosis, Polycystic echinococcosis, Polycystic neotropical echinococcosis. ICD9: 122.9 ICD10: B67.9

Clinical

In infection by *E. vogeli*, the liver is involved either as the sole site of infection (41%) or with additional abdominal (29%) or thoracic (8%) structures. [1]
• 14% of patients present with lung involvement only; and intra-abdominal cysts without liver disease are common. [2]
• Proliferation of liver cysts may extend through the hepatic capsule, to involve other abdominal organs, the diaphragm or pleural viscera. [3]
• Most cysts measure 0.5 cm to 6 cm, but may grow to over 15 cm.
• Clinical manifestations are similar to those of *E. multilocularis* infection, and include abdominal pain or mass, weight loss, jaundice, anemia and fever. [4]
• Portal hypertension and biliary obstruction are found in 25% of cases.
• 22% of patients have died during surgery or the postoperative period.

To date, only a few sporadic cases of *E. oligarthrus* infection have been described, involving muscle, subcutaneous tissues, liver [5] , orbit, and heart. [6]

This disease is endemic or potentially endemic to 13 countries.

Echinococcosis - American polycystic in Brazil

Initially, cases were reported from Acre, Mineiro (Minas Gerais) and Rondonia. [7]

Both *Echinococcus vogeli* and *E. oligarthrus* infections have been reported. [8-10]

A total of 25 cases had been reported as of 1996; 40 to 2003, including cases of *Echinococcus vogeli* infection in Para and Amapa. [11]
- 99 cases (98 *E. vogeli* and 1 *E. oligarthrus*) had been reported to 2007, accounting for 57.6% of the world's cases. [12]

Two cases have been reported in Sena Madureira, Acre State. [13]
- A series of six cases of isolated mesenteric echinococcosis without liver involvement was published from Acre in 2010. [14]

The local reservoirs are the paca (*Cuniculus paca*) [15] , bush dog (*Speothos venaticus*) and spiny rat (*Proechimys* sp.). [16]
- Coinfection by *Echinococcus vogeli* and *Calodium hepaticum* was identified in a paca *Agouti paca* in Acre. [17]

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Echinococcus oligarthrus hydatid cyst disease was diagnosed in two Brazilian agouti (*Dasyprocta leporina*). [18]

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Echinococcosis - unilocular

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Taeniidae: Echinococcus granulosus, Echinococcus canadensis
Reservoir	Dog Wolf Dingo Sheep Horse Pig
Vector	None
Vehicle	Soil Dog Feces Fly
Incubation Period	1y - 20y
Diagnostic Tests	Serology. Identification of parasite in surgical specimens.
Typical Adult Therapy	Albendazole 400 mg BID X 28d. Repeat X 3, with 2 week hiatus between cycles. Praziquantel has been used preoperatively to sterilize cyst. Follow by surgery as indicated. PAIR (puncture-aspiration-injection-reaspiration) is also used
Typical Pediatric Therapy	Albendazole 10 mg/kg/day X 28d. Repeat X 3, with 2 week hiatus between cycles. Praziquantel has been used preoperatively to sterilize cyst. Follow by surgery as indicated. PAIR (puncture-aspiration-injection-reaspiration) also used
Clinical Hints	Calcified hepatic cyst or mass lesions in lungs and other organs; brain and lung involvement are common in pediatric cases.
Synonyms	Echinococcus canadensis, Echinococcus granulosus, Echinococcus ortleppi, Hydatid cyst, Unilocular echinococcosis. ICD9: 122.0,122.1,122.2,122.3,122.4 ICD10: B67.0,B67.1,B67.2,B67.3,B67.4

Clinical

Symptoms are often absent, even when large cysts are present; and cysts are often discovered incidentally on a routine x-ray or ultrasound study. [1]

Hepatic echinococcosis often presents as abdominal pain with or without a palpable mass in the right upper quadrant. [2]
- Biliary compression or rupture of the cysts into a bile duct may mimic cholecystitis or cholelithiasis.
- Ductal compression may also result in pancreatitis. [3]
- Leakage from a cyst may produce fever, pruritis, urticaria, eosinophilia or even anaphylactic shock. [4]

Extra-hepatic echinococcosis presents as space-occupying lesions of brain [5], lung [6] and thorax [7], bone [8-18], muscles [19-27], joints [28], parapharyngeal spaces [29] or paranasal sinuses [30], heart [31-36] and heart valves [37] [38], pericardium [39] [40], breast [41-43], subcutaneous tissue [44-46], abdominal wall [47] [48], supraclavicular region [49], peripheral nerves [50], ovary, thyroid [51-53], orbits [54-59], parotid gland [60], spleen [61-66], adrenal [67], kidneys [68-72], urinary bladder [73-75], peritoneum / mesentery / omentum [76-78] or virtually any other organ. [79-85]
- In contrast to hepatic echinococcosis, extrahepatic cysts are often non-calcified and may at times be mistaken for malignancy. [86] [87]
- The brain is involved in 1 to 2% of all *Echinococcus granulosus* infections. [88]
- The spleen is involved in 0.5% to 6.0% of abdominal infections. [89]
- The clinical features of cerebral coenurosis may mimic those of echinococcosis. [90]
- Primary spinal hydatidosis occurs in 1% of cases and may be confused with space-occupying non-infectious disorders [91-95]

Pulmonary cysts [96] may rupture into the bronchial tree and produce cough, hemoptysis and chest pain. [97]
- Rupture of cysts may disseminate protoscolices to contiguous organs or into the vascular system, resulting in the formation of additional cysts.
- Late intrathoracic complications include intrapulmonary or pleural rupture, infection of the ruptured cysts, reactions of the adjacent tissues, thoracic wall invasion and iatrogenic involvement of pleura. [98]
- Rupture can occur spontaneously or as a result of trauma or surgery. [99]
- Anaphylaxis may follow cyst rupture [100] [101], but has also reported in patients with intact cysts. [102] In rare cases, anaphylactic shock (eg, following blunt trauma) may be the initial presenting feature of echinococcosis. [103]
- Secondary colonization of hydatid cysts by *Aspergillus* has been reported. [104]

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Primary superinfection of cysts by bacteria or fungi occurs in approximately 7.3% of cases. [105]

This disease is endemic or potentially endemic to 155 countries.

Echinococcosis - unilocular in Brazil

Unilocular echinococcosis is endemic to the southern region (notably Rio Grande do Sul).

A case of human infection by *Echinococcus ortleppi* (cattle strain G5) has been reported. [106]

Rate (hospitalized cases) per 100,000: 0.1 during 1966 to 1971.

Seroprevalence surveys:

3.5% (urban area) to 6.0% (rural) in Acre State (2003 publication) [107]

Dog infestation is not found in urban and suburban areas of Rio Grande do Sul; however, infestation rates in rural areas range from 11.36% to 47.37%. [108]

Also see: Echinococcosis - American polycystic

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Ehrlichiosis - human monocytic

Agent	BACTERIUM. Anaplasmataceae Ehrlichia chaffeensis Intracellular Rickettsia-like bacteria
Reservoir	Dog Tick Deer Coyote
Vector	Tick ? (Dermacentor variabilis or Amblyomma americanum)
Vehicle	None
Incubation Period	7d - 21d
Diagnostic Tests	Intramonocytic inclusions seen in blood smear. Serology. Nucleic acid amplification. Cell culture (HL60 cells).
Typical Adult Therapy	Doxycycline 100 mg PO BID X 7 to 14 days OR Rifampin 600 mg daily
Typical Pediatric Therapy	Above age 8 years: Doxycycline 2 mg/kg PO BID X 7 to 14 days. OR Rifampin 10 mg/kg/day PO
Clinical Hints	Headache, myalgia and vomiting 1 to 2 weeks following tick bite; arthralgia or macular rash may be present; leukopenia, thrombocytopenia or hepatic dysfunction common; inclusions in monocytes.
Synonyms	Candidatus Neoehrlichia mikurensis, Cowdria ruminantium, Ehrlichia canis, Ehrlichia chaffeensis, Ehrlichia muris, Ehrlichia runinantium, Human monocytic ehrlichiosis, Human monocytotropic ehrlichiosis, Neoehrlichia mikurensis, Panola Mountain Ehrlichia. ICD9: 082.41 ICD10: B28.8

Clinical

Human Monocytic Ehrlichiosis (HME) and Anaplasmosis (Human Granulocytic Ehrlichiosis, HE) are similar, and characterized by fever, headache, myalgia, thrombocytopenia, leukopenia, and elevated liver enzyme levels. [1] [2]
• A rash occurs in approximately one third of patients with HME but is less common in patients with anaplasmosis. Rash is much more common among children than adults with the disease. [3]
• Rare instances of Louria's sign (bilateral anterior thigh pain) have been reported. [4]
• Most cases of ehrlichiosis are mild; however, complications such as adult respiratory distress syndrome, renal failure, neurological disorders, myocarditis [5] , hemophagocytic lymphohistiocytosis [6] and disseminated intravascular coagulation can occur. [7] [8]
• Case-fatality ratios in severe cases are as high as 5% for HME and 10% for anaplasmosis.
• Symptomatic infection is more common among patients with AIDS than among immunocompetent persons.

Candidatus Neoehrlichia mikurensis infection is characterized by fever, cough, myalgia and hemorrhagic or thrombotic events. [9]

This disease is endemic or potentially endemic to 24 countries.

Ehrlichiosis - human monocytic in Brazil

Two cases of suspected human monocytic ehrlichiosis were reported in Minas Gerais (published 2004). [10]
- Evidence for 9 additional cases was published in 2006 [11]

Prevalence surveys:

25.4% of non-thrombocytopenic dogs (*Ehrlichia canis*, Ribeirao Preto, 2007 publication) [12]
4.8% of dogs in Rio de Janeiro (*Ehrlichia canis*, 2001 publication) [13]
7.8% of dogs in Bahia (*Ehrlichia canis*, 2008 publication) [14]
57% of dogs in Pernambuco (PCR, *Ehrlichia canis*, 2009 publication) [15]
3.7% of dogs in a semi-arid region of Paraiba (*Ehrlichia canis*, 2012 publication) [16]
38.04% of dogs in Pernambuco (PCR, *Ehrlichia canis*, 2007 to 2008) [17]
16.4% of dogs in Jataizinho, Parana State (PCR, *Ehrlichia canis*, 2012 publication) [18]
40% of dogs in Botucatu, Sao Paulo with suspected ehrlichiosis (*Ehrlichia canis*, 2001 to 2002) [19]
26.8% of thrombocytopenic dogs and 3.5% of nonthrombocytopenic dogs in Rio de Janeiro (*Ehrlichia canis*, 2005 publication) [20]

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15% of captive wild felids (2010 publication) [21]

3.4% of Brazilian Brown Brocket Deer (*Mazama gouazoubira*) and Marsh Deer (*Blastocerus dichotomus*) (*Ehrlichia chaffeensis*, Minas Gerais, 2011 publication) [22]

21.9% of *Rhipicephalus sanguineus* ticks infesting dogs in Cajazeiras and Itapua, Salvador city (*Ehrlichia canis*, 2010 publication) [23]

2.3% of *Rhipicephalus sanguineus* ticks in Monte Negro, 6.2% in Juniai (Sao Paulo), and 3.7% in Sao Paulo (Sao Paulo) are infected. This is the first report of *Ehrlichia canis* infection in *R. sanguineus*. (2007 publication) [24]

Seroprevalence surveys:

10.5% of healthy persons in Minas Gerais (2005 publication) [25]

15.07% of dogs in Minas Gerais (*Ehrlichia chaffeensis*, 1998) [26]

19.8% of dogs (*Ehrlichia canis*, 2003 publication) [27]

37.9% and 24.8% of urban and rural dogs. (17294930, Monte Negro, 2007 publication) [28]

36% of dogs in Ilheus and Itabuna, Bahia (*Ehrlichia canis*, 2011 publication) [29]

4.8% of dogs in southern Brazil (*Ehrlichia canis*, 2002 to 2003) [30]

42.5% of dogs in Cuiaba, Mato Grosso (*Ehrlichia canis*, 2010 publication) [31]

35.6% of dogs in Cajazeiras and Itapua, Salvador city (*Ehrlichia canis*, 2010 publication) [32]

70.9% of dogs in the Pantanal region of Mato Grosso State (*Ehrlichia canis*, 2011 publication) [33]

4.43% of dogs in Rio Grande do Sul (*Ehrlichia canis*, 2012 publication) [34]

69.4% of dogs in a semi-arid region of Paraiba (*Ehrlichia canis*, 2012 publication) [35]

7% of captive wild felids (2010 publication) [36]

76.76% of Brazilian marsh deer (*Blastocerus dichotomus*) prior to flooding of the Parana River (Mato Grosso do Sul, 2012 publication) [37]

Ehrlichia canis is responsible for most cases of ehrlichiosis among dogs in Brazil; however, *E. ewingii* infection has been recently suspected in five dogs. [38]

- *Ehrlichia canis* and *Ehrlichia chaffeensis* infection was been identified among cats in Maranhao. [39]
- Antibody against *E. canis* has been detected in free-ranging neotropical felids.
- *Ehrlichia chaffeensis* has been identified in Brazilian marsh deer (*Blastocerus dichotomus*) [40] and carnivorous birds. [41]

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Endocarditis - infectious

Agent	BACTERIUM OR FUNGUS. viridans streptococci, Staphylococcus aureus, enterococci, Candida albicans, et al.
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Blood culture, clinical findings, ultrasonography of heart valves.
Typical Adult Therapy	Bactericidal antibiotic appropriate to species
Typical Pediatric Therapy	As for adult
Clinical Hints	Consider in any patient with fever, multisystem disease (i.e., skin lesions, hematuria, neurological symptoms, single or multiple abscesses or bone, brain, lung, etc) and a preexisting cardiac valvular lesion.
Synonyms	Bacterial endocarditis, Endocardite, Endocarditis, Endokarditis, Fungal endocarditis, Infectious endocarditis, S.B.E.. ICD9: 421 ICD10: I33

Clinical

The definitive diagnosis of infective endocarditis requires: [1] [2]
1) Demonstration of microorganisms; and/or histological lesions in the heart or heart valves; or
2) Presence of two major criteria; or 1 major and 3 minor criteria; or 5 minor criteria, as follows:

Major Criteria:
A. Culture:
• 1. Typical microorganisms (HACEK, *Streptococcus viridans*, *Streptococcus bovis*) in 2 separate blood cultures; or community acquired *Staphylococcus aureus* or enterococcus without obvious focus.
• 2. Persistently positive blood cultures (drawn more than 12 hours apart; or three positive cultures at least one hour apart).
B. Evidence of endocardial or valvular involvement (echocardiogram, abscess, new valvular regurgitant lesion)

Minor Criteria:
A. Predisposition (heart condition, drug abuse)
B. Fever
C. Embolic phenomena, mycotic aneurysm, Janeway lesion, or intracranial hemorrhage.
D. Immunological phenomena (Osler nodes, positive rheumatoid factor)
E. Echocardiogram with suggestive, but not specific findings.
F. Positive blood culture, but not meeting Major criteria.

Etiological associations:.
• Injecting drug user: *Staphylococcus aureus*, enterococci, Enterobacteriaceae, *Pseudomonas aeruginosa*, *Candida*
• Prosthetic valve: *Staphylococcus epidermidis* Enterobacteriaceae, *Candida*, *Aspergillus*
• Rheumatic or other valvular disease: viridans Streptococci, enterococci
• "Culture negative" endocarditis: *Coxiella burnetii*, *Bartonella* spp., *Tropheryma whipplei*, et al.

This disease is endemic or potentially endemic to all countries.

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Enterobiasis

Agent	PARASITE - Nematoda. Phasmidea: Enterobius vermicularis
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Air Clothing Sexual contact (rare)
Incubation Period	14d - 42d
Diagnostic Tests	Apply scotch tape to anal verge in a.m. & paste onto glass slide for microscopy.
Typical Adult Therapy	Albendazole 400 mg PO as single dose - repeat in 2w. OR Mebendazole 100 mg PO as single dose - repeat in 2w. OR Pyrantel pamoate 11 mg/kg (max 1g) PO as single dose; or
Typical Pediatric Therapy	Mebendazole 100 mg PO as single dose (>age 2) - repeat in 2w. OR Pyrantel pamoate 11 mg/kg (max 1g) PO X 1
Clinical Hints	Nocturnal anal pruritus; occasionally vaginitis or abdominal pain; eosinophilia is rarely, if ever, encountered.
Synonyms	Enterobio, Enterobius vermicularis, Oxyuriasis, Oxyuris, Pinwom, Seatworm. ICD9: 127.4 ICD10: B80

Clinical

The typical manifestation of enterobiasis is nocturnal pruritus ani related to hypersensitivity to worm antigens.
• Local dermal "tingling" is also encountered. [1]
• Migration of adult females to the vulva may result in vaginal pain [2] and vulvovaginits [3], or predispose to urinary tract infection.
• Eosinophilia is occasionally present.

Complications are rare, and include salpingitis [4], cystitis [5], peritonitis [6] [7], hepatitis granuloma [8] and urethritis. [9]
• Although abdominal symptoms may mimic those of appendicitis, *Enterobius* is at least as common in normal as in inflamed appendices. [10-13]
• Cases of *Enterobius* prostatitis [14] and peritonitis have been reported. [15]
• Adults and ova of *Enterobius* have been identified in the kidneys [16] [17] and eyes [18] of infested patients.

The presence of diarrhea or abdominal pain suggests coinfection with *Dientamoeba fragilis*.

This disease is endemic or potentially endemic to all countries.

Enterobiasis in Brazil

Prevalence surveys:

0.5% of persons in Eirunepe, Amazon (2005 publication) [19]
15% of children below age 5 years in Santa Isabel do Rio Negro, Amazon region (2011 publication) [20]
0.8% of persons in Americaninhas, Minas Gerais (2004) [21] [22]
0.7% of persons in Maria Helena, Parana (2004 to 2006) [23]
0.3% of Terena Indians (Mato Grosso do Sul) [24]
5.9% of school children in Salvador (1997)
8% of school children in Caxias do Sul, RS (2008 publication) [25]
1.9% of children in Sao Paulo (2004 publication) [26]
1.7% of children in an urban slum in Sao Paulo (2008 publication) [27]
4% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [28]
0.6% of urban children with diarrhea in Salvador (2000 to 2002) [29]
0.5% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [30]

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6.25% of public restrooms in Uberlandia, Minas Gerais (*Ascaris* and *Enterobius vermicularis*, 2009 publication) [31]
1.4% of surgically removed appendices over a 10 year period in southeastern Brazil (Botucatu, 2007 publication) [32]

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Enterovirus infection

Agent	VIRUS - RNA. Picornaviridae: Coxsackievirus, ECHO virus, Enterovirus, Parechovirus
Reservoir	Human
Vector	None
Vehicle	Droplet Fecal-oral
Incubation Period	2d-7d
Diagnostic Tests	Viral culture (stool, pharynx, CSF). Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive. Pleconaril 200 to 400 mg PO TID X 7d has been used for severe infections
Typical Pediatric Therapy	Supportive. Pleconaril 5 mg/kg PO BID has been used for severe infections
Clinical Hints	Summer-to-autumn sore throat; occasionally chest pain, macular or vesicular rash, meningitis, myopericarditis, etc.
Synonyms	Boston exanthem [Caxsackie. A 16], Coxsackie, Coxsackievirus, ECHO, Echovirus, Enteroviruses, Hand, foot and mouth disease, Hand-foot-and-mouth disease, Herpangina [Coxsackievirus A], HEV 68, HPeVs, Human Enterovirus 68, Human Parechovirus, Ljungan virus, Myocarditis, enteroviral, Parechovirus, Pericarditis, enteroviral. ICD9: 049,079.2,008.67,074.0,074.8,074.3,070.4,078.89 ICD10: A88.0,A87.0,B08.4,B08.5,B08.8,B30.3,B34.1

Clinical

The various enteroviruses are associated with fever and pharyngitis, which may be followed by appearance of: [1] [2]
• rash
• aseptic meningitis
• encephalitis [3]
• acute disseminated encephalomyelitis [4]
• epidemic conjunctivitis
• herpangina
• hand-foot-and-mouth disease
• myocarditis
• pericarditis
• pleurodynia
• pneumonia
• acute flaccid paralysis [5-7]
• conjunctivitis, etc

Hand, foot and mouth disease (HFM) is characterized by a prodrome of fever and sore throat, followed by the appearance of vesicles on the palmar and plantar regions, and oral mucosa.
• Vesicles in the mouth are often pleomorphic, with rectangular and triangular shapes.
• Hand foot and mouth disease has been associated with onychomadesis • complete nail shedding from the proximal portion, affecting both fingernails and toenails. [8-16]
• HFM due to Enterovirus 71 is often complicated by central nervous system disease and sequelae. [17-42]

The clinical features of Enterovirus infection among neonates and infants are similar to those of Parechovirus infection. [43]

Human Enterovirus 68 infection is associated with respiratory illness ranging from relatively mild illness that did not require hospitalization to severe illness requiring intensive care and mechanical ventilation. Some infections have been fatal. [44] [45]

Echoviruses 22 and 23 have been reclassified as human parechovirus (HPeV) 1 and 2 , respectively. [46]
• Parechovirus infections have been associated with respiratory and gastrointestinal disease [47] [48] , epidemic myalgia [49] or rarely encephalitis. [50]
• HPeV2 is usually associated with gastrointestinal illness.
• HPeV3 has been associated with transient paralysis, sepsis-like syndromes, or myalgia with muscle weakness.
• HPeV4 has been associated with fever in a neonate [51]
• HPeV6 (NII561-2000) has been associated with infectious gastroenteritis, fever with rash, upper respiratory infection and

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Reye's syndrome

This disease is endemic or potentially endemic to all countries.

Enterovirus infection in Brazil

Prevalence surveys:

5.7% of acute respiratory infections in the age group < 5 years (1984 to 1986)
40.5% of children with tonsillar diseases (2912 publication) [52]
11.34% of CSF specimens from cases of suspected viral CNS infection (Sao Paulo, 2006 publication) [53]
19.8% of aseptic meningitis in Para State (2005 to 2006) [54]
83% of viruses associated with lympho-monocytic meningitis cases in Curitiba (2005 to 2006) [55]
50% of meningitis cases in a dengue-endemic area (2011 publication) [56]
10.7% (10.3% Echovirus 30, 0.4% Coxsackie B virus) of CSF samples from meningitis cases (Belem, Para state, 2002 to 2003) [57]
3.5% of influenza-like illness with proven viral etiology among adults in Sao Paulo (2001 to 2003) [58]
16.1% of children below age 6 years with enteritis - including a novel strain, HPeV-8 (Parechoviruses, Salvador, 2006 to 2007) [59]
64.28% of surface water samples in Arroio Diluvio, Porto Alegre (2009) [60]
37.5% of sewage effluent samples in Porto Alegre (2012 publication) [61]

Seroprevalence surveys:

12.4% of persons in Sao Paulo State (Enterovirus 71, neutralizing antibodies, 1999 to 2005) [62]

During 1998 to 2003, Enterovirus 30 accounted for 85.2% of enteroviral meningitis, Coxsackievirus B5 3.7%, Echovirus 13 3.7%, Echovirus 18 3%, Echovirus 6 1.2%, Echovirus 25 1.2%, Echovirus 1 0.6%, and Echovirus 4 0.6%. [63]
- 29 cases of Enterovirus 30 meningitis were identified in Belem during 2002 to 2003 [64] ; 20 in Para during 2005 to 2006. [65]

Notable outbreaks:

1983 - An outbreak of acute hemorrhagic conjunctivitis was reported in Cuiaba, Mato Grosso. [66]
1983 (publication year) - An outbreak of Echovirus 9 meningitis was reported in Rio de Janeiro. [67]
1983 to 1984 - An outbreak of Acute hemorrhagic conjunctivitis (AHC) in Sao Paulo was caused by Enterovirus 70. [68] [69]
1987 - An outbreak of AHC in Belem, Para State was caused by Coxsackievirus A24. [70]
1995 (publication year) - An outbreak (10 cases) of exanthema caused by Coxsackievirus B3 was reported in a day care center. [71]
2003 - An outbreak of acute hemorrhagic conjunctivitis caused by Coxsackievirus A24 began in South America in the spring, affecting an estimated 200,000 persons in Brazil. [72-74]
2003 - An outbreak (17 cases, 0 fatal) of aseptic meningitis due to Echovirus 13 was reported in Rio Grande do Sul. [75]
2004 to 2005 - An outbreak (60,000 cases or more) of acute hemorrhagic conjunctivitis due to Coxsackievirus A24 was reported in Rio de Janeiro and Espirito Santo. [76]
2005 - An outbreak (22 cases confirmed) of Echovirus 30 meningitis was reported in Rio de Janeiro State. [77] [78]
2009 - An outbreak of acute hemorrhagic conjunctivitis due to Coxsackievirus A24 was reported in Pernambuco State. [79]

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Entomophthoramycosis

Agent	FUNGUS. Zygomycota, Zygomecetes, Entomopthorales: Basidiobolus or Conidiobolus
Reservoir	Vegetation Soil Amphibian Reptile
Vector	None
Vehicle	Air (inhalation) Direct inoculation
Incubation Period	Unknown
Diagnostic Tests	Biopsy and fungal culture.
Typical Adult Therapy	Antifungal agents and excision as indicated. Oral potassium iodide may be helpful
Typical Pediatric Therapy	As for adult
Clinical Hints	Slowly-spreading subcutaneous nodule involving nose, upper face, pharynx; no skin ulceration or systemic signs.
Synonyms	Basidiobolomycosis, Basidiobolus, Conidiobolosis, Conidiobolus, Rhinoentomophthoramycosis, Rhinophycomycosis entomophthorae, Subcutaneous phycomycosis, Subcutaneous zygomycosis. ICD9: 117.9 ICD10: B48.8

Clinical

Basidiobolomycosis is caused by *Basidiobolus ranarum* (*B. haptosporus*, *B. meristosporus*) and involves the subcutaneous tissues and rarely the intestine. [1] [2]
• The typical infection is characterized by a single, painless, sharply circumscribed, hard subcutaneous mass • most commonly on the thigh or buttock.
• Infection of the nose and other facial structures is also reported. [3]
• The overlying skin remains intact, and bone involvement is not seen.
• Abdominal disease is characterized by abdominal pain and a palpable mass, and may mimic an intestinal malignancy or acute appendicitis. [4-6]
• Additional findings may include hepatosplenomegaly, hepatic mass lesion, jaundice, gastrointestinal hemorrhage [7] or eosinophilia. [8]

Conidiobolomycosis begins in the nasal submucosa and spreads to the skin, glabella, cheek, lips, paranasal sinuses and pharynx. [9] [10]
• Central facial swelling may progress to overt elephantiasis of the face. [11]
• Histological examination reveals hyphae surrounded by an eosinophilic halo (Spendore-Hoeppli phenomenon) [12]
• Rare cases of disseminated conidiobolomycosis have been reported. [13]

This disease is endemic or potentially endemic to 32 countries.

Entomophthoramycosis in Brazil

Cases of Entomophthoramycosis due to *Conidiobolus coronatus* have been reported in Sao Paulo [14] , Bahia [15] and Mato Grosso. [16]

Brazil accounted for 28 of 33 cases of entophthoramycosis reported in Latin America (1988 publication) [17] [18]

Prevalence surveys:
8.1% of sheep undergoing necropsy (rhinopharyngeal *Conidiobolus lamprauges* infection, 2013 publication) [19]

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Epidural abscess

Agent	BACTERIUM. Staphylococcus aureus, facultative gram negative bacilli, etc
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Imaging (CT scan, MRI). Gram-stain and culture of blood or pus.
Typical Adult Therapy	Intravenous antibiotic(s) appropriate to identified or suspected pathogens. Drainage as indicated
Typical Pediatric Therapy	Intravenous antibiotic(s) appropriate to identified or suspected pathogen. Drainage as indicated
Clinical Hints	Frontal bone abscess; or spinal cord compression with signs of infection - often in setting of injecting drug abuse or preexisting staphylococcal infection.
Synonyms	

Clinical

Intracranial epidural abscesses:
Intracranial epidural abscesses may appear gradually, with initial findings suggestive of the underlying sinusitis or otitis. [1]
• Early findings include local pain followed by generalized headache, often with alteration of mental status.
• Focal neurological signs and focal or generalized seizures appear, which reflect the local anatomy of the lesion, for example:
• abscess near the petrous bone may involve cranial nerves V and VI, with unilateral facial pain and lateral rectus weakness (Gradenigo's syndrome)
• an occipital epidural abscess may obstruct the superior sagittal sinus

Eventually, papilledema and other signs of elevated intracranial pressure develop.
• Extension into the subdural space is accompanied by rapid neurological deterioration.

Spinal epidural abscess:
Spinal epidural abscess is more common in men than in women and may occur at any age.
• The presentation may be acute or gradual, over several months. [2]
• Most begin with focal vertebral pain, which begins to radiate along the course of involved nerve roots.
• Signs of spinal cord compression (long-tract findings), later progress to paralysis below the level of the lesion.
• Hematogenous infection of the epidural space produces rapid progression with prominent systemic signs, and severe local pain.
• Chronic abscesses may mimic epidural neoplasia, often without systemic signs of infection.
• Cervical abscesses may compromise respiration, and produce rapid evolving flaccid hyporeflexia, suggestive of Guillain-Barre syndrome.
• Epidural abscess has occasionally been reported as a complication of pyomyositis. [3]

This disease is endemic or potentially endemic to all countries.

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Erysipelas or cellulitis

Agent	BACTERIUM. Erysipelas: Streptococcus pyogenes Cellulitis: Staphylococcus aureus, Streptococcus pyogenes, occasionally others
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	1d - 7d
Diagnostic Tests	Clinical diagnosis is usually sufficient. Aspiration of lesion for smear and culture may be helpful in some cases.
Typical Adult Therapy	Antibiotic directed at likely pathogens (Group A Streptococcus and Staphylococcus aureus)
Typical Pediatric Therapy	As for adult
Clinical Hints	Erysipelas is well-circumscribed, tender, edematous (peau d'orange), warm and painful; cellulitis is less painful, flat and without a distinct border.
Synonyms	Cellulite, Cellulitis, Celulite, Celulitis, Erisipela, Erysipelas, St. Anthony's fire (erysipelas), St. Francis' fire (erysipelas), Zellulitis. ICD9: 035,681,682 ICD10: A46,L03

Clinical

Erysipelas:
Erysipelas is characterized by abrupt onset of "fiery-red" superficial swelling of the face or extremities. [1]
• The lesion is typically recognized by the presence of well-defined indurated margins, particularly along the nasolabial fold; rapid progression; and intense pain. [2]
• Flaccid bullae may develop on the second or third day of illness; but extension to deeper soft tissues is rare.
• Desquamation occurs between the fifth and tenth days of illness.

Cellulitis:
Cellulitis is characterized by local pain, erythema, swelling, and heat. [3] [4]
• Cellulitis may be caused by any of a wide variety of bacteria or yeasts; however, *S. aureus* or *S. pyogenes* are most often implicated.
• A history of preceding trauma, insect bite, needle insertion or surgery is often present.
• Cultures of biopsy specimens or aspirates are positive in only 20% of cases.
• Infection by *S. aureus* often spreads out from a localized infection (abscess, folliculitis) or foreign body
• Streptococcal cellulitis tends to be more diffuse and rapid in onset, and associated with lymphangitis and fever.
• Streptococci also cause recurrent cellulitis in the setting of lymphedema resulting from elephantiasis or lymph node damage.

Recurrent staphylococcal cutaneous infections are encountered in patients with "Job's syndrome" (eosinophilia and elevated serum levels of IgE); and nasal carriers of staphylococci.

This disease is endemic or potentially endemic to all countries.

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Erysipeloid

Agent	BACTERIUM. Erysipelothrix rhusiopathiae A facultative gram-positive bacillus
Reservoir	Mammal Bird Fish
Vector	None
Vehicle	Contact with meat, mammal, poultry or fish
Incubation Period	1d - 4d
Diagnostic Tests	Culture.
Typical Adult Therapy	Penicillin V, Ampicillin, third-generation cephalosporin, Fluoroquinolone (Levofloxacin, Trovafloxacin, Pefloxacin, Sparfloxacin or Moxifloxacin), Erythromycin or Tetracycline generally adequate
Typical Pediatric Therapy	Penicillin V, Ampicillin, third-generation cephalosporin or Erythromycin generally adequate
Clinical Hints	Annular erythema or 'target lesion' on hand following contact with raw animal or fish products; local pain and swelling; no discharge is noted and fever is present in only 10% of cases.
Synonyms	Erysipelothrix rhusiopathiae, Rutlauf. ICD9: 027.1 ICD10: A26

Clinical

Erysipeloid is generally limited to the skin (mainly hands and fingers)

Infection is characterized by pain, edema and purplish erythema with sharp irregular margins which extends peripherally but clears centrally. [1] [2]
- Relapses and extensions of the lesions to distant areas are common, but there is no fever.
- There is no permanent immunity following an attack.
- Lesions of cutaneous leishmaniasis may mimic those of erysipeloid. [3]

Complications:
- 31 cases of endocarditis due to *Erysipelothrix rhusiopathiae* had been reported to 1976 [4] [5] ; and approximately 50 to 1988. [6]
- Rarely-reported complications have included chronic granulomatosis cheilitis [7] , peritonitis associated with peritoneal dialysis [8] , pneumonia [9] and spinal infection with epidural empyema. [10]

This disease is endemic or potentially endemic to all countries.

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Erythrasma

Agent	BACTERIUM. Corynebacterium minutissimum A facultative gram-positive bacillus
Reservoir	Human
Vector	None
Vehicle	Indigenous flora
Incubation Period	Unknown
Diagnostic Tests	Coral fluorescence of skin lesion under Wood's lamp. Culture (alert lab regarding diagnosis).
Typical Adult Therapy	Erythromycin 250 mg PO QID X 14d. Topical Clindamycin 2% and topical Fusidic acid have also been used
Typical Pediatric Therapy	Erythromycin 10 mg/kg PO QID X 14d. Topical Clindamycin 2% and topical Fusidic acid have also been used
Clinical Hints	Pruritic, scaling, slowly-progressive red-brown patch; usually in groin - occasionally in toe webs; common in obese or diabetic males; coral fluorescence with Wood's light.
Synonyms	Corynebacterium minutissimum, Eritrasma. ICD9: 039.0 ICD10: L08.1

Clinical

Erythrasma is characterized by slowly spreading, reddish-brown, pruritic patches • usually in the groin and axillae. [1]
• Other areas include the interdigital regions of the feet [2] , the vulva [3] and intergluteal and crural folds.
• Most patients are obese, male diabetics. [4-6]
• The lesions fluoresce red when exposed to Wood's lamp. [7-10]
• The differential diagnosis of erythrasma includes psoriasis, dermatophytosis, candidiasis and intertrigo.

The etiologic agent of erythrasma, *Corynebacterium minutissimum*, has also been associated with bacteremia [11-14] , meningitis [15] , breast abscess [16] , eye infection [17] , endocarditis [18] [19] , peritonitis [20] , cutaneous granulomas [21] , costochondral abscess [22] , pueperal infection [23] and pyelonephritis. [24] [25]

This disease is endemic or potentially endemic to all countries.

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Escherichia coli diarrhea

Agent	BACTERIUM. Escherichia coli A facultative gram-negative bacillus
Reservoir	Human Mammal
Vector	None
Vehicle	Food Water Fecal-oral
Incubation Period	1d - 3d (range 12h - 10d)
Diagnostic Tests	Stool culture. Request characterization of E. coli isolates.
Typical Adult Therapy	Supportive therapy. Avoid anti-motility drugs and antimicrobial agents. Plasma exchange may be effective in HUS Note that antimicrobial agents may increase risk for hemolytic-uremic syndrome when used in cases of E. coli O157:H7 infection
Typical Pediatric Therapy	Supportive therapy. Avoid anti-motility drugs and antimicrobial agents. Plasma exchange may be effective in HUS Note that antimicrobial agents may increase risk for hemolytic-uremic syndrome when used in cases of E. coli O157:H7 infection
Clinical Hints	Watery diarrhea or dysentery - common among travelers and infants; hemorrhagic colitis and hemolytic uremic syndrome are associated with type O157:H7 (& occasionally other types).
Synonyms	DAEC (Diffusely Adherent E. coli), E. coli diarrhea, EAEC (Enteroadherent E. coli), EAggEC (Enteroaggregative E. coli), EHEC (Enterohemorrhagic E. coli), EIEC (Enteroinvasive E. coli), EPEC (Enteropathogenic E. coli), Escherichia albertii, ETEC (Enterotoxic E. coli), Hamolytisch-uramisches Syndrom, Hemolytic Uremic Syndrome, HUS. ICD9: 008.0 ICD10: A04.0,A04.1,A04.2,A04.3,A04.4

Clinical

Enterotoxic *Escherichia coli* (ETEC) infection is characterized by a short incubation period, and watery diarrhea without blood or mucus.
• Fever and vomiting occur in a minority of patients. [1]
• The disease may be life-threatening in infants.

Enteropathogenic *E. coli* (EPEC) causes watery diarrhea with fever and vomiting, primarily among children under age 2 years.

Enteroinvasive *E. coli* (EIEC) causes watery diarrhea; only a minority of patients experience dysentery.

Enterohemorrhagic *E. coli* (EHEC) causes diarrhea without fever, often with blood and cramps at all ages. [2]
• Rare instances of toxic megacolon have been reported [3]
• One strain of EHEC, O157:H7 is an important cause of hemolytic-uremic syndrome (HUS). [4]
• Approximately 6% to 10% of patients infected by this strain develop HUS • with an overall mortality rate of 0.6% for STEC O157 infections and 4.6% for HUS. [5]
• Nearly 40% of patients with STEC-HUS require at least temporary renal replacement therapy and up to 20% will have permanent residual kidney dysfunction. [6]
• Hemolytic-uremic syndrome can also follow infection by *Clostridium difficile* [7] and by non-O157 strains of *E. coli*. [8]
• Reactive arthritis is reported in 10% of cases [9]

Enteroaggregative *E. coli* (EAggEC) causes watery, persistent diarrhea (over 2 weeks) without vomiting. [10]
• Low-grade fever may be observed, and gross blood may occasionally be present in stools. [11]

This disease is endemic or potentially endemic to all countries.

Escherichia coli diarrhea in Brazil

The first Brazilian case of Shiga toxin-producing *E. coli* infection (nonfatal HUS) was reported in 2001. [12] [13]
- The isolate was identified as *E. coli* O26:H11.

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Prevalence surveys:

Escherichia coli was found in 11.6% of infants hospitalized with acute diarrhea (Natal, 2008 publication) [14]

Enteropathic *E. coli* accounted for 7.3% of diarrhea episodes treated at a tertiary pediatric care facility in Salvador, Bahia. [15]

Enterotoxigenic *E. coli* was found in 13% of children with diarrhea and 22% of asymptomatic controls (Sao Paulo, 1982). [16]

EPEC was found in 10.8% of diarrhea among urban children Salvador, ETEC 7.5%, EAEC 4.2% (2000 to 2002) [17]

EPEC was found in 14.3% of children below age 2 years with diarrhea (1998 to 199917152) [18]

EPEC was found in 6.1% of children below age 6 years with diarrhea, in Porto Velho (Rondonia), EAEC 5.5%, ETEC 4.4% [19]

EPEC was found in 1.7% of children with diarrhea in northeastern Brazil, atypical EPEC 9.3%, EAEC 25%, ETEC 10%, EIEC 1.4% (2008 publication) [20]

EPEC was found in 22.5% of HIV-positive patients with diarrhea, and 5% of control patients (southeastern Brazil, 2006 to 2007) [21]

EPEC was found in 19.1% of asymptomatic children ages 4 to 14 months, in a day-care center (2012 publication) [22]

E. coli O157:H7 was not detected in any oysters sampled from the Cananeia estuary (Sao Paulo, 1998 to 1999) [23]

E. coli was found in 10.3% of raw cow's milk samples in Minas Gerais (2012 population) [24]

E. coli was found in 50% of recreational waters in a lagoon (Rio de Janeiro, 2012 publication) [25]

Notable outbreaks:

1967 (publication year) - An outbreak of *Escherichia coli* 0111:B4 gastroenterocolitis was reported among premature infants in a hospital in Ribeirao Preto. [26]

1998 - Outbreaks of cholera-like diarrhea caused by enterotoxigenic *Escherichia coli* were reported in the Amazon Rainforest. [27]

2011 - An outbreak (3,910 cases, 782 HUS, 46 fatal) of enteroaggregative, Shiga toxin-producing *E. coli* O104:H4 infection in Europe originated in Germany (3,785 cases, 855 HUS, 45 fatal). Additional cases were reported in Austria (5 cases, 1 HUS - travelers from Germany), Czech Republic (1 - an American tourist arriving from Germany), Denmark (26 cases, 10 HUS - 0 fatal, 2 resulting from person-to-person spread) - in addition to 1 Canadian tourist returning from Germany, France (2 cases), Greece (1 case), Luxembourg (2 cases, 1 HUS), the Netherlands (11 cases, 4 HUS), Norway (1 case), Poland (3 cases, 2 HUS), Spain (2 cases, 1 HUS), Sweden (53 cases, 18 HUS - 1 fatal case arriving from Germany), Switzerland (3 cases) , the United Kingdom (7 cases, 3 HUS - travelers from Germany). Six cases (1 fatal) were reported in the United States, including 5 among travelers from Germany; and 2 suspect cases were reported in Brazil. Contaminated bean sprouts and imported fenugreek (from Egypt) were implicated as possible vehicles. [28-171]

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Fascioliasis

Agent	PARASITE - Platyhelminthes, Trematoda. Echinostomatida, Fasciolidae: Fasciola hepatica or Fasciola gigantica
Reservoir	Sheep Cattle Snail (Lymnaea, Fossaria)
Vector	None
Vehicle	Food Aquatic plants Watercress (Nasturtium officinale)
Incubation Period	2w - 3m
Diagnostic Tests	Identification of ova in stool or duodenal aspirates (adult parasite in tissue). Serology. PCR. CT scan.
Typical Adult Therapy	Triclabendazole 10 mg/kg PO X 2 doses. OR Bithionol 50 mg/kg every other day X 10 doses OR Nitazoxanide 500 mg PO BID X 7d
Typical Pediatric Therapy	Triclabendazole 10 mg/kg PO X 2 doses. OR Bithionol 50 mg/kg every other day X 10 doses OR Nitazoxanide: Age 1 to 3y 100 mg BID X 7 d Age 4 to 11y 200 mg BID X 7d
Clinical Hints	Fever, hepatomegaly, cholangitis, jaundice and eosinophilia; urticaria occasionally observed during the acute illness; parasite may survive more than 10 years in the biliary tract.
Synonyms	Eurytrema, Fasciola gigantica, Fasciola hepatica, Hepatic distomiasis, Lederegelbefall, Sheep liver fluke. ICD9: 121.3 ICD10: B663.

Clinical

The presence and severity of disease depend on the intensity of infection and the host.

Symptoms may appear a few days after ingestion of larvae, when the immature worms reach the abdominal cavity and begin migrating across or within the liver. [1]
- Typical early symptoms include fever, abdominal pain, gastrointestinal disturbances and urticaria. [2]
- Hepatomegaly, anemia and jaundice may also be present.
- Creeping eruption has been reported. [3]
- Rare instance of ectopic adult worms are reported [4-8]

A latent phase follows during which the only finding is prominent eosinophilia.
- Eventually, the patient enters a chronic phase characterized by biliary colic [9] , epigastric pain, jaundice, hepatomegaly and abdominal tenderness. [10] [11]
- Sporadic cases of systemic vasculitis [12] and pancreatitis are encountered. [13-18]

This disease is endemic or potentially endemic to 99 countries.

Fascioliasis in Brazil

Time and Place:
- Cases have been reported in Curitiba, Parana. [19]
- The parasite has been identified in the stools of persons from Volta Redonda (Rio de Janeiro State). [20]
- *Fasciola* is found on vegetables sold in metropolitan Sao Paulo. [21]

14 cases were reported in the world's literature during 1969 to 1989; 35 to 1999. [22]

Prevalence surveys:

18.7% of cattle in Rio Grande do Sul and 10.1% in Santa Catarina (2003 to 2008) [23]
15.24% of livestock at slaughter in Espirito Santo in 2006, 23.93% in 2007, 28.57% in 2008 and 28.24% in 2009 [24]
56.2% of feral nutria (*Myocastor coypus*) in a public park in Curitiba (2009 publication) [25]

Reservoirs:

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- Infection has been documented in cows, water buffalo, sheep and goats in Santa Catarina State [26] and Rio Grande do Sul. [27]

- Infection has been documented in a capybara (*Hydrocherus hydrocheris*). [28]
- The local snail reservoir is *Lymnaea (Fossaria) columella*. [29] , with high rates in the Paraiba River Valley, Sao Paulo State. [30]

Eight outbreaks of acute and subacute fascioliasis were reported among sheep in Santa Vitoria do Palmar, southern Brazil (2012 publication). [31]

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Filariasis - Bancroftian

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Wuchereria bancrofti
Reservoir	Human
Vector	Mosquito (Anopheles, Aedes, Culex)
Vehicle	None
Incubation Period	5m - 18m (range 1m - 2y)
Diagnostic Tests	Identification of microfilariae in nocturnal blood specimen. Nucleic acid amplification. Serology may be helpful.
Typical Adult Therapy	Diethylcarbamazine : 50 mg day 1 50 mg TID day 2 100 mg TID day 3 Then 2 mg/kg TID X 18 days. OR Ivermectin 200ug/kg PO as single dose. Doxycycline 200 mg daily X 8 w is also effective.
Typical Pediatric Therapy	As for adult
Clinical Hints	Lymphangitis, lymphadenitis, eosinophilia, epididymitis, orchitis, hydrocoele or progressive edema; episodes of fever and lymphangitis may recur over several years; chyluria occasionally encountered.
Synonyms	Bancroftian filariasis, Rosetta leg, Wuchereria bancrofti. ICD9: 125.0 ICD10: B74.0

Clinical

WHO Case definition for surveillance:
Clinical case definition
• Hydrocoele or lymphedema in a resident of an endemic area for which other causes of these findings have been excluded.
Laboratory criteria for diagnosis
• Microfilaria positive, antigen positive or biopsy positive.
Case classification
Suspected: Not applicable.
Probable: A case that meets the clinical case definition.
Confirmed: A person with laboratory confirmation even if he/she does not meet the clinical case definition.

Clinical manifestations reflect either acute inflammation or lymphatic obstruction. [1-3]
• Repeated episodes of lymphangitis, lymphadenitis, fever, headache, backache and nausea may occur; and arthritis [4] , funiculitis, epididymitis, or orchitis are common.
• In long-standing cases lymphedema or persistent adenopathy may develop.
• Hydrocoele [5] is the most common clinical manifestation of lymphatic filariasis, and causes sexual disability.
• Hydrocoelectomy accounts for 25% of all surgical procedures performed in endemic areas of Ghana and Kenya.
• Lower limb involvement is characterized by initial pretibial pitting edema, which eventually becomes nonpitting and involves the entire leg.
• The skin of the leg or scrotum becomes thick, fissured, and warty; and ulceration and secondary infection may occur.
• Rare instances of pleural effusion [6] , multiple subcutaneous nodules [7] and intra-abdominal cysts are reported [8]
• Chyluria reflects rupture of swollen lymphatics into the urinary tract. [9-14] Microscopic (occasionally gross) hematuria is reported in some cases. [15] [16]
• Filarial granuloma may mimic testicular cancer. [17]

Microfilariae may be found in properly timed blood specimens, hydrocoele fluid, chylous urine and organ aspirates. [18] [19]
• Adult worms are identified in biopsy material.
• Eosinophilia usually appears only during acute episodes of inflammation.

There is extensive evidence that endosymbiont bacteria (***Wolbachia*** spp.) are necessary for the development of filarial larvae, and fertility of adult parasites. [20-23]
• Doxycycline has proven effective in therapy, presumably through inhibition of *Wolbachia* spp. [24-27]

This disease is endemic or potentially endemic to 117 countries.

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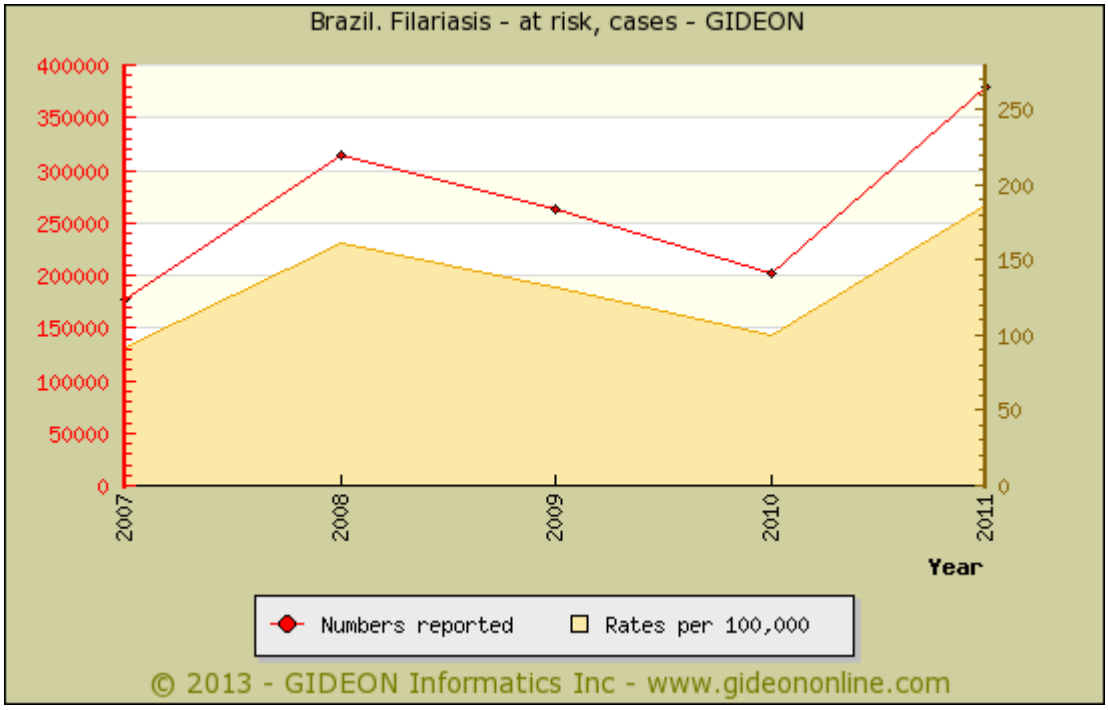
Filariasis - Bancroftian in Brazil

Bancroftian filariasis was first documented in Brazil in 1878 and in Recife in 1952.

Time and Place:
- Transmission occurs in urban areas, mainly along the coast.
- During 1951 to 1958, autochthonous transmission was documented (in decreasing order) in Ponta Grossa, Belem, Barra de Laguna, Recife, Castro Ives, Florianopolis, San Luis, Salvador, Maceio, Manaus and Porto Alegre. [28] As of the 1980's, the only cities with autochthonous transmission were Belem and Recife. As of the 1990's, additional cities (Maceio, Olinda, Jaboatao dos Guararapes and Paulista) also reported transmission. As of 2012, only four cities reported transmission - all in the region of metropolitan Recife. [29]
- Ten transmission foci were identified in 1967 - three of these in Santa Catarina State. No new cases were reported from Santa Catarina State since the 1980's.
- Over 1,500 cases were reported in Recife in 1995.
- The disease is also common in Para, other parts of Pernambuco, Bahia, Amazonas (sporadic), Alagoas State [30] and the south coast.



Graph: Brazil. Filariasis - at risk, cases

Notes:
1. Number of persons targeted for mass treatment.
2. Additional references: 2008 [31] 2010 [32]

The disease in this country is nocturnally periodic. [33]

Prevalence surveys:

0.2% nationwide (estimated)
0.08% in Cabo de Santo Agostinho, Pernambuco (2000) [34]
0.8% in Jaboatao (2002) [35]
2.6% in Belem during the 1950's, 0.16% during the 1970's and 0.02% during the 1990's. [36]
6.5% in Recife (range 0.6% to 15%) - 2.5% of military personnel in the city are infested (1989 to 1995). [37]
1.4% in Jaboatao dos Guararapes, Pernambuco (2009 publication) [38] [39]
1.1% of children and adolescents in Jaboatao dos Guararapes, Pernambuco (2011 publication) [40]

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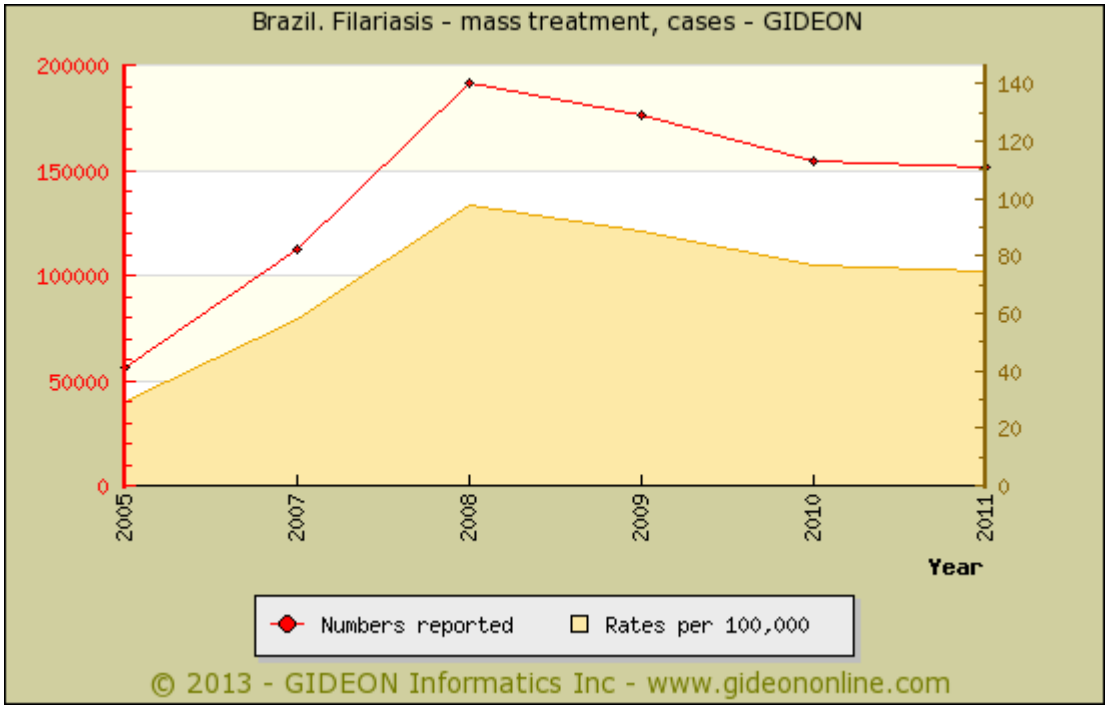

Graph: Brazil. Filariasis - mass treatment, cases

Notes:

1. Additional references: 2005 [41] 2007 [42] 2008 [43] 2010 [44]

Vectors:

The local vectors are *Culex quinquefasciatus*, *Anopheles bellator*, *An. darlingi*, *An. aquasalis*, *Aedes scapularis* and *Culex pipiens*.

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Filariasis - Brugia malayi

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Brugia malayi
Reservoir	Human Non-human primate Cat Civet Dog
Vector	Mosquito (Mansonia, Aedes, Anopheles)
Vehicle	None
Incubation Period	5m - 18m (range 1m - 2y)
Diagnostic Tests	Identification of microfilariae in nocturnal blood specimen. Nucleic acid amplification. Serology may be helpful.
Typical Adult Therapy	Diethylcarbamazine : 50 mg day 1 50 mg TID day 2 100 mg TID day 3 Then 2 mg/kg TID X 18 days. OR Ivermectin 200ug/kg PO as single dose Add Doxycycline 100 mg PO daily X 6 weeks.
Typical Pediatric Therapy	As for adult
Clinical Hints	Lymphangitis, lymphadenitis, eosinophilia, fever and progressive lower extremity edema - usually spares the knee and elbow; hydrocoele and chyluria not encountered.
Synonyms	Brugia malayi, Malayan filariasis. ICD9: 125.1 ICD10: B74.1

Clinical

WHO Case definition for surveillance:
Clinical case definition
• Hydrocoele or lymphedema in a resident of an endemic area for which other causes of these findings have been excluded.
Laboratory criteria for diagnosis
• Microfilaria positive, antigen positive or biopsy positive.
Case classification
Suspected: Not applicable.
Probable: A case that meets the clinical case definition.
Confirmed: A person with laboratory confirmation even if he/she does not meet the clinical case definition.

Bancroftian vs. Brugian filariasis:

The main clinical features which differentiate Bancroftian from Brugian filariasis are the rarity of genital lesions (hydrocoele) and chyluria in the latter. [1]

• Edema extending above the knee is also primarily a feature of bancroftian disease. [2]
• Abscess formation is often encountered with Brugian filariasis in Malaysia, Indonesia and Thailand; but not in India and other countries.
• Human infection by zoonotic *Brugia* species usually involves lymphatics of the neck, groin or axilla. [3]

Additional clinical features:

• Most cases present as painful regional lymphadenopathy, occasionally with eosinophilia.
• Microscopic (occasionally gross) hematuria is reported in some cases. [4] [5]
• Disseminated disease may be associated with pulmonary cysts and empyema [6]

This disease is endemic or potentially endemic to 16 countries. Although Filariasis - Brugia malayi is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Filariasis - Brugia malayi in Brazil

A single case of zoonotic *Brugia* infection had been reported in Brazil as of 1989. [7]

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Fungal infection - invasive

Agent	FUNGUS. Various (major syndromes such as Candidiasis, Blastomycosis, etc are discussed separately in this module)
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture of blood, urine, biopsy material. Serum antigen or antibody assay in some cases.
Typical Adult Therapy	Antifungal agent(s) directed at known or likely pathogen
Typical Pediatric Therapy	As for adult
Clinical Hints	This diagnosis should be suspected in any patient with evidence of severe local or multisystem infection, particularly in the setting of immune suppression.
Synonyms	Acremonium, Adiaspiromycosis, Allescheriasis, Alternaria, Arthrographis kalrae, Athopsis, Aureobasidium, Bipolaris, Blastobotrys proliferans, Chaetomium, Chrysosporium, Cladophialophora, Cladosporium, Curvularia, Cyphellophora, Dactylaria, Debaryomyces, Dreschslera, Emmonsia, Exophiala, Exserohilum, Fonsecaea, Fungal meningitis, Fungal sepsis, Fusarium, Geosmithia, Geotrichosis, Graphium, Hansenula, Haplomycosis, Hendersonula, Hyalophycomycosis, Kluyveromyces, Lasiodiplodia, Lasiodiplodia, Lecythophora, Malassezia furfur, Monascus, Monosporiosis, Mycocentrospora, Neocosmospora vasinfecta, Neosartorya hiratsukae, Neosartorya udagawae, Ochroconis, Oidiodendron, Paecilomyces, Paraconiothyrium, Phaeoacremonium, Phaeohyphomycosis, Phialophora, Phoma, Pichia, Pseudallescheria, Pseudallescheriasis, Pyrenochaeta, Ramichloridium, Rhinocladiella, Sarcopodium, Scedosporium, Septicemia - fungal, Taeniolella, Thielavia, Trichoderma, Ulocladium, Veronacea, Wallemia. ICD9: 117.6,117.8,117.9,118 ICD10: B43.1,B43.2,B43.8,B48.2,B48.3,B48.7,B48.8

Clinical

Major syndromes (Aspergillosis, Candidiasis, Coccidioidomycosis, Cryptococcosis, Penicilliosis, etc) are discussed elsewhere in this module.

Clinical syndromes associated with systemic fungal infection (in alphabetical order):

Adiaspiromycosis (Haplomycosis) is a pulmonary infection due to *Emmonsia* (*Chrysosporium*) species.
• Most cases have been described in Latin America and Central Europe, with additional reports from Israel and the United States.
• Three forms are recognized: solitary granuloma, localized granulomatous disease and diffuse, disseminated granulomatous disease. [1]

Arthrographis kalrae has been reported as a cause of sinusitis and meningitis in patient with AIDS.

Blastobotrys proliferans is an ascomyetous yeast that has been reported to cause peritonitis in a dialysis patient. [2]

Curvularia inaequalis has been associated with several cases of peritonitis complicating peritoneal dialysis. [3]

Exophiala jaenselmei and ***Rhinocladiella*** species have been implicated in cases of nosocomial fungemia.
• An outbreak of *Exophalia* infection in the United States was associated with contamination of injectable steroids.

Exserohilum is a dematiaceous fungus that has been associated with skin infections, keratitis, systemic infections and sinusitis. [4]

Fusarium often infects the cornea [5] , but may occasionally cause subcutaneous infection, fungemia, pneumonia, arthritis, bursitis, brain abscess and a variety of other systemic infection. [6]
• Pathogenic members of the *Fusarium solani* complex are common in the environment. [7]

Geotrichosis is a rare form of pneumonia and systemic mycosis caused by *Geotrichum candidum*.

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• The organism is ubiquitous in nature and often found in the stool of healthy humans.
• Pulmonary disease simulates tuberculosis; and mucosal infection is similar to moniliasis.

Graphium basitruncatum has been associated with fungemia in a patient with leukemia. [8]

Hansenula species have been implicated in nosocomial infections, endocarditis, fungemia and urinary tract infection

Lasiodiplodia theobromae has been reported to cause keratomycoses. [9]

Neocosmospora vasinfecta, a plant pathogen, has caused at least 3 cases of soft tissue infection (lower extremities, in Senegal) or fatal disseminated infection in immunocompromised humans. [10]

Neosartorya hiratsukae has been implicated in a case of brain abscess.

Penicillium • 31 cases of invasive infection by *Penicillium* species other than *P. marneffei* were reported during 1951 to 2001 • including 12 of pulmonary disease, and 4 prosthetic valve endocarditis.

Phaeohyphomycosis (infection by dematacеous fungi) is manifested as:
• brain abscess (typically *Cladosporium trichoides*; also *Exophiala dermatitidis* [11] , *Fonsecaea pedrosoi*, *Ramichloridium obovoideum*, *Ochroconis gallopavum*, *Chaetomium atrobruneum*, et al),
• sinusitis (*Drechslera*, *Bipolaris*, *Exsorohilum*, *Curvularia*, *Alternaria*, *Cladosporium*)
• subcutaneous infection (typically due to *Exophiala* and *Phialophora* species • occasionally *Fonsecaea*, *Cladosporidium*, *Alternaria*, *Dactylaria*, *Mycocdentrospora*, *Phaeoacremonium* [12] , *Veronaea*, *Cyphellophora pluriseptata*, etc)
• endocarditis.

Pseudoallescheriasis (Petriellidiosis) is caused by *Scedosporium apiospermum* (*Pseudoallescheria boydii*) and may present as mycetoma; or infection of the brain, bone and joints, orbits and other tissues. [13] [14]

Ramichloridium mackenziei has been reported to cause brain abscess in the Middle East.

Sarcopodium oculorum has been implicated as a cause of corneal ulcer in Brazil.

Trichoderma spp. are associated with peritonitis among dialysis patients, and disseminated infection in the immune-suppressed.

Fungal eye infection:
• Fungal endophthalmitis may be exogenous or endogenous.
• Clinically, onset is delayed and more gradual than infection due to bacteria.
• Hyaline fungi:
Fusarium species are implicated in keratitis, scleritis and intraocular infections
Aspergillus in keratitis following industrial trauma or surgery, orbital infection, dacryocystitis, scleritis and endophthalmitis
Scedosporium in keratitis, scleritis, endophthalmitis, orbital infection
Paecilomyces in keratitis, endophthalmitis and intralenticular infections
Acremonium in keratitis and endophthalmitis.
• Dematiaceous fungi
Bipolaris, *Curvularia*, *Exophiala*, *Exserohilum*, *Lecytophora* and *Phialophora* are implicated in keratitis and intraocular infections
*Lasiodiplodia*in keratitis and endophthalmitis.
• Other fungal agents (*Candida*, *Cryptococcus*, *Coccidioides*, *Paracoccidioides*, *Blastomyces*, *Histoplasma*, *Sporothrix*) which may cause ocular infection are discussed separately in this module.

This disease is endemic or potentially endemic to all countries.

Fungal infection - invasive in Brazil

Notable outbreaks:
1996 to 1997 - An outbreak (19 cases) of *Exophiala jeanselmei* fungemia was related to use of contaminated water in a hospital. [15]
1997 (publication year) - An outbreak (24 cases, 0 fatal) of *Hansenula anomala* fungemia was reported in an oncological hospital in Rio de Janeiro. [16]
1998 - An outbreak (8 cases) of *Pichia anomala* fungemia was reported in an intensive care and high risk units of a Nursery. [17]
2002 to 2004 - An outbreak (17 cases) of *Pichia anomala* fungemia in a pediatric intensive care unit was associated with use of central venous catheters. [18]

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2007 - An outbreak (6 cases) of invasive pulmonary aspergillosis was reported among patients hospitalized in a bone marrow transplant ward in Sao Paulo. [19]

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Gastroenteritis - viral

Agent	VIRUS - RNA Calicivirus (Norwalk, Hawaii, Sapporo, Snow Mountain, Norovirus); Torovirus; or Astrovirus
Reservoir	Human
Vector	None
Vehicle	Food Water Shellfish Vegetables
Incubation Period	Norwalk 1d - 2d; astrovirus 3d - 4d
Diagnostic Tests	Demonstration of virus (electron microscopy or stool antigen analysis). Serology. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions; supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Vomiting (less common with Astrovirus), abdominal pain; loose, watery diarrhea lasting 1 to 3 days; no fecal leucocytes; fever in 50% - headache and myalgia in some cases.
Synonyms	Aichi, Astroviridae, Astrovirus, Calicivirus gastroenteritis, Chiba, Cosavirus, Diarrhea, Gastroenterite virale, Hawaii agent gastroenteritis, Klassevirus, Mexico virus, Mini-reovirus, Minireovirus, Norovirus gastroenteritis, Norwalk agent gastroenteritis, Norwalk-like, Parkville virus gastroenteritis, Picobirnavirus, Recovirus, Roskilde disease, Saffold Cardiovirus, Salivirus, Sapovirus, Sapporo, Sapporo-like, Snow Mountain, SRSV gastroenteritis, Toronto virus, Torovirus, Vinterkraksjuka, Viral gastroenteritis, Winter vomiting disease. ICD9: 008.8,008.69,008.62,008.63,008.64,008.65,008.66,008.67 ICD10: A08.1,A08.2,A08.3,A08.4

Clinical

The onset of infection due to the Norwalk virus group may be gradual or abrupt, and is heralded by abdominal cramps with or without nausea.
• In most cases, both vomiting and diarrhea occur. [1]
• Four to eight non-bloody stools are passed per day; and fecal leucocytes are absent.
• 87% of patients with NLV infection develop diarrhea within 5 days; and only 60% of patients with Sapporo-like virus [SLV] infection.
• 59% of children below age 1 year develop vomiting with NLV, and 44% with SLV.
• Myalgias, malaise, headaches and benign febrile seizures [2] [3] may also be present.
• A low-grade fever occurs in 50% of cases.
• Original publications stated that symptoms remit in 48 to 72 hours without sequelae; however, recent studies suggest that illness usually persists for 5 to 6 days.
• The duration of illness has been correlated with fecal concentration of virus.
• Residual dyspepsia, constipation or gastroesophageal reflux disease may persist following Norovirus infection. [4]
• Cases of Guillain-Barre syndrome [5] and necrotizing enterocolitis in newborn infants have been ascribed to Norovirus infection. [6] [7]
• Review of the clinical features of fatal Norovirus infection • see reference [8]

Astrovirus diarrhea is similar to NLV infection; however, the former is characterized by a milder illness and lower incidence of vomiting. [9]

Rare instances of meningitis have been associated with Saffold virus infection. [10]

This disease is endemic or potentially endemic to all countries.

Gastroenteritis - viral in Brazil

Prevalence surveys:

Enteric Adenoviruses were responsible for 1.55% of diarrhea among children below age 5 years (1998 to 2000) [11]

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Enteric Adenoviruses were responsible for 10% of severe diarrhea among children in Sao Paulo (1993 publication) [12]

Adenoviruses were found in 2% of children hospitalized with acute gastroenteritis in Rio de Janeiro (1996 to 2003), and 2% of children living in the slums of Salvador with acute gastroenteritis (2001 to 2003) [13]

Adenoviruses were found in 6.4% of children below age 5 with diarrhea and 1.7% of healthy controls, in Rondonia (2007 publication) [14]

Adenoviruses were found in 3.6% of hospitalized children ages <= 3 years with diarrhea, Astrovirus 3.1%, Calicivirus 7.6% (Mato Grosso do Sul, 2000 to 2004) [15]

Norovirus was found in 20% of children hospitalized with diarrhea, and mixed Rotavirus / Norovirus infection in 4% (Rio de Janeiro, 2004) [16]

Norovirus was found in 14.5% of children with gastroenteritis in Rio de Janeiro (1998 to 2005) [17]

Norovirus was found in 35.1% of Rotavirus-negative stool samples from patients with gastroenteritis (Rio de Janeiro, 2005 to 2008) [18]

Norovirus was found in 15% of children with gastroenteritis in Recife (2004 to 2005) [19]

Norovirus was found in 29.2% of hospitalized children ages ? 5 years with acute gastroenteritis in Sao Paulo, Brazil (2004 to 2009) [20]

Norovirus was found in 44.4% of children hospitalized for acute gastroenteritis in Belem, Para (2013 publication) [21]

Norovirus was found in 33.4% of children in a day-care center with acute gastroenteritis, Rotavirus 16.1% and Astrovirus 6.3% (Rio de Janeiro, 1994 to 2008) [22]

Norovirus was found in 39.7% of children hospitalized with gastroenteritis in Vitoria city (2004 to 2006) [23]

Norovirus was found in 17% of children age <=3 years with diarrhea, vs. 13% of asymptomatic controls, in Vitoria (2003 to 2004) [24]

Norovirus (G1) was found in 7.5% of seawater samples along beaches in Florianopolis, Santa Catarina (2009 to 2010) [25]

Norovirus or enteric Adenovirus coinfection was found in 14% of rotavirus-positive samples from hospitalized patients (2012 publication) [26]

Norovirus or Rotavirus were found in 0% of recently-captured primates (Red-howler *Alouatta guariba clamitans*; Howler, *Alouatta caraya*; Marmosets, *Callithrix* spp. *Callithrix penicillata*; Black-faced lion tamarin, *Leontopithecus caissara*) in southern Brazil (2012 publication) [27]

Norovirus was found in 0% of water samples in Condordia, Santa Catarina, and Adenovirus in 100% (2012 publication) [28]

Norovirus was found in 18.8% of recreational waters in a lagoon, Rotavirus 24.3% and Adenovirus 18.7% (Rio de Janeiro, 2012 publication) [29]

Calicivirus was found in 9% of infantile gastroenteritis in Salvador (2003 to 2003) [30]

Calicivirus was found in 10% of HIV-positive patients with diarrhea, and 0% of control patients (southeastern Brazil, 2006 to 2007) [31]

Calicivirus and / or Norovirus was found in 22.6% of women in Goiania, Goias (2006 to 2007) [32]

Astrovirus was found in 5.0% of diarrhea among urban children in Salvador, Adenovirus 2.9%, Reovirus 0.7% (2000 to 2002) [33]

Astrovirus was found in 5.4% of children with gastroenteritis. At 24 months of age, 30.6% of children had been infected. (Belem, 1990 to 1992) [34]

Astrovirus was found in 14% of stool samples from children up to 5 years of age hospitalized with acute gastroenteritis, of which 6% were coinfected with Rotavirus. (Rio de Janeiro, 2004) [35]

Astrovirus was found in 5.3% of hospitalized and nonhospitalized children with diarrhea. (Rio de Janeiro, 1998 to 2005) [36]

Human Bocavirus (HBoV) was found in 2% of children below age 16 with gastroenteritis (2003 to 2005) [37]

HBoV was found in 5.8% of children with acute gastroenteritis (west-central region, 1994 to 2005) [38]

Astrovirus and Norovirus were found in 15.4% and 5.8%, respectively of water samples in the area of Manaus; Adenoviruses in 30.8% (2007 publication) [39]

Parechoviruses were identified in 16.1% of children below age 6 years with enteritis - including a novel strain, HPeV-8 (Salvador, 2006 to 2007) [40]

Seroprevalence surveys:

71% of children living in a northern shantytown were seropositive toward Norwalk-like virus (2000 publication) [41]

Seropositivity toward Norwalk virus among Amazon Amerindians ranged from 39% (Maiogong) to 100% (Kubenkrankrein) (1994 publication) [42]

Notable outbreaks:

1994 (publication year) - An outbreak (5 cases) of Astrovirus gastroenteritis was reported in a family in Sao Paulo. [43]

1996 - An outbreak (27 cases) of Astrovirus gastroenteritis was reported at a day care center in Rio de Janeiro. [44]

2004 - An outbreak (100 cases or more) of Astrovirus gastroenteritis was reported among Maxakali Indians in Southeast Brazil. [45]

2005 - An outbreak of pediatric gastroenteritis was reported in public day care centers in Paraiba valley. Norovirus was detected in 66% of samples. [46]

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2010 - An outbreak of Norovirus gastroenteritis was reported in Sao Paulo [47] ; and outbreaks of Rotavirus and Norovirus infection were reported at two correctional facilities. [48]

2010 - An food-borne outbreak of Norovirus gastroenteritis was reported aboard a cruise ship. [49]

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Gianotti-Crosti syndrome

Agent	UNKNOWN
Reservoir	Unknown
Vector	None
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Clinical features and skin biopsy findings.
Typical Adult Therapy	None
Typical Pediatric Therapy	None
Clinical Hints	Generalized skin eruption involving the extremities, face and buttocks; lymphadenopathy of the axillae and inguinal region; anicteric hepatitis; resolves in 15 to 42 days. Rare outbreaks have been reported.
Synonyms	Acrodermatitis papulosa infantilis, Papular acrodermititis of childhood, Papulovesicular acrolocated syndrome. ICD9: 693.0 ICD10: L27.8

Clinical

Most patients are in the age group 2 to 6 years; however, the disease has occasionally been reported in infants and young adults. [1] [2]

Clinical features are largely limited to discrete flat-topped papules on the face, extensor surfaces of the extremities and buttocks. [3]
• The eruption is symmetrical, occasionally pruritic, either skin-colored or erythematous, and evolves over a period of two to three days.
• The skin lesions measure 2 to 4 mm in diameter, with a tendency for larger lesions among young children. [4]
• Koebner phenomenon has been described.
• In most cases, the exanthem resolves after 15 to 20 days, but may persist for as long as 5 weeks.
• Hemorrhagic skin lesions and petechiae have been described in some cases. [5]
• Prominent lymphadenopathy is noted, primarily in the inguinal and axillary regions.
• Hepatomegaly and anicteric hepatitis are common.

Gianotti-Crosti syndrome may be the only presenting manifestation of Epstein-Barr virus infection. [6]

The features of Gianotti-Crosti syndrome may mimic those of atopic dermatitis. [7]

The diagnosis is confirmed by skin biopsy, which reveals spongiosis of the upper epidermis and upper dermis, with perivascular lymphocytic and histiocytic infiltrates. [8]

This disease is endemic or potentially endemic to all countries.

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Giardiasis

Agent	PARASITE - Protozoa. Archezoa, Metamonada, Trepomonadea. Flagellate: Giardia lamblia [G. intestinalis, G. duodenalis]
Reservoir	Human Beaver Muskrat Dog Cat Carnivores Sheep Goat Horse Cattle
Vector	None
Vehicle	Food Water Fecal-oral Fly
Incubation Period	1w - 3w (range 3d - 6w)
Diagnostic Tests	String test (gelatin capsule containing string). Stool microscopy or antigen assay. Nucleic acid amplification.
Typical Adult Therapy	Metronidazole 250 mg PO TID X 5d. OR Nitazoxanide 500 mg PO BID X 3d OR Tinidazole 2 g PO X1. OR Furazolidone 100 mg PO QID X 7d. OR Paromomycin 10 mg/kg PO TID X 7d OR Quinacrine 100 mg PO TID X 5d
Typical Pediatric Therapy	Metronidazole 5 mg/kg PO TID X 5d. OR Tinidazole 50 mg PO X 1 (maximum 2g). OR Furazolidone 1.5 mg/kg QID X 7d OR Nitazoxanide: Age 1 to 3y 100 mg BID X 7 d Age 4 to 11y 200 mg BID X 7d
Clinical Hints	Foul smelling, bulky diarrhea, nausea and flatulence; may "wax and wane"; weight loss and low-grade fever are common.
Synonyms	Beaver fever, Giardia duodenalis, Giardia intestinalis, Giardia lamblia, Lambliasis. ICD9: 007.1 ICD10: A07.1

Clinical

The usual interval between infection and the onset of acute symptoms ranges from one to two weeks.

In most instances, the individual will experience sudden explosive, watery, foul-smelling diarrhea; excessive gas; abdominal pain; bloating; nausea; asthenia; and anorexia. [1]
- Symptoms consistent with irritable bowel syndrome and functional dyspepsia are reported in 80.5% and 24.5% of patients, respectively [2]
- Upper gastrointestinal symptoms such as vomiting may predominate. [3]
- Fever is unusual, and asymptomatic infection is common.
- Blood or mucus in the stool is rare, and there is neither leucocytosis nor eosinophilia.

Occasionally, the illness may last for months, or even years, causing recurrent episodes of impaired digestion, lactose intolerance, diarrhea, depression, asthenia and weight loss. [4-8]
- Recurrence of symptoms is also common following effective treatment. [9]
- Severe and prolonged infections are reported among patients with IgA deficiency and malnutrition.
- Infection in children may result in stunted growth, delayed development [10] [11] and vitamin A deficiency. [12]

Sequelae:
- Reactive arthritis may occasionally follow infection by *Giardia intestinalis*. [13]
- Giardiasis has been implicated in the etiology of irritable bowel syndrome and chronic fatigue syndrome. [14]

This disease is endemic or potentially endemic to all countries.

Giardiasis in Brazil

27.4% of children living in slums were found to acquire *Giardia* infestation when followed for at least 3 months (Fortaleza, 1989 to 1993). [15] [16]

Prevalence surveys:

8.9% of school children in Salvador
14% of school children in Lages (2004 publication) [17]
24% of school children in Caxias do Sul, RS (2008 publication) [18]

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78.3% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [19]
27.5% of children in Uberlandia, Minas Gerais (2008 publication) [20]
5.5% of Sao Paulo children below age 5 years (1995 to 1996) [21]
8.2% of first grade school children in Campinas (1997 publication) [22]
13.5% of children (ages 1 to 4 years) in Salvador, Bahia State (2008 publication) [23]
13% of children ages 3 to 6 years in day-care centers (Salvador, 2012 publication) [24]
7.3% of children in Sao Paulo (2004 publication) [25]
16.7% of children in an urban slum in Sao Paulo (2008 publication) [26]
14.1% of children ages 1 to 4in 2003; 5.3% during 2003 to 2004 (Salvador) [27]
14.6% of urban children with diarrhea in Salvador (2000 to 2002) [28]
9.3% of children with diarrhea in northeastern Brazil (2008 publication) [29]
4.7% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [30]
9.9% of children hospitalized for diarrhea (Goiania, Goias State, 2007 publication) [31]
18% of children living in sub-standard settlement areas. (Juiz de For a, Minas Gerais State, 2007 publication) [32]
26.88% of children in day care centers in Botucatu, Sao Paulo (2006 publication) [33]
15% of children in a day care center in Sao Paulo (2009 publication) [34]
21.4% of children in day care centers in Sao Paulo (2003) [35]
9.5% of children in public daycare centers in Belo Horizonte, Minas Gerais (2008 publication) [36]
23.7% (2002) and 21.4% (2003) of children at 5 municipal daycare centers. (Botacatu, Sao Paulo, 2002 to 2003) [37]
51.8% of children in day care centers in Araguari, Minas Gerais (2007 to 2008) [38]
21.5% of children presenting to outpatient clinics in Manaus (2009 publication) [39]
1% of persons in Eirunepe, Amazon (2005 publication) [40]
4.0% of elderly residents at long-term residency institutions in southeastern Brazil (2013 publication) [41]
48.6% of the Parakana indigenous community in southwestern Para State (1992 to 1995) [42]
33.3% of Hupda and 10.7% of other indigenous peoples in Iauarete (2001) [43]
8% of Terena Indians (Mato Grosso do Sul) [44]
9.8% of persons in Uberlandia (Minas Gerais, 1998)
6.3% of persons in Maria Helena, Parana (2004 to 2006) [45]
12.5% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [46]
5.9% of persons in Campos do Jordao (2003 to 2004) [47]
19.6% of persons in an agricultural settlement in the Amazon Basin (Granada, Acre State, 2007 publication) [48]
5% of HIV-positive patients with diarrhea, and 0% of control patients (southeastern Brazil, 2006 to 2007) [49]
7.9% of HIV-positive patients before HAART vs. 1% after HAART (Ceara, 2008 publication) [50]
3.5% of HIV-positive patients in northeastern Sao Paulo (1998 to 2008) [51]
0.5% of adult renal transplant recipients (Porto Alegre, 2001 to 2005) [52]
8.4% of food handlers in Cascavel, Parana (2009 publication) [53]
24% of shelter dogs in Curituba, Parana State (2008 publication) [54]
0.8% of fecal samples from dogs in public squares in Itabuna, Bahia (2008 publication) [55]
17.3% of dogs in south-central Sao Paulo State (2011 publication) [56]
7.5% of dairy cattle in Sao Paulo State (2012 publication) [57]
42.8% of river water in the Ivai Indigenous Land (2008 publication) [58]
46.1% of water samples from watershed catchments and treated water sources (2010 publication) [59]
27% of water sources in Sao Paulo (2004 publication) [60]
62.5% of water sources in Sao Paulo (2011 publication) [61]
42% of sewage samples in Sao Paulo (2011 publication) [62]
49.5% of surface water samples in Sao Paulo (2012 publication) [63]
87.5% of surface water samples in Campinas (2005 to 2006) [64]

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Glanders

Agent	BACTERIUM. Burkholderia mallei An aerobic gram negative bacillus
Reservoir	Horse Mule Donkey
Vector	None
Vehicle	Infected secretions Contact
Incubation Period	5d - 14d (range 1d - 21d)
Diagnostic Tests	Culture of blood or exudate. Serology.
Typical Adult Therapy	Sulfonamides 25 mg/kg QID (up to 6g) X 3w
Typical Pediatric Therapy	As for adult
Clinical Hints	Ulcerating skin nodule with lymphangitis; or fever, myalgia, pneumonia and pleuritis; history of contact with horses in most cases.
Synonyms	Burkholderia mallei, Farcy, Rotz. ICD9: 024 ICD10: A24.0

Clinical

Four clinical forms of glanders are described: septicemia, pulmonary infection, acute localized infection and chronic infection
• One form of the disease may progress to another

Localized infections are characterized by nodules, abscesses and ulcers in the mucous membranes, skin or subcutaneous tissues at the site of inoculation.
• Dermal nodules are white or gray and firm, with a caseous or calcified center. They are surrounded by areas of inflammation, and may progress to gangrene. [1]
• Mucous membranes may exhibit a mucopurulent or blood-tinged discharge. Nodules and deep ulcers may also develop in the mucosa of the nasal septum and nasal turbinates.
• Lesions are accompanied by fever, sweats and swelling of regional lymph nodes which may suppurate.
• Mucosal or skin infections may disseminate, resulting in a papular or pustular rash and abscesses of the liver, spleen, lungs, subcutaneous tissues and muscles.

Lung infection results from either inhalation of *B. mallei* or hematogenous spread.
• The patient experiences acute onset of fever, diaphoresis, cough, chest pain and dyspnea.
• Pulmonary lesions are characterized by small nodules which have caseous or calcified centers surrounded by inflammatory zones.
• Untreated pulmonary disease often progresses to septicemia.

This disease is endemic or potentially endemic to 23 countries.

Glanders in Brazil

During 1993 to 2012, glanders was reported among equines in 15 states in Brazil. [2]

2000 - Cases of equine glanders were reported in the states of Pernambuco, Alagoas, Sergipe and Ceara. [3]
2004 - Cases of equine glanders were reported in the states of Paranha and Santa Catarina. [4]
2007 - Equine glanders was reported in Ceara. [5]
2010 - An infected horse was reported in Distrito Federal. [6]
2012 - Five infected equines were reported in Ceara, and five in Minas Gerais. [7] [8]

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Gnathostomiasis

Agent	PARASITE - Nematoda. Phasmidea: Gnathostoma spinigerum (rarely G. hispidum, G. doloresi and G. nipponicum)
Reservoir	Cat Dog Poultry Frog Fish
Vector	None
Vehicle	Food Fish Amphibian Reptile
Incubation Period	3w - 4w (range 2d - 1y)
Diagnostic Tests	Identification of larva in tissue. Serological testing in specialized laboratories.
Typical Adult Therapy	Albendazole 400 mg daily for 21 days has been recommended as an adjunct to surgical excision Ivermectin, 200 ug/kg PO as a single dose has also been advocated.
Typical Pediatric Therapy	As for adult
Clinical Hints	Follows ingestion of raw meat, poultry, fish or frog; migratory nodules of skin, soft tissues, brain or eye; eosinophilia; parasite may survive for more than 10 years in human tissue.
Synonyms	Gnathostoma, Gongylonematiasis, Larva migrans profundus, Nodular migratory eosinophilic panniculitis, Physaloptera, Spiruroid larva migrans, Wandering swelling, Yangtze edema. ICD9: 128.1 ICD10: B83.1

Clinical

Initial symptoms may include nausea, abdominal pain or urticaria.
• The presence of worms in skin or soft tissue results in migratory, pruritic or painful swellings which may be erythematous and attain a size of several centimeters. [1]
• Swellings may last for 1 to 4 weeks in a given area, and then reappear in a new location • a pattern which can continue for months or years.
• Findings of central nervous system infection (less than 1% of patients with subcutaneous gnathostomiasis) have included radiculopathy, meningitis, encephalitis, subarachnoid or intercerebral hemorrhage or paralysis. [2-4]
• Other syndromes include eye infestation [5-10] , persistent abdominal pain with hepatomegaly, or pneumonitis. [11]
• 74 cases of intra-ocular gnathostomiasis had been reported as of 2012 • including 14 from India. [12]
• Eosinophilia is prominent. Eosinophiles may also be found in CSF and pleural effusions. [13]

This disease is endemic or potentially endemic to 22 countries. Although Gnathostomiasis is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Gnathostomiasis in Brazil

A Brazilian traveler acquired gnathostomiasis in Peru (2009 publication) [14]

A subsequent case of autochthonous gnathostomiasis was reported (2012 publication) [15]

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Gonococcal infection

Agent	BACTERIUM. Neisseria gonorrhoeae An aerobic gram-negative coccus
Reservoir	Human
Vector	None
Vehicle	Sexual contact Childbirth Exudates
Incubation Period	2d - 7d
Diagnostic Tests	Smear (male), culture. Consult laboratory for proper acquisition & transport. Nucleic acid amplification.
Typical Adult Therapy	Ceftriaxone 250 mg IM X 1. PLUS Azithromycin 1 g PO as single dose.
Typical Pediatric Therapy	Weight <=45 kg: Ceftriaxone 125 mg IM X 1 Weight >45 kg: as for adult. PLUS Azithromycin
Clinical Hints	Copious urethral discharge (male) or cervicitis beginning 2 to 7 days after sexual exposure; PID; fever, painful pustules and suppurative arthritis (primarily encountered in postmenstrual females).
Synonyms	Blennorragie, Blenorragia, Gonococcemia, Gonore, Gonorre, Gonorrea, Gonorrhea, Gonorrhee, Gonorrho, Gonorrhoe, Infeccion gonococica, Infeccoes gonococicas, Neisseria gonorrhoeae. ICD9: 098 ICD10: A54

Clinical

Gonorrhea:
Gonorrhea in males typically presents as urethral discomfort, dysuria, and discharge.
• The degree of discomfort and discharge are variable.
• Asymptomatic infection is common among females, but may also occur in males [1]
• Gonococcal epididymitis presents with unilateral pain and swelling localized posteriorly within the scrotum.
• Gonorrhea in the female are usually manifest as vaginal discharge and endocervicitis.
• The discharge is thin, purulent and mildly odorous.
• Dysuria or a scant urethral discharge may be present.
• Non-gonococcal urethritis, including infection by *Chlamydia trachomatis* and other *Neisseria* species [2] may mimic gonococcal infection.
• Infection can be passed to the male urethra from the pharynx through fellatio. [3]
• Levels of serum Prostate-specific Antigen (PSA) may be elevated in patients with gonorrhea. [4]

Gonococcal PID:
Pelvic or lower abdominal pain suggests infection of the endometrium, fallopian tubes, ovaries or peritoneum.
• Pain may be midline, unilateral, or bilateral.
• Fever and vomiting may be present.
• Right upper quadrant pain from perihepatitis (Fitz-Hugh-Curtis syndrome) may occur following the spread of organisms upward along peritoneal planes to the hepatic capsule [5-7] (The syndrome is also reported as a complication of gonorrhea in males) [8]

Other clinical forms: [9]
Gonococcal proctitis is often asymptomatic, but rectal pain, pruritus, tenesmus, bloody diarrhea and rectal discharge may be present.
Gonococcal pharyngitis may be asymptomatic, or associated with severe inflammation. *Neisseria gonorrhoeae* is often present in throat specimens from patients with urethritis. [10]
Gonococcal conjunctivitis is usually unilateral in adults; however, neonatal infection (ophthalmia neonatorum) involves both eyes.
• Symptoms include pain, redness, and a purulent discharge and may result in blindness.
• Rare instances of corneal perforation are reported. [11]
Disseminated gonococcal infection is characterized by joint or tendon pain, of single or multiple joints. [12]
• Severe pain, swelling, and decreased mobility in a single joint (usually the knee) suggest purulent arthritis.
• Tenosynovitis is common, usually affecting the small joints of the hands.
• A rash is present in 25% of patients with gonococcemia.
• Additional complications include meningitis, endocarditis, septic shock with ARDS [13] , subcutaneous abscess, Fournier's

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gangrene [14] , pyomyositis [15] and other localized infections. [16]

This disease is endemic or potentially endemic to all countries.

Gonococcal infection in Brazil

2,464,000 cases were estimated for 1996.

Prevalence surveys:

6.4% of women in rural Alagoas (1997)
1.9% of female adolescents in Vitoria (2004 publication) [17]
9.5% of young women in Rio de Janeiro seeking HIV testing (2004 publication) [18]
2% of women ages 14 to 49 attending a primary health unit (2008 publication) [19]
1.9% of women ages 18 to 40 years, in Sao Paulo (2011 publication) [20]
1.0% of women ages 15 to 24 years, attending public hospitals (2009) [21]
4.0% of cervical specimens from patients attending public health services in Belo Horizonte and Contagem, Minas Gerais (2011 publication) [22]
0% of candidates for in-vitro fertilization (Sao Paulo, 2008 to 2009) [23]
1.5% of sexually-active urban women ages 16 to 23 (southern Brazil, 2011 publication) [24]
1.2% of sexually-active women ages 12 to 49 in Ceara, Northern Brazil (2007 publication) [25]
2.1% of sexually-active women ages 15 to 19 (2009 publication) [26]
3% of women ages <24 years attending a family planning clinic (Campinas, 2009 publication) [27]
7.6% of female prisoners in Espirito Santo State (2000 publication) [28]
1.5% of pregnant women in Manaus, Fortaleza, Goiania, Rio de Janeiro, Sao Paulo and Porto Alegre (2004 to 2005) [29]
2.2% of high school students in Rio Branco-Acre (2000 publication) [30]
1.1% of men and 0.3% of women in Alto Solimoes, Amazon border region (2012 publication) [31]
18.4% of male STD patients (2005) [32]
12.9% of AIDS patients (1986 to 1992) [33]
0% of HIV-positive women in Salvador, Bahia (2012 publication) [34]

During 2006 to 2010, 16.5% of *Neisseria gonorrhoeae* isolates in Rio de Janeiro were found to be quinolone-resistant. [35]

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Granuloma inguinale

Agent	BACTERIUM. Klebsiella granulomatis (formerly Calymmatobacterium granulomatis) An gram-negative bacillus
Reservoir	Human
Vector	None
Vehicle	Sexual contact Direct contact
Incubation Period	7d - 30d (range 3d - 1 year)
Diagnostic Tests	Identification of organism in stained smears. Culture in specialized laboratories (HEp-2 cells).
Typical Adult Therapy	Doxycycline 100 mg BID PO X 3w. Alternatives: Azithromycin 1 g daily X 3 w. Sulfamethoxazole/ trimethoprim 800/160 mg BID X 3w Erythromycin 500 mg QID X 3w.
Typical Pediatric Therapy	Doxycycline 2 mg/kg BID X 2 to 3w (above age 8). Alternatives: Sulfamethoxazole/trimethoprim , Erythromycin or Azithromycin
Clinical Hints	Slowly expanding, ulcerating skin nodule with friable base; usually painless; may be complicated by edema or secondary infection - rarely spreads to bone or joints.
Synonyms	Calymmatobacterium granulomatis, Donovanosis, Granuloma genitoinguinale, Granuloma inguinale tropicum, Granuloma venereum, Sixth venereal disease. ICD9: 099.2 ICD10: A58

Clinical

The primary lesion of granuloma inguinale appears on the perineum or genitals in 80% to 90% of cases.
• Infection begins as a small painless papule or indurated nodule which progresses to a painless beefy-red ulcer with rolled edges and a friable surface.
• Multiple ulcers may coalesce, and new lesions may also form through autoinoculation. [1]
• Scar formation, deformity, keloids and lymphedema may develop. [2]
• The most common sites of infection are the prepuce, coronal sulcus, and penile shaft; the labia and the fourchette .
• Rectal lesions may follow anal intercourse.
• Systemic disease of bones, joints, liver and lymphatics is rare, and may follow infection of the uterine cervix.
• Granuloma inguinale may present as mass lesions which mimic malignancy [3] or elephantiasis [4] ; and cutaneous metastases from mucinous carcinoma may mimic granuloma inguinale. [5]

This disease is endemic or potentially endemic to all countries.

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Group C viral fevers

Agent	VIRUS - RNA. Bunyaviridae, Orthobunyavirus. At least 10 human pathogens described
Reservoir	Rodent Marsupial ? Bat
Vector	Mosquito (Culex, Aedes, Limatus, Wyeomyia, Coquillettidia, Mansonia and Psorophora spp)
Vehicle	None
Incubation Period	3d - 12d
Diagnostic Tests	Viral culture (blood). Serology. Biosafety level 2.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Self limited febrile illness, often with myalgia, photophobia and conjunctivitis; acquired while working or residing in forested areas.
Synonyms	Apeu, Caraparu, Itaqui, Madrid, Marituba, Murutucu, Nepuyo, Oriboca, Ossa, Restan. ICD9: 066.3 ICD10: A92.8

Clinical

Abrupt onset of fever (lasting 2-5 days) is associated with severe headache, vertigo, nausea, myalgia and arthralgia.
- A rash may occur.
- Additional findings of cough and eye pain have been described in patients with Murutucu fever.

This disease is endemic or potentially endemic to 9 countries.

Group C viral fevers in Brazil

The local strains are: Apeu, Caraparu [1] , Guama [2] , Itaqui [3] , Marituba, Murutucu, Nepuyo [4] and Oriboca. [5]

Seroprevalence surveys:

4.9% of inhabitants of Rio Branco, Acre (Caraparu virus, 1999). [6]

Seropositive birds have been identified in Sao Paulo State (Caraparu virus, 1978 to 1990) [7]

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Hantavirus infection - Old World

Agent	VIRUS - RNA. Bunyaviridae, Hantavirus - Old world : Hantaan, Puumala, Dobrava/Belgrade, Saaremaa & Seoul viruses
Reservoir	Field mouse (Apodemus agrarius-Hantaan) Vole (Myodes glareolus-Puumala) Rat (Rattus norvegicus-Seoul) ? Bat ? Bird
Vector	None
Vehicle	Animal excreta
Incubation Period	12d - 21d (range 4d - 42d)
Diagnostic Tests	Serology. Viral culture. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive. Suggest Ribavirin: 1g IV q6h X 4d, then 0.5g q6h X 6d
Typical Pediatric Therapy	Supportive. Suggest Ribavirin
Vaccine	Hantavirus [old world]
Clinical Hints	Headache, backache, myalgia, diarrhea, vomiting, conjunctivitis, hemorrhage and azotemia; proteinuria and thrombocytopenia common; history of local rodent infestation may be elicited; case-fatality rates 0.1% (Puumala) to 15% (Belgrade).
Synonyms	Acute epidemic hemorrhagic fever, Bosnian hemorrhagic fever, Churilov disease, Dobrava/Belgrade, Endemic benign nephropathy, Epidemic hemorrhagic fever, Far eastern hemorrhagic fever, Haemorrhagic nephrosonephritis, Hantaan, Hemorrhagic fever & renal syndrome, Infectious hemorrhagic fever, Khabarovsk, Korean hemorrhagic fever, Mouse fever, Muju, Muroid virus nephropathy, Nephropathia epidemica, Puumala, Rodent-borne viral nephropathy, Saaremaa, Sandinavian epidemic nephropathy, Sangassou, Seoul, Sochi virus, Songo fever, Sorkfeber, Thailand virus, Thottapalayam, Topografov, Tula, Viral hemorrhagic fever, Viral hemorrhagic fevers. ICD9: 078.6 ICD10: A98.5

Clinical

The course of severe Hemorrhagic Fever and Renal Syndrome (HFRS • Nephropathia epidemica [1]) involves five overlapping stages: [2]
• febrile
• hypotensive
• oliguric
• diuretic
• convalescent

Febrile stage:
It is not uncommon for one or more of these stages to be inapparent or absent. [3] [4]
• The onset of the disease is sudden, with intense headache, backache, fever, and chills.
• Hemorrhage is manifested during the febrile phase as a flushing of the face or injection of the conjunctiva and mucous membranes.
• A petechial rash may appear on the palate and axillary skin folds.
• Extreme albuminuria, typically appearing on the fourth day, is characteristic of severe HFRS.
• Severe renal disease [5] is more likely in patients with thrombocytopenia. [6]

Hypotensive stage:
As the febrile stage ends, hypotension may develop and last for hours to days, accompanied by nausea and vomiting.
• One-third of deaths occur during this phase, related to vascular leakage and shock.
• The severity and mortality rate for Seoul virus infection are reported to be lower than those for Hantaan virus infection. [7]

The final (convalescent) stage can last weeks to months.

Additional findings:
• Electrocardiographic abnormalities are present in 57% of patients with Puumala virus infection [8] [9] and patients who recover from the infection are at increased risk for death from cardiovascular disease. [10]
• Approximately 50% of deaths occur during the subsequent (oliguric) phase.

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• Both interstitial nephritis and glomerulonephritis are encountered. [11]
• Patients who survive and progress to the diuretic phase show improved renal function but may still die of shock or pulmonary complications.
• Overt lung disease suggestive of Hantavirus pulmonary syndrome has been reported in patients infected with Puumala virus. [12]
• Residual hypopituitarism may follow severe Dobrava [13] or Puumala virus infection. [14]
• Visual impairment is common [15] , and acute angle-closure glaucoma has been reported in some cases. [16]
• Additional findings have included pancreatitis, cholangitis, myocarditis, pericarditis, orchitis [17] , meningitis, cerebritis and gastrointestinal hemorrhage. [18]
• Severe central nervous system manifestations of Puumala virus infection appear to be most common among young male patients. [19]

Case-fatality rates range from less than 0.1% for Hemorrhagic Fever Renal Syndrome [HFRS] caused by Puumala [PUU] virus to approximately 5% to 10% for HFRS caused by Hantaan [HTN] virus.

Scrub typhus and hemorrhagic fever with renal syndrome (HFRS) often coexist in areas of Asia. [20]
• Retro-orbital, lumbar, or flank tenderness; proteinuria; and microscopic hematuria, and hemorrhagic manifestations are most common in HFRS.
• Dermal eschar, regional lymphadenopathy, and maculopapular rash should suggest scrub typhus.
• Gastrointestinal symptoms of Hantavirus infection may mimic those of acute appendicitis. [21]

This disease is endemic or potentially endemic to 93 countries. Although Hantavirus infection - Old World is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Hantavirus infection - Old World in Brazil

Serologic evidence for infection by Seoul and Puumala viruses has been found in rural areas of Amparo Island, Paranagua Bay, Parana State and Ribeira Valley (Sao Paulo). [22]
- [In retrospect this may be a cross reaction with Hantavirus pulmonary syndrome viruses - discovered after these data were published]

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Hantavirus pulmonary syndrome

Agent	VIRUS - RNA. Bunyaviridae, Hantavirus: Sin Nombre, Black Creek Canal, Bayou, New York-1, Andes, et al
Reservoir	Rodent - Deer mouse (Peromyscus maniculatus); ? Pinon mouse Harvest mouse Cotton rat Brush mouse Western chipmunk Rarely Human
Vector	None
Vehicle	Animal excreta Rarely bite, direct contact, or person-to-person
Incubation Period	9d - 33d
Diagnostic Tests	Serology. Viral culture. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive Ribavirin may be effective against Andes virus
Typical Pediatric Therapy	As for adult
Clinical Hints	Rapidly progressive illness characterized by fever, myalgia, bilateral interstitial infiltrates and hypoxia; exposure history (agriculture, hiking, exposure to rodents) often elicited; case-fatality rate 45% to 50%.
Synonyms	Anajatuba virus, Andes, Araraquara-like, Ararquara, Bayou, Black Creek Canal, Bormejo, Calabazo, Cano Delgado, Carrizal, Castello dos Sonhos, Castelo dos Sonhos, Castelos dos Sonhos, Central Plata, Choclo, El Moro Canyon, Hantavirus resp. distress syndrome, HU39694, Huitzilac, Jabora, Juquitiba, Laguna Negra, Limestone Canyon, Maporal, Maripa, Monongahela, Montano, Muerto Canyon, New York virus, Oran, Paranoa, SCPH, Sin nombre, Sindr. cardiopulmonar por hantavirus. ICD9: 078.89 ICD10: B33.4

Clinical

The clinical case definition consists of fever >38.3 C and bilateral diffuse pulmonary infiltrates with respiratory compromise which requires supplemental oxygen within 72 hours of hospitalization.

Over 93% of cases reported in the United States have been above the age of 17 • and infection below the age of 10 years is exceptionally rare (2010 publication). [1]

The typical illness is characterized by fever, chills, headache and occasionally gastrointestinal symptoms. [2] [3]
• Five days after onset, patients develop dyspnea, with rapid progression to pulmonary edema / ARDS [4] within as little as 24 hours.
• Clinical features at this stage may include dyspnea, tachycardia, hypotension, metabolic acidosis, lymphocytopenia, hemoconcentration [5] , leukocytosis, thrombocytopenia and azotemia. [6] [7]
• Fatal cases are defined as unexplained fatal respiratory illness with noncardiogenic pulmonary edema.
• As of 2006, the case-fatality rate in the United States has ranged from 36% to 38%.
• Andes virus infection carries a case fatality rate of 37% in Chile [8] ; infection in Brazil as high as 54.3%.
• Person-to-person transmission occurs; thus appropriate isolation procedures are required. [9-14]

Rare instances of Andes virus encephalitis have been reported. [15]

Overt lung disease suggestive of Hantavirus pulmonary syndrome has been reported in patients infected with Puumala virus. [16]

This disease is endemic or potentially endemic to 14 countries.

Hantavirus pulmonary syndrome in Brazil

Time and Place:
The country's first three cases (two fatal) were reported from Sao Paulo in 1993. [17] [18]
- A cumulative total of 99 cases were reported to December 2000 [other sources list 65 cases (39 fatal)] - including 21 in Sao Paulo, 29 in Parana, 14 in Minas Gerais and 18 in Rio Grande do Sul.

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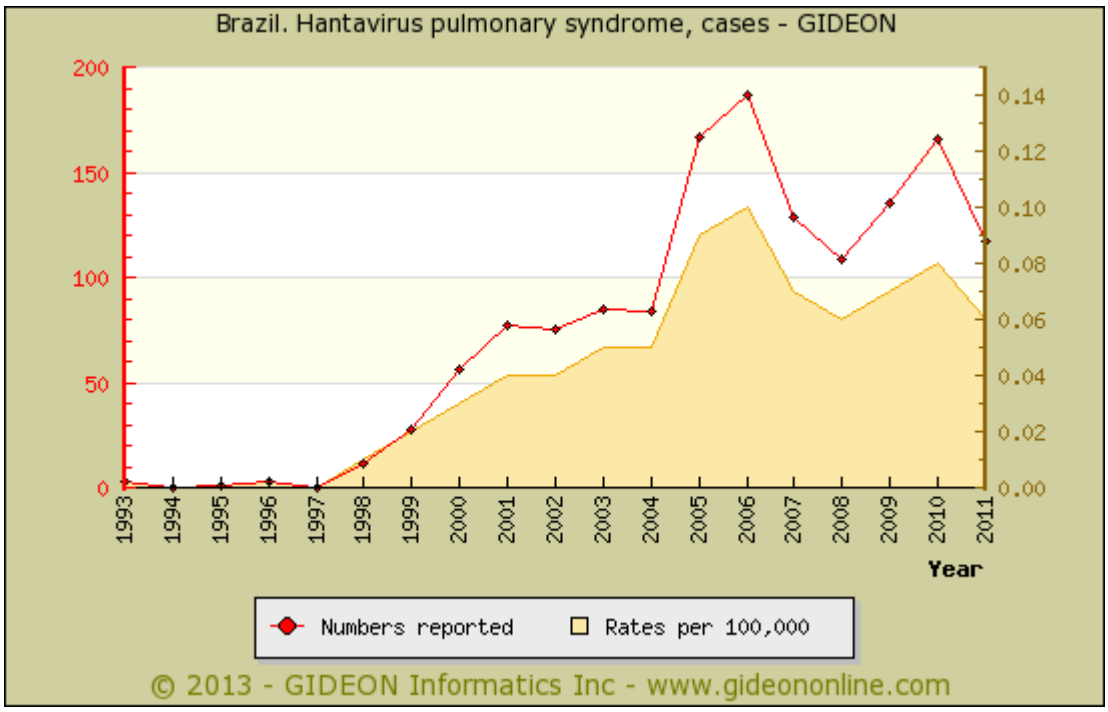
- 1,246 cases were reported in Brazil during 1993 to 2009. [19]
- 338 cases were reported to December 2003 - including 62 in Sao Paulo, 91 in Parana, 54 in Minas Gerais and 33 in Rio Grande do Sul
- 353 cases (161 fatal) were reported to June 2004, including 92 in Parana, 60 in Minas Gerais and 51 in Santa Catarina.
- 855 cases (39.3% fatal) were reported during 1993 to 2006 - 33.5% during spring and 27.6% during winter, predominantly among young adult males in rural areas. [20]
- Extension of highways through the Amazon region may contribute to the spread of viral strains in Brazil. [21]



Graph: Brazil. Hantavirus pulmonary syndrome, cases

Notes:
Individual years:
1993 - In Sao Paulo
1995 - In Para
1996 - In Sao Paulo
1998 - Included two cases (fatal) in the City of Guariba (northeastern Sao Paulo).
2008 - 34 cases (17 fatal) were reported during January to June. [22] 10 cases (3 fatal) were reported in Parana. [23]

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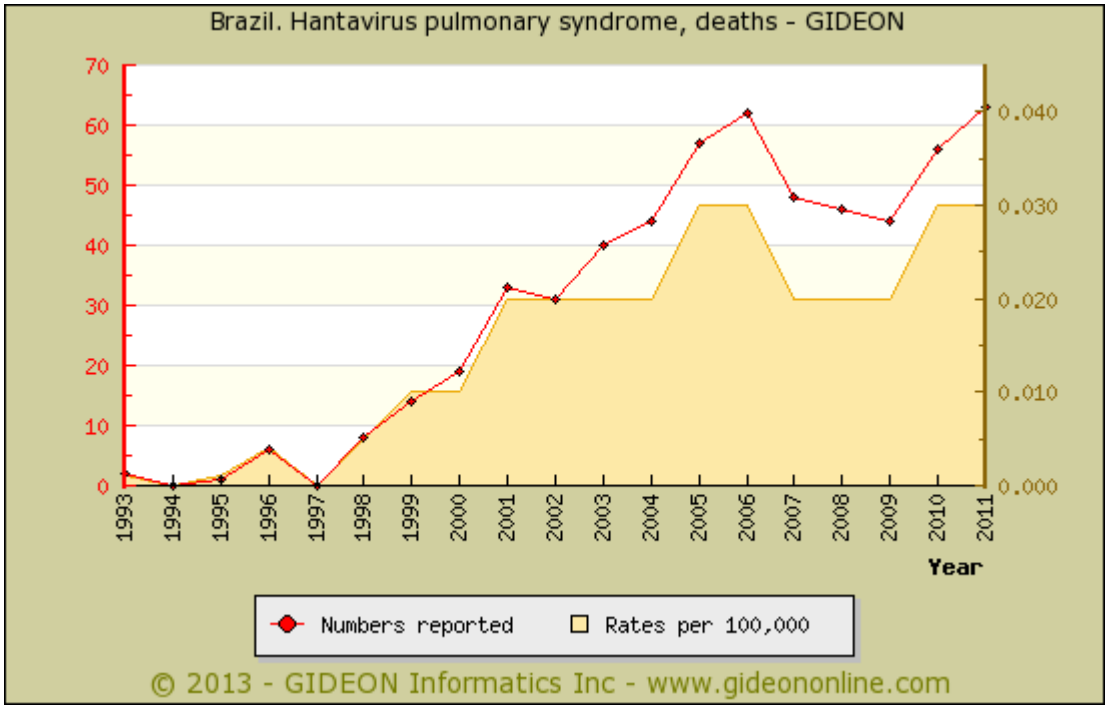

Graph: Brazil. Hantavirus pulmonary syndrome, deaths

Notes:

1. Fatality rates as high as 54.3% are reported in Ribeirao Preto (1998 to 2007) [24]

Geographical notes:

- Belo Horizonte [25] , Amazonas and Goias reported their first cases in 2004.
- Federal District (Brasilia) reported 33 cases (11 fatal) in 2004 - one half of these in Sao Sebastiao; 7 (2 fatal) in 2007. [26] [27]
- Maranhao State reported the first outbreak in the Brazilian Amazon, in 2000. [28] [29]
  7 cases (4 fatal) were reported in Maranhao during 2000 to 2003.
  The next fatal case in Maranhao was reported in 2006. [30]
- Mato Grosso State reported 49 cases (20 fatal) in 2006; 25 (9 fatal) in 2007. [31]
- Minas Gerais State reported seven fatal cases in 2002; 3 cases (2 fatal) during January to June 2008 [32] ; 14 (7 fatal) in 2010. [33]
- Parana State reported its first cases of Hantavirus infection in 1999 (7 cases, 5 fatal) - 4 in General Carneiro, 2 in Cruz Machado and one in Honorio Serpa.
  Twelve cases (2 fatal) were confirmed in Parana State in 2000 - from General Carneiro and Campina Grande do Sul, with additional suspect cases reported in Guarapava, Paulo Frontin and Pinhao.
  Parana reported 187 cases (67 fatal) during 1992 to 2009 [34] ; 93 during 1998 to 2004 (one third of all Brazilian cases). [35]
- Ribeirao Preto, Sao Paulo State reported 70 cases during 1998 to 2007. [36]
- Santa Catarina State reported 38 cases (6 fatal) during 2000 to 2003.

Prevalence surveys:

0% of dengue-like illness (Federal District, 2008) [37]
4.5% of pygmy rice rats (*Oligoryzomys utiaritensis*) in Campo Novo do Parecis, Mato Grosso State (Castelo dos Sonhos virus, 2005 to 2007) [38]
18% of rodents, opossums and bats in the rain forest of Sao Paulo (Araraquara virus, 2012 publication) [39]

Seroprevalence surveys:

1.23% of hospital inpatients in endemic areas (1999)
0.6% of persons in Amazonas (2012 publication) [40]
14.3% of adults in Jardinopolis County, Sao Paulo (2001) [41]
3% of persons in Uberlandia, Minas Gerais (2006) [42]
4.7% of persons in Maranhao (2004 to 2006) [43]
10.7% of persons in Maranhao (Anajatuba virus, 2005) [44]

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13% of persons in rural Marcelandia, Mato Grosso State (2012 publication) [45]
3.5% of persons in western Santa Catarina State (2011 publication) [46]
1.97% of persons in western Santa Catarina State (2012 publication) [47]
4.7% of persons in Cassia dos Coqueiros County, Sao Paulo (1987) [48]
2.3% of persons in rural Turvo Country, Santa Catarina State (2012 publication) [49]
2.4% of humans and 2.5% of rodents in southern Brazil (2006 to 2011) [50]
2.5% of wild rodents in Ribeirao Preto region - 4% of *Necromys lasiurus*, 1.9% of *Calomys tener* and 1.5% of *Akodon* sp (2005 to 2008) [51]

Infecting species:
- Juquitiba virus [52] , Araraquara virus [53-55] , Laguna Negra virus and Castello dos Sonhos virus [56-59] have been implicated in human infections in Brazil [60]
- Jabora strain Hantavirus was identified in *Akodon serrensis* and *Akodon montensis*; and Araucaria (Juquitaba-like) strain was detected in *Oligoryzomys nigripes*, *Oxymycterus judex*, *A. montensis*, and *Akodon paranaensis* (southern Brazil, 2012 publication) [61]
- Anajatuba (ANAJ) virus (reservoirs *O. fornesi* and *Necromys lasiurus*) [62] and Rio Mearim virus (reservoir *H. sciureus*) have also been identified. [63]
- Jabora virus has been found in rodents (*Akodon montensis* and *Akodon paranaensis*) in Santa Catarina State [64] [65]
- Paranoa (Araraquara-like) virus has been implicated in an outbreak in central Brazil. [66]
Araraquara virus has been identified in marsupials and bats in the rain forest of Sao Paulo (2012 publication) [67]
- Laguna Negra and Castelo dos Sonhos viruses have been reported from Mato Grosso (2009 publication) [68-70]
- Laguna Negra virus has been identified in humans and rodents (*Calomys callidus*) in Mato Grosso. [71]

Reservoirs:
- The presumed reservoir of Juquitiba-like virus is the black-footed pigmy mouse (*Oligoryzomys nigripes*). [72]
- The hairy-tailed Bolo mouse (*Bolomys lasiurus*) has been implicated in transmission of Araraqquara virus in Goias and Sao Paulo States (Seropositive *Oligoryzomys* have also been identified in Sao Paulo State). [73] [74]
- There is evidence for rodent seropositivity in Rio de Janeiro (2008 publication) [75]
- The pygmy rice rat (*Oligoryzomys utiaritensis*) [76] and *Oligoryzomys moojeni* have been identified as the host of Castelo dos Sonhos virus in Mato Grosso. [77]
- Hantaviruses have been identified in *Akodon montensis*, *Akodon cursor*, *Necromys lasiurus*, *Juliomys* sp., *Thaptomys nigrita*, *Oligoryzomys nigripes*, and *Oryzomys* sp. (Sao Paulo, 2011 publication). [78]
- Jabora strain Hantavirus was identified in *Akodon serrensis* and *Akodon montensis*; and Araucaria (Juquitaba-like) strain was detected in *Oligoryzomys nigripes*, *Oxymycterus judex*, *A. montensis*, and *Akodon paranaensis* (southern Brazil, 2012 publication) [79]
- Araraquara virus has been identified in marsupials (*Micoureus paraguayanus*, *Monodelphis ihering*, and *Didelphis aurita*) and bats (*Diphylla ecaudata* and *Anoura caudifer*) in the rain forest of Sao Paulo (2012 publication) [80]

Notable outbreaks:
2004 - An outbreak (164 cases, including 30 in Federal District) of Hantavirus pulmonary syndrome was reported in central Brazil. [81]

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Hepatitis A

Agent	VIRUS - RNA. Picornaviridae, Hepatovirus: Hepatitis A virus
Reservoir	Human Non-human primate
Vector	None
Vehicle	Fecal-oral Food Water Fly
Incubation Period	21d - 30d (range 14d - 60d)
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions; supportive
Typical Pediatric Therapy	As for adult
Vaccines	Hepatitis A Hepatitis A + Hepatitis B Immune globulin
Clinical Hints	Vomiting, anorexia, dark urine, light stools and jaundice; rash and arthritis occasionally encountered; fulminant disease, encephalopathy and fatal infections are rare (case-fatality rate 0.15% to 2.7%, depending on age).
Synonyms	Botkin's disease, Epatite A, HAV, Hepatite per virus A, Infectious hepatitis. ICD9: 070.0 ICD10: B15.0, B15.9

Clinical

WHO Case definition for surveillance of acute viral hepatitis (all types):
Clinical description
• Acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue, and right upper quadrant tenderness.
• Biological signs include increased urine urobilinogen and >2.5 times the upper limit of serum alanine aminotransferase.
• Note: Most infections occur in early childhood. A variable proportion of adult infections is asymptomatic.
Laboratory criteria for diagnosis
• Hepatitis A: IgM anti-HAV positive
• Hepatitis B: positive for Hepatitis B surface antigen (HBsAg) or IgM anti-HBC-positive
• Non-A, non-B: IgM anti-HAV and IgM anti-HBc (or HBsAg) negative
Note 1: The anti-HBc IgM test, specific for acute infection, is not available in most countries.
• HBsAg, often available, cannot distinguish between acute new infections and exacerbations of chronic hepatitis B, although continued HBsAg seropositivity (>6 months) is an indicator of chronic infection.
Note 2: For patients negative for hepatitis A or B, further testing for a diagnosis of acute hepatitis C, D, or E is recommended:
Hepatitis C: anti-HCV positive
Hepatitis D: HBsAg positive or IgM anti-HBc positive plus anti-HDV positive (only as co-infection or super-infection of hepatitis B)
Hepatitis E: IgM anti-HEV positive
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory confirmed or, for hepatitis A only, a case compatible with the clinical description, in a person who has an epidemiological link with a laboratory-confirmed case of hepatitis A (i.e. household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

Clinical features of Hepatitis A:

The prodrome is characterized by anorexia, asthenia, headache, myalgia and moderate fever.
• Patients develop nausea, vomiting and right upper abdominal pain • and later overt jaundice. [1]
• Symptoms persist for 4 to 8 weeks, and the patient may remain asthenic and anorectic for several months thereafter.
• As many as 90% of cases in children less than 5 years of age are asymptomatic; fewer 50% among adults.
• Relapses may occur for up to 6 months following the initial infection.
• Rare instances of acute disseminated encephalomyelitis [2] , myelitis [3] , meningoencephalitis [4] , acute cholestatic syndrome [5] , acalculous cholecystitis [6] [7] , urticaria [8] , pancreatitis [9] [10] , pleural effusion or ascites [11] [12] , acute

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glomerulonephritis or renal failure [13-22] , pure red-cell aplasia [23] , cerebral venous thrombosis [24] and rhabdomyolysis have been reported. [25]
• Concurrent HIV infection may prolong the duration of viremia in patients with hepatitis A. [26]

Hepatitis A accounts for 3.1% of acute hepatic failure cases (United States, 1998 to 2005) [27]
• The case-fatality rate is 0.1% among children below age 4 years; 0.4% ages 5 to 29 years; and 1% above age 40.
• 55% of hepatitis A patients with acute hepatic failure recover • the remainder either die of the disease or require transplantation. [28]

A false positive serological reaction toward Epstein-Barr virus has been associated with Hepatitis A. [29]

This disease is endemic or potentially endemic to all countries.

Hepatitis A in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Cost-effectiveness analysis of universal childhood hepatitis A vaccination in Brazil - see reference [30]

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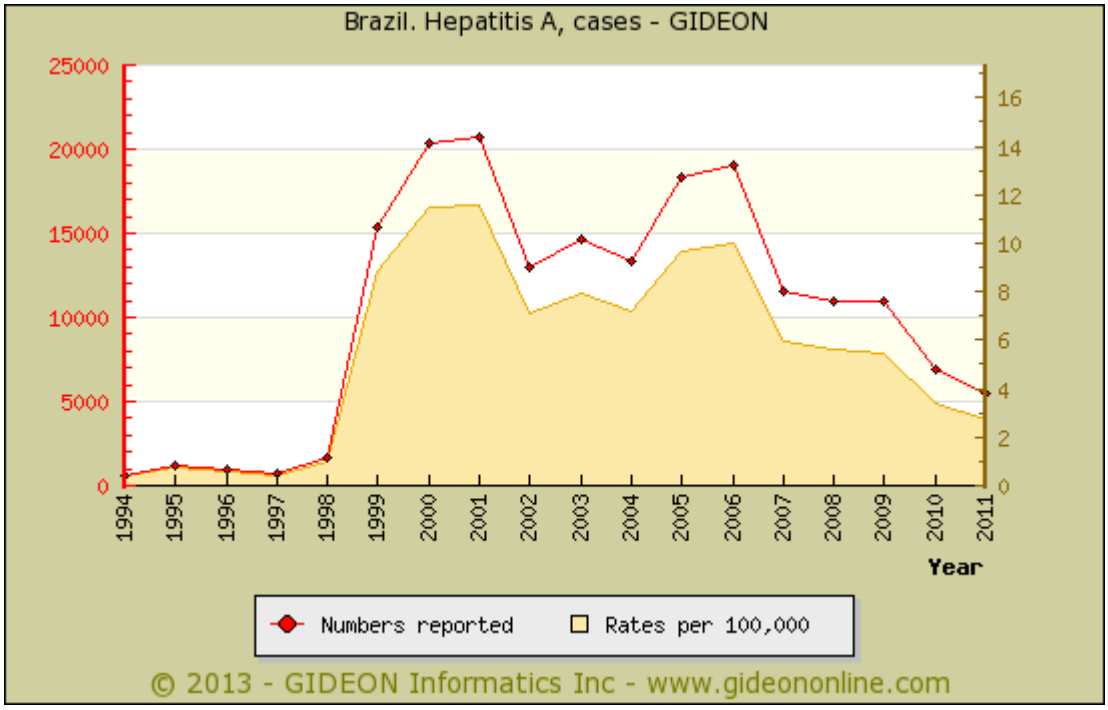

Graph: Brazil. Hepatitis A, cases

Notes:
Individual years:
2004 - 51 fatal

Prevalence surveys:

43% of viral hepatitis in Santa Catarina (1996 to 2002)
16.7% of sludge and 66.6% of wastewater samples in Floranopolis City (2010 publication) [31]
92% of environmental water samples in the Amazon basin (2007 publication) [32]
8.3% of water samples and 0% of oyster samples in Florianopolis (2007 to 2008) [33]
12% of surface water samples in Florianopolis, Santa Catarina (2012 publication) [34]
51.5% of seawater samples along beaches in Florianopolis, Santa Catarina (2009 to 2010) [35]
58% of raw sewage samples in Rio de Janeiro (2009 to 2010) [36]

Seroprevalence surveys:

64.7% of the general population, with highest rates in the north (92.8% seropositivity) (1996 to 1997) [37]
37.6% of persons ages 1 to 19 years tested in a public laboratory, and 46.1% of those tested in a private laboratory (2007 to 2009) [38]
87.9% in an area of Rio de Janeiro which had undergone an outbreak (2008 publication) [39]
98% of Amerindians (2004 publication) [40]
74.6% of persons in isolated communities in the western Amazon region (2009 publication) [41]
56% in the South and Southeast to 93% in the North (2005 publication) [42]
83.3% in the semiarid Northeast (2006 publication) [43]
75.6% of isolated Afro-Brazilian communities in the central region (2002 to 2003) [44]
80.9% of the Kalunga (Afro-Brazilian) community in Goiania (2004) [45]
97.7% of Indians from Xingu National Park (2007 publication) [46]
73.7% of health-care workers and 85.7% of blood donors in Rio de Janeiro (1999)
85.9% of persons in Cavunge, Bahia State (2006 publication) [47]
65.5% of children and adolescents in Rio de Janeiro in 1978, and 32.1% in 1995
9.72% of children and teenagers in Santos, Sao Paulo (2012 publication) [48]
46% of children ages 10 to 14 and 64% ages 15 to 19 (Sao Paulo, 2006 publication) [49]
71.5% of children ages 7 to 14 in public schools and 36.5% in private schools (Sao Luis, Maranhao, 2002 to 2004) [50]
41.5% of children ages 5 to 9 and 57.4% ages 10 to 19 in the Northeast; 32.3% and 56.0%, respectively for the Midwest; and 33.8% and 65.1% for the Federal District (2004 to 2005) [51]
19.8% of children and adolescents ages 1 to 14 years in Curitiba, Parana (2006) [52]
17.5% of urban children (2011 publication) [53]

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79.8% of HIV-positive patients (1988 to 2004) [54]
98.1% of adults patients with hepatic cirrhosis (Uberlandia, 2005 to 2006) [55]

Notable outbreaks:

1982 (publication year) - An outbreak of water-borne hepatitis A was reported in Rio de Janeiro. [56]
1999 - An outbreak (25 cases) was reported among students at a municipal school in Rio de Janeiro. [57] [58]
1999 to 2001 - An outbreak was reported in the area of Rio de Janeiro. [59]
2006 - An outbreak in Vitoria, Espirito Santo was related to contaminated water. [60]
2009 - An outbreak in Luziania, Goias State was associated with ingestion of contaminated drinking water. [61]
2011 (publication year) - An outbreak was reported at a daycare center in Rio de Janeiro. [62]

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Hepatitis B

Agent	VIRUS - DNA. Hepadnaviridae, Orthohepadnavirus: Hepatitis B virus
Reservoir	Human Non-human primate
Vector	None
Vehicle	Blood Infected secretions Sexual contact Transplacental
Incubation Period	2m - 3m (range 1m - 13m)
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Needle precautions; supportive. For post-exposure or chronic infection: Peginterferon alfa-2a or Peginterferon alfa-2b ; OR Lamivudine; OR Adefovir
Typical Pediatric Therapy	As for adult
Vaccines	Hepatitis A + Hepatitis B Hepatitis B + Haemoph. influenzae Hepatitis B immune globulin Hepatitis B
Clinical Hints	Vomiting and jaundice; rash or arthritis occasionally noted; risk group (drug abuse, blood products, sexual transmission); cirrhosis or hepatoma may follow years after acute illness; fulminant and fatal infections are encountered.
Synonyms	Epatite B, HBV, Hepatite per virus B, Serum hepatitis. ICD9: 070.1 ICD10: B16.2,B16.9, B16.1

Clinical

WHO Case definition for surveillance of acute viral hepatitis (all types):
Clinical description
• Acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue, and right upper quadrant tenderness.
• Biological signs include increased urine urobilinogen and >2.5 times the upper limit of serum alanine aminotransferase.
• Note: Most infections occur in early childhood. A variable proportion of adult infections is asymptomatic.
Laboratory criteria for diagnosis
• Hepatitis A: IgM anti-HAV positive
• Hepatitis B: positive for Hepatitis B surface antigen (HBsAg) or IgM anti-HBc positive
• Non-A, non-B: IgM anti-HAV and IgM anti-HBc (or HBsAg) negative
Note 1: The anti-HBc IgM test, specific for acute infection, is not available in most countries.
• HBsAg, often available, cannot distinguish between acute new infections and exacerbations of chronic hepatitis B, although continued HBsAg seropositivity (>6 months) is an indicator of chronic infection.
Note 2: For patients negative for hepatitis A or B, further testing for a diagnosis of acute hepatitis C, D, or E is recommended:
Hepatitis C: anti-HCV positive
Hepatitis D: HBsAg positive or IgM anti-HBc positive plus anti-HDV positive (only as co-infection or super-infection of hepatitis B)
Hepatitis E: IgM anti-HEV positive
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory confirmed or, for hepatitis A only, a case compatible with the clinical description, in a person who has an epidemiological link with a laboratory-confirmed case of hepatitis A (i.e. household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

Clinical features of Hepatitis B:

Infection can be asymptomatic (particularly in young children) or quite mild, with only fatigue, anorexia, and malaise.
• Clinical disease with jaundice occurs in 50% of adults and 10% of young children.
• Extrahepatic manifestations include arthralgia, arthritis, rash [1] ,inflammatory myopathy [2] , focal segmental glomerulosclerosis [3] and acute glomerulonephritis. [4-8]
• Rare instances of pure red cell aplasia [9] , symmetric sensorimotor polyneuropathy [10] , and pancreatitis have been

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reported. [11]
• Chronic infection occurs in most young children and in 5% to 10% of adults, and can lead to persistent hepatitis, retarded growth in children [12] , active hepatitis, cirrhosis, or hepatocellular carcinoma. [13]
• Acute exacerbation of chronic Hepatitis B may occur. [14]
• Patients with HBV-HDV coinfection appear to have more severe acute disease and a higher risk of fulminant hepatitis (2% to 20%) compared with those infected with HBV alone [15] ; however, chronic HBV infection appears to occur less frequently in persons with HBV-HDV coinfection.
• Concurrent HIV infection increases the incidence of cirrhosis and HCC among Hepatitis B carriers. [16] [17]

One to two million deaths are attributed to hepatitis B annually. 25% of chronic carriers died of primary liver cancer or cirrhosis as adults.
• Hepatitis B is responsible for 60% to 80% of the world's primary liver cancer.
• Primary liver cancer is one of the three leading causes of cancer death in East Asia, Southeast Asia, the Pacific Basin and sub-Saharan Africa.
• Hepatitis B predominates among patients with hepatocellular carcinoma in most Asian, African and Latin American countries; while hepatitis C predominates in Japan, Pakistan, Mongolia, Egypt, Europe and the United States. [18]

This disease is endemic or potentially endemic to all countries.

Hepatitis B in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Hepatitis B vaccination was initiated for 396,000 persons in 1997.

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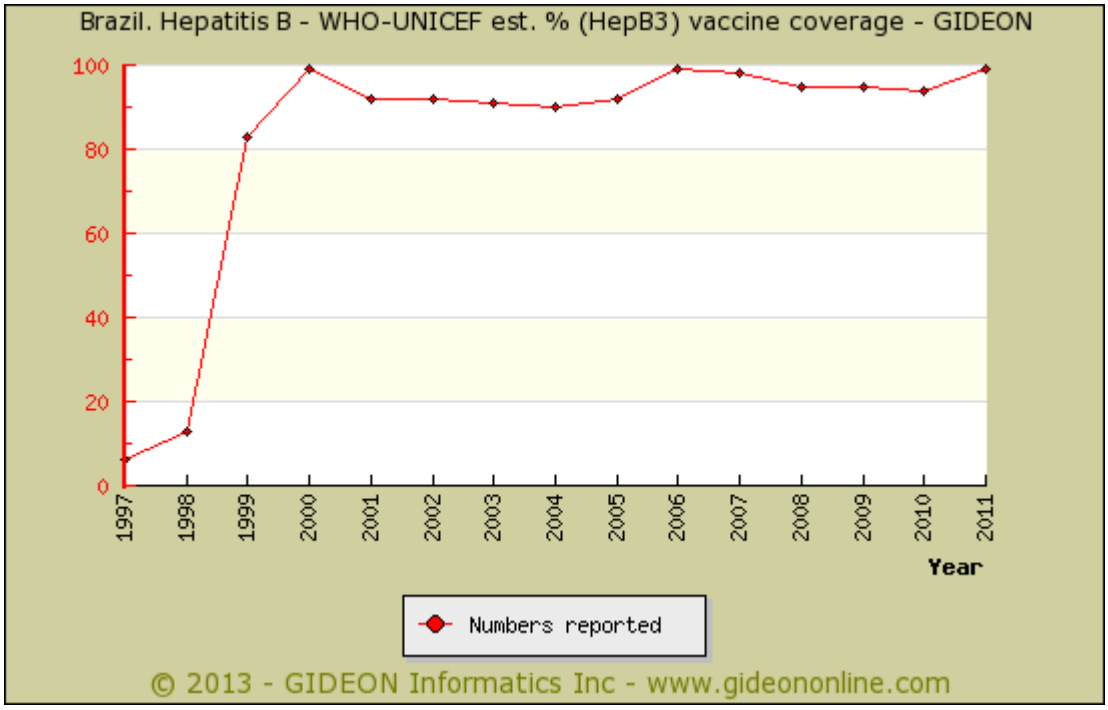
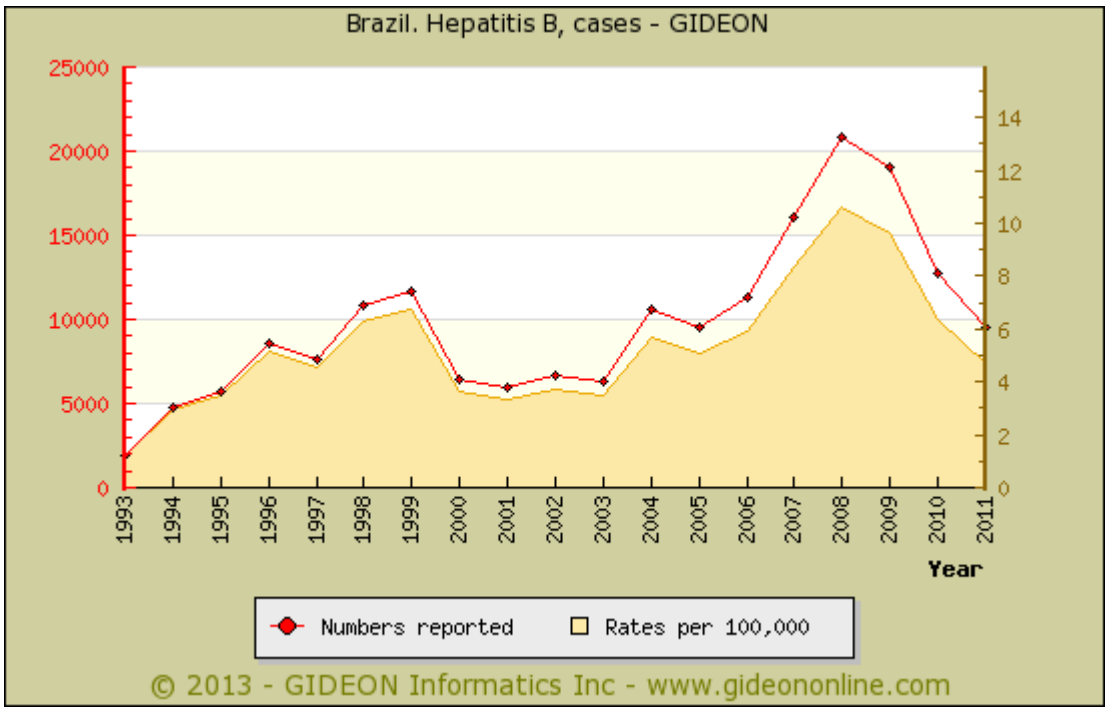


Graph: Brazil. Hepatitis B - WHO-UNICEF est. % (HepB3) vaccine coverage



Graph: Brazil. Hepatitis B, cases

Notes:
1. Hepatitis B accounts for 25% of viral hepatitis in Santa Catarina (1996 to 2002).
2. Infection is most common in the Amazon region, with carriage rates as high as 12% in some areas.

Individual years:
1998 - Included 2,726 from Parana and 1,648 from Rio Grande do Sul.
1999 - Included 2,440 from Parana and 1,149 from Rio de Janeiro.
2004 - 223 fatal

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Hepatitis B accounts for 16% of hepatocellular carcinoma cases in Brazil. (2004 to 2009) [19]
- Hepatitis B was implicated in <=7% of fatal cases of hepatocellular carcinoma. [20]
- Hepatitis B was implicated in 28.8% of non-alcoholic fatty liver disease (Western Amazon, 2010 to 2011) [21]
- Serological markers for hepatitis B are found in 50.7% of patients with clinical hepatitis (Goias).
- Serological markers for hepatitis B are found in 18.5% of pregnant women in Parana [22]
- Anti-HBc and/or HBsAg were found in 30% of patients with hepatosplenic schistosomiasis (Recife, 2008) [23]

HBsAg-positivity surveys:

0.8% of Blacks and 0% of Caucasians (1970 publication) [24]
0.1% to 4% in the southeast, 1.0% in the south, 1.6% of Rio (blood donors), 2.5% in the northeast and 8.0% in Amazonia
2.6% in the semiarid Northeast (2006 publication) [25]
3.6% in Para (2002 to 2005) [26]
8.4% in an Afro-Brazilian community in Mato Grosso do Sul (2008 publication) [27]
2.5% of persons in Frechal Quilombo community at Maranhao (2011 publication) [28]
2.88% in Buriticupu, Amazon region (2010 publication) [29]
7.5% of men and 4.6% of women in Alto Solimoes, Amazon border region (2012 publication) [30]
1.9% of alcoholics (8.0% of cirrhotic alcoholics)
7.8% of IDU in Rio de Janeiro during 1994 to 1996 [31] , 3.4% during 1999 to 2001 [32]
1.1% of non-injecting drug users in central-western Brazil (2009 publication) [33]
3.3% of homeless persons in Sao Paulo (2002 to 2003) [34]
0.8% of female crack cocaine users in Bahia, Salvador (2007 publication) [35]
29.8% of hemodialysis patients in Goias (2002) [36]
15.4% of hemodialysis patients in Sao Paulo (2010 publication) [37]
3.1% of sickle cell anemia patients in Bahia (1995 to 2009) [38]
0% of adults with psoriasis (Salvador, Bahia, 2012 publication) [39]
4.6% of HIV-positive patients (2006 publication) [40]
36.9% of HIV-positive patients in Goiania City, Goias (2008 to 2010) [41]
6.4% of HIV-positive patients in the Amazon region (2006 publication) [42]
3.8% of HIV-positive patients in Vitoria (1993 to 2004) [43]
5.9% of HIV-positive patients in Sao Paulo (2009 publication) [44]
3.2% of HIV-positive women in Salvador, Bahia (2012 publication) [45]
2.3% of pregnant HIV-positive women (Rio Grande do Sul, 2012 publication) [46]
51.3% of HTLV-1/2-positive blood donors in Ribeirao Preto City (2000 to 2010) [47]
14.7% of patients with mental illness (2009 publication) [48]
3.3% in the Western Brazilian Amazon (2005 publication) [49]
7.1% of Amazon gold miners
9.7% of Amazon Indians
0% of Brazilian Amazon riparians (2011 publication) [50]
0% to 0.5% of Kaingang Indians in Parana (2006 publication) [51]
1.1% to 5.4% of Parakana Indians in Para (2004) [52]
1.9% of persons in riverside communities of the Tucurui Dam, Para (2012 publication) [53]
2.2% in isolated Afro-Brazilian communities (2005 publication) [54]
6.2% of persons in rural Labrea, Brazilian Amazon (2012 publication) [55]
10.2% of persons in the western Brazilian Amazon (2008) [56]
1.8% of the Kalunga (Afro-Brazilian) community in Goiania (2004) [57]
1.2% of low-income postpartum and 1% of pregnant women treated in Greater Metropolitan Vitoria, Espirito Santo State (1999) [58]
0.9% of young women in Espirito Santo (2006) [59]
0.8% of pregnant women in Parana (1996 to 1998)
0.7% of pregnant women in the western Amazon (2008) [60]
0.5% of pregnant women in Parana (2010) [61]
0.9% of pregnant women in Sao Luis, Maranhao (2012 publication) [62]
1.8% of pregnant women in Sao Paulo (2011 publication) [63]
0.46% of pregnant women in Itajai, Santa Catarina State (2002 to 2007) [64]
1.5% of women in rural Alagoas (1997)
0.9% of young women in Vitoria, Espiritu Santo (2006) [65] [66]
0.7% of laboratory workers in Goiania, Goias (2005 publication) [67]
0.8% of health care workers (1994 to 1999) [68]

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1.2% of school children in Peixoto de Azevedo (Brazilian Amazon, 1998)
0.76% of children and adolescents in the southern region (2011 publication) [69]
0.7% of teenagers in Araguaia region, Central Brazil (2011 publication) [70]
0.2% of adolescents in Chapeco, Santa Catarina State (2011 publication) [71]
0% of adolescents ages 10 to 16 years in metropolitan Florianopolis, Santa Catarina (2010 publication) [72]
0.6% of adolescents from low income families in Central Brazil (2006 publication) [73]
0.6% of adolescents in Itajai, Santa Catarina (2008) [74]
6.9% of first-time blood donors in southwestern Goias (2011 publication) [75]
0.66% of blood donors during 2000 to 2001; 0.50 in 2002
0.289% of blood donors (2007) [76]
0.6% of blood donors in Ribeirao Preto (1996 to 2001) [77]
0.36% of blood donors in 1998; 0.14% in 2005 (Rio de Janeiro) [78]
0.18% of first time male and 0.68% of female blood donors (Sao Paulo, Salvador and Manaus, 2007 publication) [79]
1.3% of potential solid organ donors (Santa Catarina, 2006 to 2007) [80]
1.5% of eye tissue donors (Parana, 2006 to 2007) [81]
1% of leprosy outpatients (central Brazil, 2011 publication) [82]
2.2% in Afro-Brazilian communities (2005 publication) [83]
2.6% of army conscripts (2002) [84]
1% of firefighters in Campo Grande, Mato Grosso do Sol (2012 publication) [85]
14.2% of long distance truck drivers (2008 publication) [86]
18.9% of truck drivers (2008 publication) [87]
0.6% of dentists in Campo Grande, Mato Grosso do Sul (2006 publication) [88]
2.4% of incarcerated adolescents (Salvador, 2008 publication) [89]
0.5% of prison inmates in Campo Grande, Mato Grosso do Sul (2010 publication) [90]
58% of breast milk samples from HBsAg-positive mothers (2009 publication) [91]

Anti-HBc antibody is present in 7.9% of the population (1996 to 1997), with highest rates above age 16 (suggestive of sexual transmission).

Notable outbreaks:

1995 to 1997 - An outbreak of hepatitis B involved two hemodialysis units, in Sao Paulo and Rio de Janeiro. [92]

1998 (publication year) - An outbreak of Hepatitis B was reported among persons who had recently arrived to the Amazon. [93] [94]

2001 (publication year) - An outbreak (26 cases) was reported in a hemodialysis unit. [95]

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Hepatitis C

Agent	VIRUS - RNA. Flaviviridae, Hepacivirus: Hepatitis C virus
Reservoir	Human
Vector	None
Vehicle	Blood Sexual contact Transplacental
Incubation Period	5w - 10w (range 3w - 16w)
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Needle precautions; supportive. If hepatocellular disease: Weekly Peginterferon alfa-2a 180 mcg SC or Peginterferon alfa-2b 1.5 mcg SC AND Ribavirin 400 mg in AM & 600 mg in PM daily AND Telepravir OR Bocepravir Duration per viral genotype
Typical Pediatric Therapy	Peginterferon alfa-2b 3 MU/m2 SC x1 weekly AND Ribavirin 15mg/kg
Clinical Hints	Vomiting and jaundice; may be history of transfusion within preceding 1 to 4 months; chronic hepatitis and fulminant infections are encountered.
Synonyms	Epatite C, HCV, Hepatite per virus C, Non-A, non-B parenteral hepatitis. ICD9: 070.2,070.3,070.44,070.51,070.54,070.7 ICD10: B17.1

Clinical

WHO Case definition for surveillance of acute viral hepatitis (all types):
Clinical description
• Acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue, and right upper quadrant tenderness.
• Biological signs include increased urine urobilinogen and >2.5 times the upper limit of serum alanine aminotransferase.
• Note: Most infections occur in early childhood. A variable proportion of adult infections is asymptomatic.
Laboratory criteria for diagnosis
• Hepatitis A: IgM anti-HAV positive
• Hepatitis B: positive for Hepatitis B surface antigen (HBsAg) or IgM anti-HBc positive
• Non-A, non-B: IgM anti-HAV and IgM anti-HBc (or HBsAg) negative
Note 1: The anti-HBc IgM test, specific for acute infection, is not available in most countries.
• HBsAg, often available, cannot distinguish between acute new infections and exacerbations of chronic hepatitis B, although continued HBsAg seropositivity (>6 months) is an indicator of chronic infection.
Note 2: For patients negative for hepatitis A or B, further testing for a diagnosis of acute hepatitis C, D, or E is recommended:
Hepatitis C: anti-HCV positive [1]
Hepatitis D: HBsAg positive or IgM anti-HBc positive plus anti-HDV positive (only as co-infection or super-infection of hepatitis B)
Hepatitis E: IgM anti-HEV positive
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory confirmed or, for hepatitis A only, a case compatible with the clinical description, in a person who has an epidemiological link with a laboratory-confirmed case of hepatitis A (i.e. household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

Clinical features of Hepatitis C:

Patients with acute infection typically are either asymptomatic or have a mild clinical illness. [2]
• 60% to 70% of patients have no symptoms
• 20% to 30% of patients have jaundice
• 10% to 20% of patients have non-specific symptoms, such as anorexia, malaise, or abdominal pain.

Clinical illness in patients with acute hepatitis C who seek medical care is similar to that of other types of viral hepatitis.
• The average time period from exposure to symptom onset is 6-7 weeks, whereas the average time period from exposure to seroconversion is 8-9 weeks.
• Anti-HCV can be detected in 80% of patients within 15 weeks after exposure, in >90% within 5 months after exposure, and in >97% by 6 months after exposure.

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- Rarely, seroconversion is delayed for as long as 9 months after exposure.
- Rare instances of optic neuritis have been reported. [3]

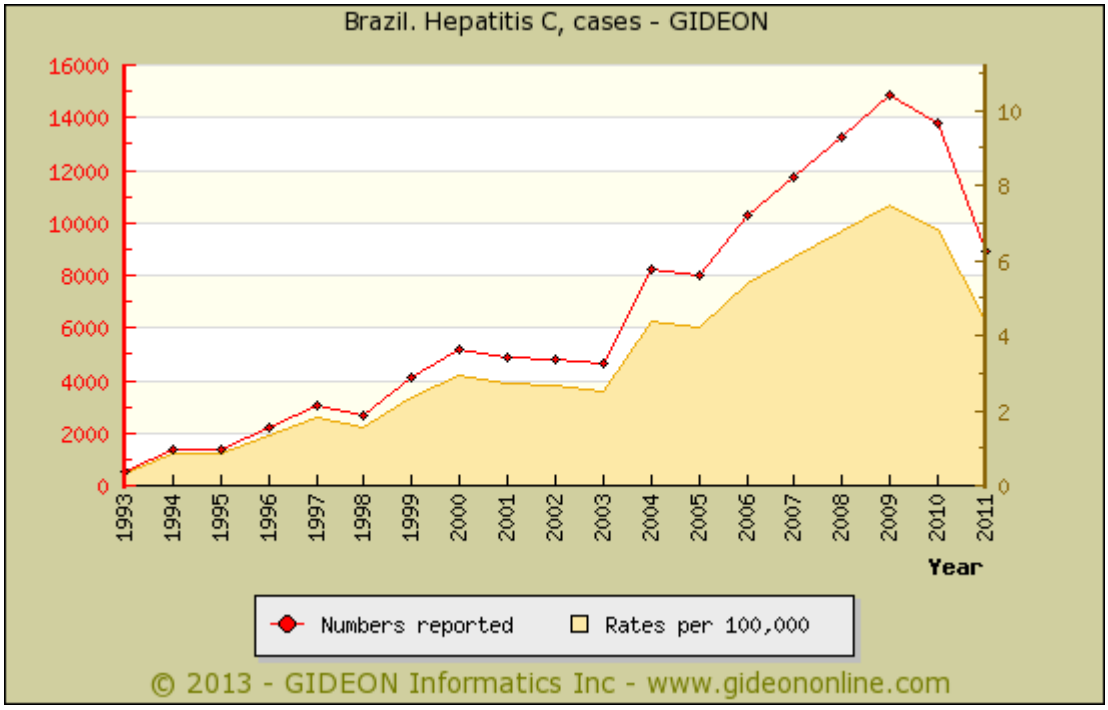
The clinical course is variable; and fluctuating elevations in serum ALT levels, are the most characteristic feature. [4] [5]
- Fulminant hepatic failure following acute infection is rare.
- 15% to 25% of infections resolve without sequelae.
- Chronic HCV infection develops 75% to 85% of patients who exhibit persistent or fluctuating ALT elevations.
- 75% to 85% of patients with acute hepatitis C infection progress to chronic disease, and 20% to cirrhosis within 20 to 25 years. [6]
- No clinical or epidemiological features among patients with acute infection are predictive of persistent infection or chronic liver disease.
- Chronic liver disease is usually insidious, progressing without symptoms or physical signs in the majority of patients during 20 or more years following acute infection.
- Cirrhosis develops in 10% to 20% of persons with chronic hepatitis C over a period of 20 to 30 years; and hepatic cell carcinoma in 1% to 5%.
- HCV infection appears to have little short-term impact on survival after bone marrow transplantation, but is a risk factor for veno-occlusive disease and graft-versus-host disease. [7]
- Concurrent HIV infection shortens the time to development of chronic liver disease in patients with Hepatitis C. [8-13]

Hepatitis B predominates among patients with hepatocellular carcinoma in most Asian, African and Latin American countries; while hepatitis C predominates in Japan, Pakistan, Mongolia, Egypt, Europe and the United States. [14]

Additional manifestations seen in patients with chronic hepatitis C infection [15] may include mixed cryoglobulinemia with systemic vasculitis of the skin, erythema induratum [16] , retarded growth in children [17] , renal disease [18-23] ; CNS vasculitis [24] , acute disseminated encephalomyelitis [25] , dorsal root ganglionopathy [26] and other nervous system disorders [27-30] ; thrombocytopenia [31-33] ; non-Hodgkin lymphoma; porphyria cutanea tarda and lichen planus [34] ; hypothyroidism [35] ; lymphocytic sialoadenitis (similar to that of Sjogren's syndrome) [36] ; autoimmune and other rheumatological disorders [37-40] , nectolytic acral erythema [41] ; scleritis [42] ; and orbital plasmacytoma. [43]

This disease is endemic or potentially endemic to all countries.

Hepatitis C in Brazil



Graph: Brazil. Hepatitis C, cases

Notes:
1. 18,686 cases were reported during 1993 to 2000 - including 4,827 in Rio de Janeiro and 4,803 in Rio Grande do Sul.

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Individual years:
2004 - 255 fatal.

Most cases of acute Hepatitis C in Brazil are related to hospital procedures (2000 to 2008) [44]

Prevalence surveys:

1% to 2% of the general population (2011 publication) [45]
2.60%, nationwide (estimated)
8.8% of persons in Bedebouro, Sao Paulo (2007) [46]
9.7% of persons in Rondonia State (1999 to 2005) [47]
5% of HIV-positive persons in the Amazon region (2006 publication) [48]
6.0% of HIV-positive patients (Mato Grosso State, 2007 publication) [49]
2.9% of patients with idiopathic dilated cardiomyopathy (2007 publication) [50]
7.2% of former athletes (Ribeirao Prete, 2008 publication) [51]
10.6% of hemodialysis patients in Juiz de For a, Minas Gerais (2007) [52]
20.9% of hemodialysis patients in Porto Alegre (2005 to 2006) [53]
54% of patients with hepatocellular carcinoma (2004 to 2009) [54]
71.2% of patients with non-alcoholic fatty liver disease (Western Amazon, 2010 to 2011) [55]

Seroprevalence surveys:

1.38%, nationwide (2005 to 2009) [56]
1.7% of persons in the Purus and Acre river regions
1.53% of persons in Criciuma-SC (2008 publication) [57]
3.2% of persons in Salvador in 1998 - 1.5% viremic (2006 publication) [58]
5.9% of persons in the Amazon (2003) [59]
6.16% of persons from two inland regions (2012 publication) [60]
5.76% of persons in Buriticupu, Amazon region (2010 publication) [61]
4.28% of persons in Tamboara, Parana State (2011 publication) [62]
0.3% of persons visiting a public health facility in Sao Jose dos Pinhais, Parana (2012 publication) [63]
0.4% of persons in the semiarid Northeast (2006 publication) [64]
0.2% of persons in Quilombo remnant communities in Mato Grosso do Sul (2008 publication) [65]
0.7% of men and 0.7% of women in Alto Solimoes, Amazon border region (2012 publication) [66]
0% of adolescents ages 10 to 16 years in metropolitan Florianopolis, Santa Catarina (2010 publication) [67]
0% of children and adolescents in the southern region (2011 publication) [68]
0% of adolescents in Chapeco, Santa Catarina State (2011 publication) [69]
0.6% of the Kalunga (Afro-Brazilian) community in Goiania (2004) [70]
8.8% of Brazilian Amazon riparians (2011 publication) [71]
3.7% of persons in Para (2002 to 2005) [72]
2.2% of persons in riverside communities of the Tucurui Dam, Para (2012 publication) [73]
6% of persons undergoing voluntary testing in Rio Grande (2010 publication) [74]
1.4% of long distance truck drivers (2010 publication) [75]
0.41% of blood donors in Rio Grande do Sul (2009 publication) [76]
0.34% of blood donors in Santa Catarina (2001)
0.52% of blood donors nationwide (2002)
0.191% of blood donors (2007) [77]
1.2% of blood donors in Ribeirao Preto (1996 to 2001) [78]
0.9% of blood donors in Apucarana (1997 to 1999) [79]
0.3% of blood donors in Uberaba, Minas Gerais (2009 publication) [80]
1.04% of blood donors in Rio de Janeiro in 1998; 0.79% in 2004 [81]
1.25% of blood donors in Manaus in 1995; 0.32% in 2007 [82]
0.46% of blood donors in Para (2004 to 2006) [83]
0.2% of pregnant women in Campo Grande, Mato Grosso do Sul (2002 to 2005) [84]
0.38% of first time male and 0.97% of female blood donors (Sao Paulo, Salvador and Manaus, 2007 publication) [85]
6.9% of street youth age 17 to 20 (Goiania City)
3.8% of alcoholics (6.0% among alcoholics with cirrhosis)
8.5% of homeless persons in Sao Paulo (2002 to 2003) [86]
2.63% of patients with mental illness (2009 publication) [87]
75% of IDU in 1994, and 20.6% in 2001 (cross-sectional studies) [88]

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69.6% of IDU in Rio de Janeiro
10.1% of short-term vs. 23.4% of long-term IDU in Rio de Janeiro (2009 publication) [89]
83% of IDU in Sao Paulo (2003) [90]
6.4% of IDU in Cuiaba, Mato Grosso (2009 publication) [91]
35.6% of IDU, 29.8% of ex-IDU and 5.3% non-IDU in Salvador (2010 publication) [92]
2.4% of female crack cocaine users in Bahia, Salvador (2007 publication) [93]
32.3% of cocaine users in Para (2013 publication) [94]
6.9% of drug users in Goiana and Campo Grande (2005 to 2006) [95]
1.1% to 2.4% of the general population in the Amazon region (2004 publication) [96]
0.5% of Kaingang Indians in Parana (2006 publication) [97]
2.1% of Amazon gold miners
0.6% of young women in Vitoria (2006) [98]
0.8% of pregnant women in Parana (1996 to 1998)
0.4% of pregnant women in Cuiaba, Mato Grosso (2001 to 2002) [99]
0.15% of pregnant women in Goiania City, central Brazil (2004 to 2005) [100]
0.7% of pregnant women in Sao Paulo (2011 publication) [101]
0.17% of pregnant women in Mato Grosso do Sul (2005 to 2007) [102]
1.8% of low-income postpartum and 0.6% of pregnant women treated in Greater Metropolitan Vitoria, Espirito Santo State (1999) [103]
1.4% of hospitalized patients with diabetes, and 1% of controls (Mato Grosso, 2005) [104]
44.6% of hemophiliacs (1997); 63.3% in central Brazil (1997)
42.2% of hemophiliacs in Bahia (2005 publication) [105]
37.7% of hemophiliacs in Maringa (2008 publication) [106]
13.4% of sickle cell anemia patients in Bahia (1995 to 2009) [107]
14.1% of patients with sickle cell anemia (Pernambuco, 1998) [108]
3.9% of pre-dialysis patients in Sao Paulo (2008 publication) [109]
65% of hemodialysis patients in Rio de Janeiro
52% of hemodialysis patients in Fortaleza (2004 publication) [110]
16.5% of hemodialysis patients in Goiania City (2002) [111]
23.8% of hemodialysis patients in Salvador (2001 publication) [112]
16.9% of hemodialysis patients in Mato Grosso (2002 to 2005) [113]
8.4% of hemodialysis patients in Recife (2007 publication) [114]
11% of hemodialysis patients in Campo Grande, Mato Grosso do Sul (2008 publication) [115]
13.9% of hemodialysis patients in Manaus (2009 publication) [116]
14.8% of hemodialysis patients in Juiz de For a, Minas Gerais (2007) [117]
16.1% of renal transplant recipients in Goias (2008 publication) [118]
47.9% of liver transplant recipients (1997 to 2007) [119]
7.4% of patients with hepatosplenic schistosomiasis (Recife, 2008) [120]
2.6% of leprosy outpatients (central Brazil, 2011 publication) [121]
7.1% of adults with psoriasis vs. 1.5% of the general population (Salvador, Bahia, 2012 publication) [122]
15% of alcoholic patients admitted to a detoxification unit - of these, 71% had detectable serum HCV-RNA (Porto Alegre, Southern Brazil, 2006 publication) [123]
2.53% of psychiatric patients (2005 to 2007) [124]
7.5% of COPD patients in Rio Grande do Sul, vs. 0.41% of blood donors (2009 publication) [125]
38.5% of HIV-positive patients (2006 publication) [126]
10.9% of HIV-positive patients (Mato Grosso State, 2007 publication) [127]
4.1% of HIV-positive patients (Recife, 2003) [128]
14% of HIV-positive patients (Sao Paulo, 2009 publication) [129]
8.1% of HIV-positive women in Salvador, Bahia (2012 publication) [130]
31.2% of HIV-positive patients (Rio Grande do Sul, 2010 publication) [131]
16.7% of HIV-positive outpatients (2005 to 2007) [132]
10.8% of pregnant HIV-positive women (Rio Grande do Sul, 2012 publication) [133]
35.9% of HTLV-1/2-positive blood donors in Ribeirao Preto City (2000 to 2010) [134]
7.5% of patients with tuberculosis in Goias State; and 67% of tuberculosis patients who were coinfected by HCV were found to be HIV-positive (2011 publication) [135]
4.42% of AIDS patients (Amazonas, 2000 to 2007) [136]
16.2% of female prisoners (Sao Paulo, 2006 publication) [137]
6.4% of incarcerated adolescents (Salvador, 2008 publication) [138]
9.7% of prison inmates (Rio Grande do Sul, 2012 publication) [139]

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8.7% of prison inmates (Sao Paulo, 2003) [140]
3.1% of prison inmates (Sergipe, 2011 publication) [141]
1.5% of army conscripts (2002) [142]
7.2% of former soccer players (Recife, 2011 publication) [143]
0.9% of dentists in Belo Horizonte (2009 publication) [144]
4.8% of health care workers in Rio Branco, Acre (2003 to 2004) [145]
1.7% of health care professionals and 0.2% of blood donor candidates in southeastern Brazil (1994 to 1999) [146]
12.4% of recyclable waste collectors in Santos (2005) [147]

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Hepatitis D

Agent	VIRUS - RNA. Deltavirus: Hepatitis D virus - a 'satellite' virus which is encountered as infection with a co-virus (Hepatitis B)
Reservoir	Human
Vector	None
Vehicle	Infected secretions Blood Sexual contact
Incubation Period	4w - 8w (range 2w - 20w)
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Needle precautions; supportive Interferon alfa 2-a has been used.
Typical Pediatric Therapy	As for adult
Clinical Hints	Vomiting and jaundice - biphasic course often noted; occurs as a coinfection or superinfection of hepatitis B; may be chronic or fulminant (combined hepatitis B and delta carries a worse prognosis than seen with hepatitis B alone).
Synonyms	Epatite D, Hepatitis delta. ICD9: 070.41,070.52 ICD10: B17.0

Clinical

WHO Case definition for surveillance of acute viral hepatitis (all types):
Clinical description
• Acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue, and right upper quadrant tenderness. [1]
• Biological signs include increased urine urobilinogen and >2.5 times the upper limit of serum alanine aminotransferase.
• Note: Most infections occur in early childhood. A variable proportion of adult infections is asymptomatic.
Laboratory criteria for diagnosis
• Hepatitis A: IgM anti-HAV positive
• Hepatitis B: Positive for Hepatitis B surface antigen (HBsAg) or IgM anti-HBc positive
• Non-A, non-B: IgM anti-HAV and IgM anti-HBc (or HBsAg) negative
Note 1: The anti-HBc IgM test, specific for acute infection, is not available in most countries.
• HBsAg, often available, cannot distinguish between acute new infections and exacerbations of chronic hepatitis B, although continued HBsAg seropositivity (>6 months) is an indicator of chronic infection.
Note 2: For patients negative for hepatitis A or B, further testing for a diagnosis of acute hepatitis C, D, or E is recommended:
Hepatitis C: anti-HCV positive
Hepatitis D: HBsAg positive or IgM anti-HBc positive plus anti-HDV positive (only as co-infection or super-infection of hepatitis B)
Hepatitis E: IgM anti-HEV positive
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory confirmed or, for hepatitis A only, a case compatible with the clinical description, in a person who has an epidemiological link with a laboratory-confirmed case of hepatitis A (i.e. household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

Clinical features of Hepatitis D:

Hepatitis D is characterized by gradual onset of abdominal pain and vomiting, followed by development of jaundice.
• A biphasic course often noted.
• When coinfection by hepatitis B is often present, the course may be chronic or fulminant. [2]
• 80% of patients with chronic hepatitis D infection progress to cirrhosis within 5 to 10 years. [3]

This disease is endemic or potentially endemic to all countries.

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Hepatitis D in Brazil

Hepatitis D is common in Sao Paulo and the western Amazon region.

Seroprevalence surveys:

34.4% of HBsAg-positive patients
3.8% of HBsAg-positive persons in Maranhao (2011 publication) [4]
13.4% to 66.6% HBsAg-positive Indians in the Western Amazon (2001 publication) [5]
41.9% of HBsAg-positive persons in the Western Amazon (2012 publication) [6]
0% of HBsAg-positive Parakana Indians in Para (2004) [7]
1.2% of HIV-HBV co-infected patients in Sao Paulo (2011 publication) [8]

Notable outbreaks:

2004 - An outbreak (20 or more fatal cases) was reported among Marubo Indians in the Javari Valley (western Amazonas). [9]

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Hepatitis E

Agent	VIRUS - RNA. Caliciviridae: Hepatitis E virus
Reservoir	Human Rodent Pig
Vector	None
Vehicle	Fecal-oral Water Shellfish Blood (rare) Meat (rare)
Incubation Period	30d - 40d (range 10d - 70d)
Diagnostic Tests	Identification of virus by immune electron microscopy (stool). Serology. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions; supportive
Typical Pediatric Therapy	As for adult
Vaccine	Hepatitis E
Clinical Hints	Clinically similar to hepatitis A - no chronic residua; severe or fatal if acquired during pregnancy (10% to 24% case-fatality rate).
Synonyms	Epatite E, Non-A, non-B enteric hepatitis. ICD9: 070.43,070.53 ICD10: B17.2

Clinical

WHO Case definition for surveillance of acute viral hepatitis (all types):
Clinical description
• Acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue, and right upper quadrant tenderness.
• Biological signs include increased urine urobilinogen and >2.5 times the upper limit of serum alanine aminotransferase.
• Note: Most infections occur in early childhood. A variable proportion of adult infections is asymptomatic.
Laboratory criteria for diagnosis
• Hepatitis A: IgM anti-HAV positive
• Hepatitis B: positive for Hepatitis B surface antigen (HBsAg) or IgM anti-HBc positive
• Non-A, non-B: IgM anti-HAV and IgM anti-HBc (or HBsAg) negative
Note 1: The anti-HBc IgM test, specific for acute infection, is not available in most countries.
• HBsAg, often available, cannot distinguish between acute new infections and exacerbations of chronic hepatitis B, although continued HBsAg seropositivity (>6 months) is an indicator of chronic infection.
Note 2: For patients negative for hepatitis A or B, further testing for a diagnosis of acute hepatitis C, D, or E is recommended:
Hepatitis C: anti-HCV positive
Hepatitis D: HBsAg positive or IgM anti-HBc positive plus anti-HDV positive (only as co-infection or super-infection of hepatitis B)
Hepatitis E: IgM anti-HEV positive
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspected case that is laboratory confirmed or, for hepatitis A only, a case compatible with the clinical description, in a person who has an epidemiological link with a laboratory-confirmed case of hepatitis A (i.e. household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

Clinical features of Hepatitis E:

In contrast to hepatitis A, hepatitis E is characterized by:
• relatively long incubation period
• prolonged clinical course
• severe and often fatal illness among pregnant women [1-4], patients with pre-existing hepatic cirrhosis [5], hemodialysis patients [6] and possibly women taking oral contraceptive medication. [7]
• poor protective value of immune serum globulin. [8]

In most hepatitis E outbreaks, the highest rates of clinically evident disease have been among young to middle-age adults.
• Lower disease rates in younger age groups may be the result of anicteric and/or subclinical HEV infection.
• Clinical disease in western countries and Japan is most common among males and persons above age 60 years. [9]

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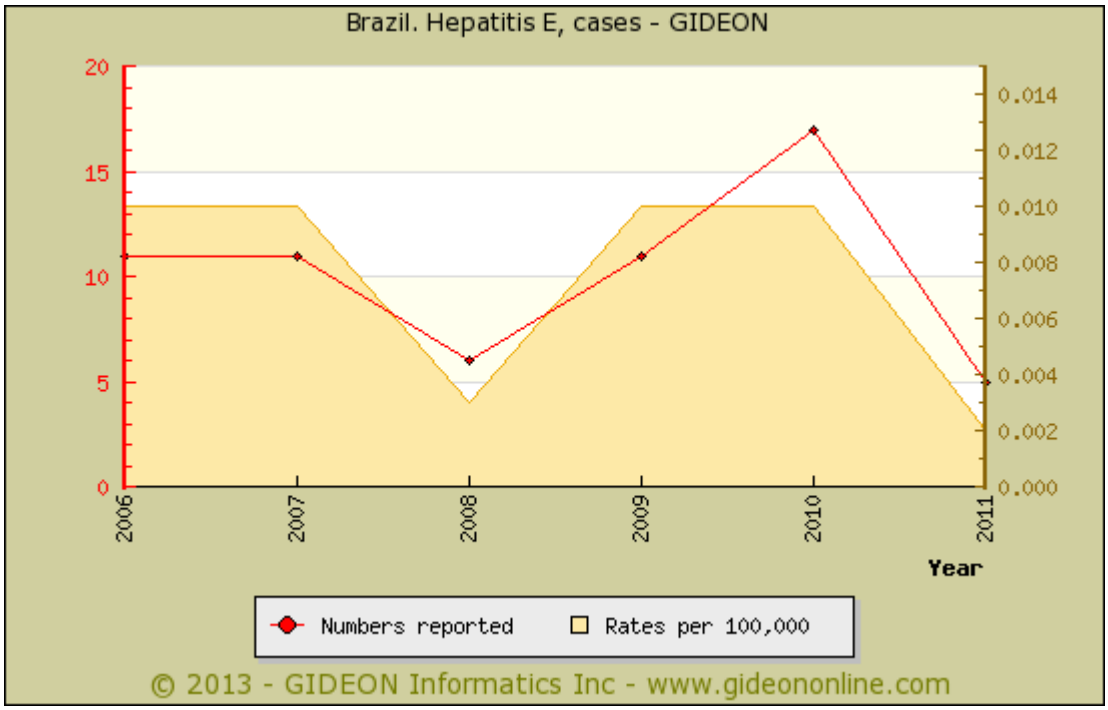
Clinical signs and symptoms are similar to those of other types of viral hepatitis and include abdominal pain anorexia, dark urine, fever, hepatomegaly, jaundice, malaise, nausea, and vomiting. [10]
• Less common findings include arthralgia, arthritis [11] , diarrhea, acute pancreatitis [12-14] , pruritus, an urticarial rash, severe thrombocytopenia [15] , photophobia, Guillain-Barre syndrome [16-18] , inflammatory polyradiculopathy [19] , encephalitis [20] [21] , aplastic anemia [22] , pregnancy associated with fetal ascites [23] , and hemophagocytic syndrome. [24]
• A false positive serological reaction toward Epstein-Barr virus has been reported in Hepatitis E virus infection. [25]
• The case fatality rate for young adults is 0.5% to 3%; 15% to 20% for pregnant women. [26]
• A subsequent publication estimated the CFR for all cases at 0.019 among non-pregnant patients vs. 0.198 among pregnant women. [27]

The period of infectivity following acute infection is not known; however, virus excretion in stools has been demonstrated up to 14 days after illness onset.
• The period of viral excretion appears to be prolonged among patients with hematological malignancy. [28]

Sporadic cases of chronic Hepatitis E virus infection are reported, notably among immunosuppressed patients. [29-39]
• Rare cases of chronic hepatitis E infection have been reported in immuno-competent individuals. [40] [41]

This disease is endemic or potentially endemic to all countries.

Hepatitis E in Brazil



Graph: Brazil. Hepatitis E, cases

Prevalence surveys:

3.1% of liver transplant recipients with unexplained liver enzyme abormalities (2013 publication) [42]

Seroprevalence surveys:

4% (Purus and Acre Rivers) to 6.2% (Mato Grosso) of healthy adults in the Amazon (2002 publication) [43]
2% of blood donors (Salvador, 1992 to 1994)
1.74% to 2.1% of persons with NANBNC hepatitis; 4% to 4.3% of blood donors, 11% to 11.8% of IDU, 2% to 6.2% of hemodialysis patients; 1% of pregnant women; and 0.74% to 2.1% of healthy individuals in Rio (2001 to 2005 publications) [44] [45]

2.3% of voluntary blood donors in Londrina, Para State (1999) [46]
2.4% of persons in northern Rio de Janeiro (2002 publication) [47]

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4.5% of school children in Mato Grosso (2002 publication) [48]

4% of persons exposed to swine, in Mato Grosso (2012 publication) [49]

6.3% of pig handlers, 1.42% of cows, dogs 6.97%, chickens 20%, swine 24.3%, and rodents 50% in southeastern Brazil (2005 publication) [50]

9.6% of bile samples from a swine slaughterhouse (Rio de Janeiro, 2010 publication) [51]

0.84% of bile samples and 1.7% of liver samples from pigs at slaughter (Parana, 2012 publication) [52]

8.6% of pigs at slaughter, in Belem (2010) [53]

62.5% of pig farms and 15.3% of pigs (south Brazil, 2012 publication) [54]

There is evidence for natural infection among swine in Brazil. [55]

Notable outbreaks:

1997 (publication year) - An outbreak of probable hepatitis E was reported in the Amazon region. [56]

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Hepatitis G

Agent	VIRUS - RNA. Flaviviridae, Hepacivirus: Hepatitis G virus. HGBV-A, B and C appear to be related
Reservoir	Human
Vector	None
Vehicle	Blood Vertical transmission has also been documented Sexual transmission suspected
Incubation Period	Unknown
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive. Alpha interferon has been shown to ? transiently eliminate the carrier state
Typical Pediatric Therapy	As for adult
Clinical Hints	Acute or chronic hepatitis acquired from blood (needles, etc); clinically milder than hepatitis C - most cases limited to anicteric elevation of hepatic enzyme levels; viremia documented for as long as 10 years.
Synonyms	Epatite G, Hepatitis GB. ICD9: 070,59 ICD10: B17.8

Clinical

Hepatitis G is characterized by acute or chronic hepatitis acquired from blood (needles, etc). [1]

- The disease is milder than hepatitis C, with most cases limited to anicteric elevation of hepatic enzyme levels. [2]
- Viremia has been documented for as long as 10 years.
- A case of aplastic anemia complicating Hepatitis G infection has been reported. [3]

This disease is endemic or potentially endemic to all countries.

Hepatitis G in Brazil

Prevalence surveys:

5.1% of healthy persons in Sao Paulo - 8.3% among persons ages 30 to 39 (2002 publication) [4]
10% of blood donors in Rio de Janeiro (1998 publication) [5]
38.6% of blood donors in Fortaleza (1999 publication) [6]
7.1% of blood donors in Goiania (Goias State, 2002 publication) [7]
9.7% of blood donors in Sao Paulo (2003 publication) [8]
14.6% of dialysis and kidney transplant recipients in Central Brazil (2004 publication) [9]
15% of hemodialysis patients and 19% of non A-C hepatitis patients (Rio de Janeiro, 1996) [10]
13% of patients with liver disease (Rio de Janeiro)
28% of patients with hepatocellular carcinoma (Recife, 2007 publication) [11]
9.5% of non A-E hepatitis patients
4.3% of preoperative cardiac surgery patients (Rio de Janeiro, 1996 to 1999) [12]

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Herpes B infection

Agent	VIRUS - DNA. Herpesviridae, Alphaherpesviridae, Simplexvirus: Cercopithecine herpesvirus 1 (Herpes B virus)
Reservoir	Monkey (usually Macaca species and cynomolgus)
Vector	None
Vehicle	Contact or bite
Incubation Period	10d - 20d (range 2d - 60d)
Diagnostic Tests	Viral culture (skin exudates). Nucleic acid amplification. Biosafety level 4.
Typical Adult Therapy	Therapy: Acyclovir 12 mg/kg IV q8h. OR Ganciclovir 5 mg/kg IV q12h. Follow with prolonged Acyclovir 800 mg PO 5X daily. Postexposure prophylaxis: Valacyclovir 1g PO q8h X 14 days. OR Acyclovir 800 mg PO X 5 X 14 days
Typical Pediatric Therapy	Acyclovir or Ganciclovir as for adult.
Clinical Hints	Vesicles, lymphadenopathy, myalgia, singultus, major neurological signs; usually within one month following contact with monkey; case-fatality rates exceed 80%. permanent neurological residua are common.
Synonyms	Cercopithecine herpesvirus 1, Herpes B, Herpesvirus simiae, Macacine herpesvirus 1. ICD9: 078.89 ICD10: B00.4

Clinical

Most human infections have been fatal, consisting of myelitis and hemorrhagic encephalitis with concomitant multiorgan involvement. [1]

The illness begins with fever, malaise, diffuse myalgia, nausea, abdominal pain and headache.
- Lymphadenitis is seen proximal to the site of inoculation.
- Dermal vesicles may be present.
- Abdominal pain and nausea may occur.
- Neurological findings then predominate, with dysesthesia, ataxia, diplopia, seizures, and ascending flaccid paralysis. [2]
- A lymphocytic CSF pleocytosis and elevated protein levels are noted, often with numerous erythrocytes.
- In contrast to herpes simplex infection, the encephalitis is multifocal.
- Rarely, isolated skin infection and even an isolated meningitis may be encountered.

This disease is endemic or potentially endemic to all countries.

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Herpes simplex encephalitis

Agent	VIRUS - DNA. Herpesviridae, Alphaherpesvirinae, Simplexvirus: Human herpesvirus (usually type I)
Reservoir	Human
Vector	None
Vehicle	Infected secretions, including Sexual contact
Incubation Period	Unknown
Diagnostic Tests	Viral culture CSF usually negative. CT brain. Compare CSF/blood antibody levels. Nucleic acid amplification.
Typical Adult Therapy	Acyclovir 10 mg/kg IV Q8h
Typical Pediatric Therapy	Acyclovir 10 mg/kg IV Q8h
Clinical Hints	Rapidly-progressive severe encephalitis, usually without exanthem; often unilateral, temporal and parietal lobe predominance; permanent residua and high case-fatality rate in untreated cases.
Synonyms	

Clinical

Although fever, headache, behavioral changes, confusion, focal neurological findings, and abnormal cerebrospinal fluid are suggestive of herpetic encephalitis, signs are not pathognomonic.

• Typical findings include fever, headache, psychiatric symptoms, altered consciousness, dysphagia, seizures [1] and vomiting. [2]

• Relatively severe and atypical presentations of encephalitis may occur in immunosuppressed patients. [3]

• Focal weakness, ataxia, hemiparesis, and memory loss are common.

• In some cases, patients exhibit memory loss, psychiatric disorders [4-11] , photophobia, cranial nerve deficits, papilledema, loss of visual fields, olfactory disturbance [12] , choreoathetosis [13] or other movement disorders. [14]

• Concurrent herpetic encephalitis and cutaneous herpes simplex are uncommon.

• Infection is usually frontotemporal and unilateral, and characterized by severe, often fatal disease. [15]

• Unilateral involvement of the temporoparietal region is typical, and helps distinguish herpetic infection from other forms of viral encephalitis • which tend to be bilateral and symmetrical. [16]

• Cases of overt cerebral hemorrhage [17] , acute disseminated encephalomyelitis [18] , Charles Bonnet syndrome (complex visual hallucinations) [19] and symmetric brain stem encephalitis have been reported. [20]

• West Nile viral encephalitis may mimic herpes simplex encephalitis. [21]

Herpes encephalitis is a risk factor for acute retinal necrosis. [22]

Relapse of encephalitis occurs in 12% of treated patients. [23] [24]

This disease is endemic or potentially endemic to all countries.

Herpes simplex encephalitis in Brazil

Prevalence surveys:

5% of presumed viral CNS infections in Ribeirao Preto (2007 publication) [25]

17.6% of encephalitis cases and 15% of meningitis cases in a dengue-endemic area (HSV-1, 2011 publication) [26]

10% of viruses associated with lympho-monocytic meningitis cases in Curitiba (2005 to 2006) [27]

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Herpes simplex infection

Agent	VIRUS - DNA. Herpesviridae, Alphaherpesvirinae, Simplexvirus: Human herpesvirus I and II
Reservoir	Human
Vector	None
Vehicle	Infected secretions, including Sexual contact
Incubation Period	1d - 14d
Diagnostic Tests	Viral culture or microscopy of lesions. Serology. Nucleic acid amplification.
Typical Adult Therapy	Acyclovir 400 mg PO TID X 7d. OR Famciclovir 250 mg PO TID X 7d. OR Valacyclovir 1 g PO BID X 7d
Typical Pediatric Therapy	Acyclovir 10 mg/kg PO QID X 7 d
Clinical Hints	Recurring localized crops of painful vesicles on a red base; regional adenopathy often present; may follow a prodrome of neuropathy or hyperesthesia.
Synonyms	Herpes gladiatorum, Herpes rugbiorum, Herpes simplex, Scrum pox. ICD9: 054.0,054.1,054.2,054.4,054.5,054.6,054.7,054.8,054.9 ICD10: A60,B00

Clinical

The initial attack of herpes simplex is generally more overt than recurrent episodes; however, primary infections are often asymptomatic. [1]

• Symptoms will also vary depending on the site of infection (eye [2] [3] , esophagus [4] [5] , anal region, etc).

Signs and symptoms:

Following a prodrome of local discomfort, tender papular, vesicular or ulcerative lesions on an erythematous base appear. [6]

• Anorexia, malaise and fever may accompany individual episodes.
• The lesions coalesce, and tender bilateral lymphadenopathy develops.
• Skin lesions usually heal over the next several days to weeks.
• Patients may give a history of occupational exposure (ie, herpetic whitlow, found in medical or dental personnel; herpes gladiatorum among wrestlers).
• Vesicular skin lesions of tularemia may mimic those of herpes simplex [7] ; and herpetic infection may present as folliculitis. [8]

Complications:

Immunosuppressed patients and neonates are at particular risk for disseminated and severe infections. [9-13]

• Lesions of the tongue may present as herpetic geometric glossitis. [14]
• Mucosal herpetic lesions may serve as a portal for bacterial invasion. [15]
• Ocular complications include conjunctivitis, scleritis [16] , severe keratitis and retinal necrosis. [17] [18]
• Corneal infection may present as epithelial keratitis (dendritica/geographica), stromal keratitis (necrotizing vs. non-necrotizing or "interstitial keratitis"), endotheliitis (disciform keratitis), neurotrophic keratopathy (metaherpetic keratitis) or vascularized corneal scars. [19-21]
• Over 10% of keratouveitis cases are complicated by secondary glaucoma [22] Herpetic keratitis may complicated ocular steroid injection [23]
• Herpes simplex infection has been etiologically linked to facial (Bell's) palsy. [24] [25]
• Pancreatitis [26] , esophagitis [27] , cardiomyopathy [28] and rhabdomyolysis with renal failure have been reported to complicate herpes simplex infection. [29]
• Herpes simplex hepatitis is most common in the setting of pregnancy or immune suppression [30] ; however, rare instances of hepatitis and fulminant hepatic failure due to HSV infection have been reported in immunocompetent persons. [31-35]
• HSV-related erythema multiforme [36] [37] has been reported in stem-cell transplant recipients [38]
• Disseminated infection among patients with eczema (Eczema herpeticum) may resemble smallpox [39] [40] or present as atopic dermo-respiratory syndrome. [41]
• Chronic (>1 month) mucocutaneous infections may occur in HIV-positive patients, in the absence of disseminated disease. [42]
• Herpetic lesions in HIV-positive patients may be vegetative, hypertrophic, condyloma-like, nodular, ulcerative, or tumor-like

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nodules or plaques. [43] [44]
• Herpes simplex may contribute to the pathology of periodontitis. [45]
• Herpes simplex has been reported to cause Gerhardt syndrome (inspiratory dyspnea without dysphonia) from vocal cord paralysis [46]

Anterior uveitis • differential diagnosis:
Anterior uveitis due to Rubella virus is characterized by younger age at onset and a chronic course, typically associated with cataract at presentation. [47]
• Rubella virus has been implicated in the etiology of Fuchs heterochromic iridocyclitis. [48]
• Anterior uveitis due to Herpes simplex and Varicella-Zoster viruses is more common in adults, and often follows an acute course.
• Herpes simplex anterior uveitis presents with conjunctival redness, corneal edema, a history of keratitis, and the presence of posterior synechiae. Anterior chamber inflammation is common with Herpes simplex virus, while vitritis is more common with Rubella and Varicella-Zoster virus.
• Rubella, Herpes simplex and Varicella-zoster viruses are associated with intraocular pressure of more than 30 mmHg and development of glaucoma (18%-30%; P = 0.686).
• Focal chorioretinal scars were present in 22% of Rubella cases, 0% of HSV and in 11% of VZV uveitis cases.

Acquisition of Herpes simplex by the newborn at the time of delivery is associated with severe illness and results in death in approximately 50% of cases. [49]
• Neonatal herpes simplex infection is characterized by vesicular rash, hypothermia, lethargy, seizures, respiratory distress, hepatosplenomegaly, thrombocytopenia, hepatic dysfunction and cerebrospinal fluid pleocytosis. [50] [51]

Herpes simplex virus is an important cause of encephalitis (discussed separately in this module) and keratitis. [52]

This disease is endemic or potentially endemic to all countries.

Herpes simplex infection in Brazil

Prevalence surveys:

56% of all ocular viral cultures, 76% of lid and 88% of corneal isolates. (HSV-1). (Sao Paulo, 1987 to 2001) [53]
5% of CSF specimens from cases of suspected viral CNS infection (HSV-1, Sao Paulo, 2006 publication) [54]
17.6% of encephalitis cases and 15% of meningitis cases in a dengue-endemic area (HSV-1, 2011 publication) [55]

Seroprevalence surveys:

67.2% (HSV-1) and 11.3% (HSV-2) of persons ages 1 to 40 years (four geographic regions, 1996 to 1997) [56]
46% of women (HSV-2, Sao Paulo, 1993 publication) [57]
55.4% of urban children (HSV, 2011 publication) [58]
73% of blood donors (HSV-2, Rio de Janeiro, 1994) [59]
66.3% of students, 97.1% of pregnant women and 99.0% of STD patients (Campinas, 1999 publication) [60]
53% of men and 50.7% of women with AIDS in Niteroi, Rio de Janeiro (HSV-2, 2006 publication) [61]
45.7% of MSM in Rio de Janeiro (HSV-2, 2009 publication) [62]
83.5% (HSV-1) and 63.4% (HSV-2) of HIV-negative male homosexuals and bisexuals (2011 publication) [63]
51.1% of men and 72.1% of women in Alto Solimoes, Amazon border region (HSV-2, 2012 publication) [64]
26.6% of truck drivers crossing the southern Brazilian border at Foz do Iguacu (HSV-2, 2003 to 2005) [65]

Natural infection by human herpesvirus 1 has been identified in marmosets (*Callithrix* spp) from the Campo Grande district of Rio de Janeiro. [66]

Notable outbreaks:

2006 - An outbreak (13 cases) of Human Herpesvirus 1 infection was reported among black-tufted marmosets (*Callithrix penincillata*) . [67]

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Herpes zoster

Agent	VIRUS - DNA. Herpesviridae, Alphaherpesvirinae: Varicella-zoster virus
Reservoir	Human
Vector	None
Vehicle	Air Direct contact
Incubation Period	Unknown
Diagnostic Tests	Viral culture (vesicles). Serology. Nucleic acid amplification.
Typical Adult Therapy	Acyclovir 800 mg PO X 5 daily X 7 to 10d. OR Famciclovir 500 PO TID. OR Valacyclovir 1 g PO TID
Typical Pediatric Therapy	Acyclovir 20 mg/kg PO QID X 7 to 10d
Vaccine	Herpes zoster
Clinical Hints	Unilateral dermatomal pain, tenderness and paresthesia followed in 3 to 5 days by macular, erythematous rash evolving to vesicles; trunk and chest most common, but other areas possible; patients usually above age 50.
Synonyms	Fuocodi Saint'Antonio, Shingles, Zona, Zoster. ICD9: 053 ICD10: B02

Clinical

The condition represents reactivation of dormant Varicella-Zoster virus in dorsal root ganglia.

Disease is characterized by grouped vesicular lesions distributed along one to three sensory dermatomes, usually unilateral and on the trunk or face. [1]
• Mild pruritis or excruciating pain may be present, and persis after the disappearance of the rash.
• Although pain typically presents for 1 to 3 days prior to the appearance of a rash, the pre-eruptive prodromal period may persist for as long as 18 days. [2]
• In immunocompromised individuals, herpes zoster may become disseminated.
• A chronic verrucous form of herpes zoster seen in HIV-positive patients is associated with antiviral drug-resistance. [3]

Most healthy persons recover without complications; however, individuals above age 50 years are at increased risk of postherpetic neuralgia which may persist for months to years after the rash has healed.
• The possible effect of antiviral drugs in prevention of pos-herpetic neuralgia is controversial. [4]
• Immunocompromised patients are risk for chronic herpes zoster; or infection of the central nervous system [5] , liver, lungs or pancreas.
• Chronic (>1 month) mucocutaneous infections may occur in HIV-positive patients, in the absence of disseminated disease. [6]
• Visual impairment or scleral damage may follow zoster ophthalmia. [7-9] Over 10% of keratouveitis cases are complicated by secondary glaucoma [10] Rare instances of orbital apex syndrome [11] and optic neuritis are also reported. [12]
• VZ virus infection may be associated with myotomal paresis [13] , facial nerve palsy [14] or Ramsay-Hunt syndrome (Bell palsy unilateral or bilateral, vesicular eruptions on the ears, ear pain, dizziness, preauricular swelling, tingling, tearing, loss of taste sensation, and nystagmus) [15]
• VZ virus infection can be a presenting symptom of hyperparathyroidism and occurs twice as often in persons with hypercalcemia than age-matched controls. [16]
• In some cases, reactivation of VZ virus may present as radiculitis, cranial nerve palsy or other features of herpes zoster • but without rash (zoster sine herpete). [17]

This disease is endemic or potentially endemic to all countries.

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Heterophyid infections

Agent	PARASITE - Platyhelminthes, Trematoda. Plagiorchiida, Heterophyidae: Heterophyes heterophyes, H. nocens, et al
Reservoir	Snail (Cerithidea cingulata, Pirenella conica) Fish
Vector	None
Vehicle	Fish (mullet and Tilapia)
Incubation Period	7d - 14d
Diagnostic Tests	Identification of ova or adults in stool.
Typical Adult Therapy	Praziquantel 25 mg/kg TID X 3 doses
Typical Pediatric Therapy	As for adult
Clinical Hints	Abdominal pain and mucous diarrhea with eosinophilia beginning 1 to 2 weeks after ingesting undercooked fish; infestation resolves spontaneously within two months.
Synonyms	Acanthogobius, Apophallus, Ascocotyle, Brachylaima, Carneophallus, Cotyluris, Cryptocotyle, Diorchitrema, Gymnophalloides, Gynaecotyla, Haplorchis, Heterophyes, Heterophyopsis, Paralecithodendrium, Phagicola, Phaneropsolus, Procerovum, Prosthodendrium, Pygidiopsis, Small intestinal trematodes, Spelotrema, Stellantchasmus, Stictodora. ICD9: 121.6 ICD10: B66.8

Clinical

As a group, these infestations are characterized by abdominal pain and mucous diarrhea, often associated with eosinophilia. [1]

- Asymptomatic infection is common.
- Rarely, metastatic granulomata in various organs have been ascribed to ectopic ova.

This disease is endemic or potentially endemic to 38 countries. Although Heterophyid infections is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Heterophyid infections in Brazil

Infection by *Ascocotyle* (*Phagicola*) spp., trematodes acquired from freshwater fish, have been reported in Cananeia and Registro (Sao Paulo State) - the first case in 1987. [2] [3]

- The local reservoir is raw mullet (*Mugil* spp.). [4]
- The first intermediate host is the cochliopid snail, *Heleobia australis* [5]

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Histoplasmosis

Agent	FUNGUS. Ascomycota, Euascomycetes, Onygenales: Histoplasma capsulatum var. capsulatum A dimorphic fungus
Reservoir	Soil Caves Chicken roosts Bat
Vector	None
Vehicle	Air
Incubation Period	10d - 14d (range 5d - 25d)
Diagnostic Tests	Fungal culture. Serologic tests less helpful. Antigen tests currently under study. Nucleic acid amplification.
Typical Adult Therapy	Itraconazole 200 mg daily X 9m For severe or immunocompromized patients: Amphotericin B 0.4 mg/kg/d X 6w, then 0.8 mg/kg qod X 8w
Typical Pediatric Therapy	Itraconazole 2 mg/kg daily X 9 m. For severe or immunocompromized patients: Amphotericin B 0.4 mg/kg/d X 6w, then 0.8 mg/kg qod X 8w
Clinical Hints	Fever, cough, myalgia, pulmonary infiltrates and calcifying hilar lymphadenopathy; chronic multisystem infection often encountered.
Synonyms	Darling's disease, Histoplasma capsulatum, Histoplasmose, Ohio River Valley Fever, Ohio Valley disease, Reticuloendothelial cytomycosis. ICD9: 115.0 ICD10: B39.0,B39.1,B39.2,B39.3,B39.4

Clinical

Asymptomatic infection is common, and may be found as an incidental finding on chest X-ray, or through serological or skin tests. [1]

Pulmonary histoplasmosis:

Acute benign respiratory infection is characterized by weakness, fever, chest pains, and cough. [2]
• The severity of illness is related to the magnitude of the exposure.
Chronic pulmonary infection occurs in persons with pre-existing lung diseases such as emphysema.
• The infection is most common in males over the age of 40.
• Chronic pulmonary lesions are characterized by extensive cavitation, but may resemble those of tuberculosis. [3]

Disseminated histoplasmosis:

Disseminated infection is seen in immunocompromised patients (AIDS [4-6], leukemia, corticosteroid therapy, anti-TNF therapy [7], etc) and may be characterized by fever, anemia, hepatitis, pneumonia, pleuritis, pericarditis [8], acalculous cholecystitis [9], meningitis, atypical skin lesions (10% of cases) [10] [11] and ulcers of the mouth [12], tongue [13], nose [14], paranasal sinuses [15], esophagus [16] [17], colon [18-20] and larynx. [21] [22]
• Associated findings include upper lobe cavitation with fibrosis (similar to tuberculosis); sclerosing mediastinitis with obstruction of the superior vena cava, pulmonary arteries and veins; esophagus; and constrictive pericarditis. [23]
• Fungemia is most common in patients with immunosuppression or neutropenia (<3,000 per cu mm). [24]
• Central nervous system infection can present at chronic meningitis, focal parenchymal lesions of the brain or spinal cord, stroke due to infected emboli, and diffuse encephalitis. [25]
• Spinal infection may mimic tuberculosis spondylodiscitis. [26]
• Adrenal masses [27-31] and renal infection are occasionally reported [32] [33] and may mimic carcinoma. [34]
• Peritoneal histoplasmosis has been reported as a complication of peritoneal dialysis. [35]
• Epididymo-orchitis and prostatitis are occasionally reported. [36-38]
• Gastrointestinal infection may mimic colonic carcinoma [39] [40] or abdominal tuberculosis. [41]
• Dermatological manifestations include erythema nodosum [42], erythema multiforme [43], purpuric lesions, or the appearance of ulcerating verrucous plaques [44] [45] Primary infection may present as a dermal nodule with regional adenopathy. [46] Skin lesions may mimic secondary syphilis. [47]
• Hypercalcemia has been reported in some cases. [48-52]

"Ocular histoplasmosis syndrome" is characterized by peripapillary atrophy, punched out lesions, a macular disciform lesion or scar in one eye without vitritis.

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• The role of *Histoplasma capsulatum* in this condition is unclear. [53] [54]
• Overt *Histoplasma* keratitis has been reported [55]

Acute disseminated infection is also seen in infants and young children and is marked by fever, cough, exhaustion and hepatosplenomegaly. [56]
• Roentgenographic findings include multiple nodules (3 to 4 mm) changing into punctate calcifications; histoplasmoma (non-calcifying nodules <3 mm); a "target lesion" (ie, central calcification); or hilar/mediastinal adenopathy ("popcorn" calcification).

Primary histoplasmosis of the mouth has been reported. [57]

This disease is endemic or potentially endemic to 93 countries.

Histoplasmosis in Brazil

The disease is found in scattered areas of the north, south and center [58] ; however, most outbreaks are reported from Rio de Janeiro state. [59]

Nine cases were reported to 1952 - including 5 cases identified during 1939 to 1945. [60]

156 cases were registered in Porto Alegre (Rio Grande do Sul) during 1978 to 1999. [61]
- 11 cases of oral histoplasmosis (5 HIV-positive) were treated in a single institution in Porto Alegre during 1987 to 2008. [62]
- 30 cases were registered at a University Hospital in Mato Grosso do Sul during 1998 to 2005. 97.7% had disseminated infection and 83.3% had AIDS. [63]
- 164 cases were reported among HIV-positive patients in Fortaleza (2007 publication) [64]
- 208 cases were reported among HIV-positive patients in Ceara during 2006 to 2010. [65]

Prevalence surveys:

0.1% of solid organ transplant recipients (2001 to 2006) [66]

0.2% of solid organ transplant recipients (2001 to 2006) [67]

13% of autopsied AIDS patients (Amazonas, 1996 to 2003) [68]

Systemic fungal diseases were found in 4.6% of AIDS patients in Cuiaba, Mato Grosso - *Cryptococcus neoformans* in 50% of these, *Cryptococcus neoformans var. gattii* 1.6%, *Cryptococcus* spp in 6.6%, *Histoplasma capsulatum* 38.3% and *Paracoccidioides brasiliensis* 3.3% (2005 to 2008) [69]

10.1% of deaths among AIDS patients (1996 to 2006) [70]

3.6% of insectivorous bats (*Molossus molossus*, *Nyctinomops macrotis*, *Tadarida brasiliensis*, *Molossus rufus* and *Eumops glaucinus*) in Sao Paulo State (2003 to 2008) [71]

Seroprevalence surveys:

3.9% of Xacriaba Indians (skin test, Minas Gerais, 2002 publication) [72]

44% of persons in Ibia town, Minas Gerais (skin test, 2008 publication) [73]

9.7% of dogs in Rio de Janeiro (skin-test, 1987 publication) [74]

1.78% of dogs in northeastern Brazil (2011 publication) [75]

Notable outbreaks:

1967 (publication year) - An outbreak of histoplasmosis was reported in Sao Paulo. [76]

1976 (publication year) - An outbreak was reported in Sao Paulo. [77]

1993 - An outbreak (5 cases) was reported among Japanese tourists following a visit to a cave in Manaus. [78]

1997 - An outbreak (4 cases, 0 fatal) was reported following contact with a bat cave in Pedro Leopoldo (Minas Gerais State). [79]

2000 (publication year) - An outbreak (4 cases) was reported among members of a family. [80]

2007 (publication year) - An outbreak (2 cases) was reported in Santa Catarina. [81]

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HIV infection - initial illness

Agent	VIRUS - RNA. Retroviridae, Lentivirinae: Human Immunodeficiency Virus
Reservoir	Human
Vector	None
Vehicle	Blood Semen Sexual Transplacental Breast-feeding
Incubation Period	1w - 6w
Diagnostic Tests	HIV antibody (ELISA, Western blot). HIV or HIV antigen assays. Nucleic acid amplification.
Typical Adult Therapy	Supportive; 'prophylactic' Zidovidine + additional drugs (DDI, 3TC, etc) should be considered - particularly during pregnancy
Typical Pediatric Therapy	Supportive; role for 'prophylactic' Zidovidine + additional drugs (DDI, 3TC, etc) should be considered
Clinical Hints	Fever, diarrhea, sore throat and a mononucleosis-like illness in a 'high risk' patient (eg, men who have sex with men, drug abuser, etc).
Synonyms	HIV, HIV infection. ICD9: 042 ICD10: B20,B21,B22,B23,B24

Clinical

The clinical features of acute HIV infection are protean and often characterized by fever, generalized lymphadenopathy, headache, fatigue, myalgia, rash, nausea, vomiting, night sweats, sore throat, diarrhea or weight loss. [1]
• 40% to 90% of persons have symptoms suggestive of an acute viral infection.
• Symptoms tend to subside within two weeks; however, some patients continue to be ill for as long as ten weeks.
• In most cases, a history of likely acquisition within the past several weeks can be established: unprotected sex, extra-medical injection, transfusion, etc.

This disease is endemic or potentially endemic to all countries.

HIV infection - initial illness in Brazil

Data and background information regarding HIV infection are included in the note for AIDS

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Hookworm

Agent	PARASITE - Nematoda. Phasmidea: Necator americanus, Ancylostoma duodenale, A. ceylonicum (in Calcutta and the Philippines)
Reservoir	Human
Vector	None
Vehicle	Soil Contact
Incubation Period	7d - 2y
Diagnostic Tests	Examination of stool for ova.
Typical Adult Therapy	Albendazole 400 mg X 1 dose. OR Mebendazole 100 mg BID X 3d. OR Pyrantel pamoate 11 mg/kg (max 3g) X 3d; or
Typical Pediatric Therapy	As for adult
Clinical Hints	Pruritic papules (usually of feet) - later cough and wheezing; abdominal pain and progressive iron-deficiency anemia; eosinophilia common; dyspnea and peripheral edema in heavy infections; *Ancylostoma caninum* implicated in eosinophilic enteritis.
Synonyms	Anchilostoma, Ancylostoma ceylanicum, Ancylostoma duodenale, Ancylostomiasis, Anquilostomiasis, Cyclodontostomum, Eosinophilis enteritis, Hakenwurmer-Befall, Miner's anemia, Necator americanus, Necatoriasis, Uncinariasis. ICD9: 126.0,126.1 ICD10: B76.0,B76.1,B76.8

Clinical

Initial manifestations of hookworm consist of pruritus, erythema, and a papular, or vesicular rash at the site of larval penetration ("ground itch"). [1]

• Migration of larvae through the lungs may result in a Loeffler-like syndrome with transitory cough, wheezing, diffuse opacities on x-ray and eosinophilia in sputum and blood. [2] [3]

• Migration of *A. duodenale* larvae to the breast, with infection of nursing infants ('hypobiosis') has been described. [4]

• The major finding in overt infection is iron-deficiency anemia. [5]

• Heavy intestinal infection may also produce local symptoms of abdominal pain, diarrhea, and occasionally malabsorption with weight loss (most commonly in children).

• Rare instances of overt bleeding [6] or melena have been reported. [7]

This disease is endemic or potentially endemic to all countries.

Hookworm in Brazil

Prevalence surveys:

5.6% of persons in Uberlandia (Minas Gerais, 1998)

8.5% of homeless persons in Rio de Janeiro (2002 publication) [8]

9.9% of persons in Eirunepe, Amazon (2005 publication) [9]

38.5% of individuals in a community in Minas Gerais were coinfected with hookworm and *Schistosoma mansoni* (2009 publication) [10]

33.3% of the Parakana indigenous community in southwestern Para State (1992 to 1995) [11]

75% of Hupda and 19.3% of other indigenous peoples in Iauarete (2001) [12]

7% of Terena Indians (Mato Grosso do Sul) [13]

9.4% of persons in southeastern Piau (2003 publication) [14]

71.1% of persons in southeastern Brazil (2008 publication) [15]

80.2% of persons in Sao Lourenco da Mata, rural Recife (Pernambuco, 1989) [16]

68.2% of persons in Americaninhas, Minas Gerais (2004) [17]

69.8% of persons in Americaninhas, Minas Gerais (2004) [18]

9.5% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [19]

0.2% of persons in Maria Helena, Parana (2004 to 2006) [20]

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12.1% of school children in northwestern Minas Gerais (2002 publication) [21]
72.45% of school children in Americanihnas, Minas Gerais (2008 publication) [22]
1.7% of primary school children in Aracaju, Sergipe (1999 publication) [23]
6% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [24]
3.1% of children in Uberlandia, Minas Gerais (2008 publication) [25]
15.7% of children in rural Bahia (2006 publication) [26]
15.3% of children in northeastern Brazil. (2010 publication) [27]
0% of children in an urban slum in Sao Paulo (2008 publication) [28]
8.4% of school children in Salvador (1997) [29]
0% of urban children with diarrhea in Salvador (2000 to 2002) [30]
13.7% of HIV-positive patients before HAART vs. 2% after HAART (Ceara, 2008 publication) [31]
1.4% of HIV-positive patients in northeastern Sao Paulo (1998 to 2008) [32]
9.1% of dog keepers working in breeding kennels in Uberlandia, southeastern Brazil. (Minas Gerais, 2007 publication) [33]
6.9% of soil samples in Fernandopolis, Sao Paulo State (*Ancylostoma* sp., 2007 to 2008) [34]
46.8% of soil samples from public parks and squares in Guarulhos, Sao Paulo State (2010) [35]

Hookworm-related cutaneous larva migrans was detected in 4.4% of village dwellers during the rainy season, and 1.7% during the dry season; prevalence was highest (14.9%) among children below 5 years of age. (North-Eastern Brazil, 2008 publication) [36]

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Human herpesvirus 6 infection

Agent	VIRUS - DNA. Herpesviridae, Betaherpesvirinae, Roseolovirus: Herpesvirus 6 (Herpesvirus 7 is also implicated)
Reservoir	Human
Vector	None
Vehicle	Droplet Contact
Incubation Period	10d - 15d
Diagnostic Tests	Viral isolation and serologic tests rarely indicated. Nucleic acid amplification has been used
Typical Adult Therapy	Supportive Gancyclovir has been used in unusual and severe cases.
Typical Pediatric Therapy	As for adult
Clinical Hints	High fever followed by sudden defervescence and fleeting rash; most patients are below the age of 2 years; only 10% to 20% of herpesvirus 6 infections are associated with a rash.
Synonyms	Dreitagefieber, Exanthem criticum, Exanthem subitum, Herpesvirus 6, HHV-6, Pseudorubella, Roseola, Roseola infantilis, Roseola subitum, Sixth disease, Zahorsky's disease. ICD9: 057.8 ICD10: B08.2

Clinical

Roseola typically is characterized by high fever (often to 40 C) lasting from three to seven days, followed by rapid defervescence and a characteristic pink rash. [1] [2]

- The rash is maculopapular or erythematous, beginning on the trunk and spreading to the neck and extremities. [3]
- Skin lesions are discrete, not pruritic, blanch on pressure and fade within 3 to 48 hours.

Diarrhea, cough and irritability are common, and seizures may rarely occur in individual cases. [4]

- HHV-6 infection accounts for 10% to 20% of febrile seizures in children below the age of two years. [5] [6]
- Other findings may include bulging anterior fontanel, Nagayama spots (erythematous papules on the soft palate and uvula), periorbital edema, inflamed tympanic membranes, cervical, post auricular, and post occipital lymphadenopathy, splenomegaly, meningitis with radiculitis [7] , encephalopathy or encephalitis [8-16] , arthropathy (4.3% of cases) [17] , rhabdomyolysis [18] , uveitis [19] [20] , optic neuritis [21] , corneal inflammation [22] and conjunctival injection.
- Rare instances of acute hepatic failure [23] and purpura fulminans have been reported. [24]

Reactivation and severe disease have been encountered in bone-marrow, solid organ transplant and other immune-deficient patients. [25-28]

- HHV-6-associated pleurisy has been reported following stem-cell transplantation (2007 publication) [29]
- Fatal hepatitis and myocarditis has been reported in immunocompetent adults. [30] [31]

This disease is endemic or potentially endemic to all countries.

Human herpesvirus 6 infection in Brazil

Prevalence surveys:

43.5% of children with rash, after discounting measles, rubella, dengue fever, and parvovirus B19 (Rio de Janeiro, 1998 to 2006) [32]

38.4% of (? symptomatic) children in whom measles, rubella, dengue fever and parvovirus B19 infection were excluded (Rio de Janeiro, 1998 to 2006) [33]

2.1% of patients with maculopapular rash (Niteroi, Rio de Janeiro State, 1994 to 1998) [34]

17% of patients with fever and rash, initially diagnosed as measles (Sao Paulo, 2000 to 2004) [35]

60.1% of patients with fever and rash in Campinas (2003 to 2004) [36]

3% of viruses associated with lympho-monocytic meningitis cases in Curitiba (2005 to 2006) [37]

9.8% of healthy adults (Rio de Janeiro, saliva PCR, 2010 publication) [38]

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39.2% of adult liver transplant recipients, within 6 months of transplantation (antigenemia, 2011 publication) [39]
26.7% of liver transplant recipients, within 6 months post-transplantation (antigenemia, 2012 publication) [40]
36.6% of liver transplant recipients (pre-transplant biopsy, 2011 publication) [41]
40% of liver transplant recipients, within 6 months to one year (Sao Paulo, 2011 publication) [42]
25% of pretransplant and 27% of post renal transplant recipients (Sao Paulo, 2007 to 2008) [43]

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Hymenolepis diminuta infection

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Hymenolepididae: Hymenolepis diminuta
Reservoir	Rodent Various insects
Vector	None
Vehicle	Arthropod - ingestion
Incubation Period	2w - 4w
Diagnostic Tests	Identification of ova in stool
Typical Adult Therapy	Praziquantel 25 mg/kg as single dose. OR Niclosamide 2g, then 1g/d X 6d
Typical Pediatric Therapy	Praziquantel 25 mg/kg as single dose. OR Niclosamide 1g, then 0.5g/d X 6d (1.5g, then 1g for weight >34kg)
Clinical Hints	Nausea, abdominal pain and diarrhea; eosinophilia may be present; primarily a pediatric disease, in rodent-infested areas; infestation resolves spontaneously within 2 months.
Synonyms	Hymenolepis diminuta, Mathevotaenia, Rat tapeworm. ICD9: 123.6 ICD10: B71.0

Clinical

Patients, usually children, may develop mild abdominal pain, nausea diarrhea and eosinophilia. [1]

This disease is endemic or potentially endemic to all countries.

Hymenolepis diminuta infection in Brazil

Prevalence surveys:

4% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [2]
5% of children in Uberlandia, Minas Gerais (2008 publication) [3]

Hymenolepis diminuta ova have been identified on vegetables consumed by school children in Sao Paulo. [4]

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Hymenolepis nana infection

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Hymenolepididae: Hymenolepis (Rodentolepis) nana
Reservoir	Human Rodent (especially hamster)
Vector	None
Vehicle	Food Water Fecal-oral
Incubation Period	2w - 4w
Diagnostic Tests	Identification of ova in stool
Typical Adult Therapy	Praziquantel 25 mg/kg once. OR Nitazoxanide 500 mg daily for 3 days OR Niclosamide 2g/d X 1, then 1g/d X 6d
Typical Pediatric Therapy	Praziquantel 25 mg/kg once. OR Nitazoxanide 100 mg (age 1 to 3 years) to 200 mg (age 4 to 11 years) BID X 3d OR Niclosamide 1g/d X 1, then 0.5g/d X 6d (1.5g, then 1g for weight >34kg)
Clinical Hints	Nausea, abdominal pain, diarrhea, irritability and weight loss; eosinophilia may be present; infection is maintained by autoinfection (worm reproduces within the intestinal lumen).
Synonyms	Dwarf tapeworm, Hymenolepis nana, Rodentolepis (Hymenolepis) microstoma, Rodentolepsiasis, Vampirolepis nana. ICD9: 123.6 ICD10: B71.0

Clinical

Infestation by *Hymenolepis nana* is largely asymptomatic. [1]

- Children are most likely to exhibit symptoms consisting of abdominal pain and diarrhea. [2]
- Pruritis ani and behavioral and sleep disturbances are occasionally encountered. [3]
- Most patients have eosinophilia (5% to 10% of total leucocyte count).

This disease is endemic or potentially endemic to all countries.

Hymenolepis nana infection in Brazil

Prevalence surveys:

9.4% of persons in southeastern Piau (2003 publication) [4]
0.2% of persons in Americaninhas, Minas Gerais (2004) [5] [6]
8.3% of Terena Indians (Mato Grosso do Sul) [7]
1.3% of school children in Salvador (1997)
0.5% of children in Sao Paulo (2004 publication) [8]
0.2% of Sao Paulo children below age 5 years (1995 to 1996)
6.7% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [9]
7.5% of children in Uberlandia, Minas Gerais (2008 publication) [10]
0% of HIV-positive patients before HAART vs. 1% after HAART (Ceara, 2008 publication) [11]

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Ilheus and Bussuquara

Agent	VIRUS - RNA. Flaviviridae, Flavivirus. Ilheus virus and Bussuquara virus
Reservoir	? Wild bird
Vector	Mosquito (Aedes, Culex, Coquillettidia, Haemagogus, Psorophora, Sabethes, Trichoprosopon and Wyeomyia spp.)
Vehicle	None
Incubation Period	Unknown
Diagnostic Tests	Viral culture (blood). Serology. Biosafety level 4.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, headache, arthralgia and myalgia; encephalitis occasionally encountered; no fatalities or complications reported to date.
Synonyms	Bussuquara, Ilheus. ICD9: 062.8 ICD10: A83.8

Clinical

Ilheus is usually self-limited, and characterized by fever, headache, photophobia, myalgia and arthralgia. [1]
• Encephalitis occurs in approximately 15% of patients.
• No fatal cases have been reported.

This disease is endemic or potentially endemic to 10 countries.

Ilheus and Bussuquara in Brazil

Ilheus virus was first identified in Ilheus City, eastern Brazil, in 1944.

Seroprevalence surveys:

5.9% of inhabitants of Rio Branco, Acre (1999) [2]
2% in Corte de Pedra, Valencia (Bahia, 1986 publication) [3]
26.6% of horses in the Brazilian Pantanal (1993 publication) [4]
10.7% of horses and caimans from the Pantanal (2009) [5]

In addition to Ilheus, another flavivirus named Bussuquara has been implicated in cases of fever with arthralgia in this country. [6]
- The vector is *Culex melaconion* spp., and a rodent reservoir is suspected.

Five cases of infection by a virus closely related to Ilheus virus were published in 2000 - acquired Maranhao, Parana, Goias, Amazonas, and Rondonia. [7]

Ilheus virus has been isolated from birds (*Sporophila caerulescens* and *Molothrus bonariensis*).
- Specific serum antibodies are detected in additional bird species (*Columbina talpacoti*, *Geopelia cuneata*, *Sicalis flaveola* and *Molothrus bonariensis*), marmosets (*Callithrix jacchus* and *Callithrix penicillata*) and coati (*Nasua nasua*). [8]
- Seropositive birds have been identified in Sao Paulo State (1978 to 1990) [9]

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Infection of wound, puncture, IV line, etc

Agent	BACTERIUM. Staphylococcus aureus, streptococci, facultative or aerobic gram negative bacilli, anaerobes, et al
Reservoir	Human Soil Water Air (spores) Various animals and plants
Vector	None
Vehicle	Trauma Water Medications Bandages Autoinoculation
Incubation Period	Variable
Diagnostic Tests	Smear and culture of catheter, material from wound.
Typical Adult Therapy	Drainage, remove catheter, debridement and antibiotics appropriate to infecting species
Typical Pediatric Therapy	As for adult
Clinical Hints	Source (ie, venous line, postoperative, marine, animal bite) may suggest species; onset less than 24 hrs = group A Strep. or *Cl. perfringens*; 2 to 7 days *S. aureus*; over 7 days gram negative bacilli; foul odor anaerobes.
Synonyms	Intravenous catheter infection, Line infection, Surgical wound infection, Wound infection. ICD9: 686.9,451 ICD10: T79.3,I80.0, Y95

Clinical

Wound infection is a self-defined illness.

The features and severity of infection are largely determined by the health status of the patient, and the nature of the wound and infecting organism.

Signs of infection which develop in a patient with an intravenous catheter should be assumed to be related to the catheter until proven otherwise.

This disease is endemic or potentially endemic to all countries.

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Infectious mononucleosis or EBV infection

Agent	VIRUS - DNA. Herpesviridae. Gammaherpesvirinae, Lymphocryptovirus: Human herpesvirus 4 (Epstein Barr virus)
Reservoir	Human
Vector	None
Vehicle	Saliva Blood transfusion
Incubation Period	28d - 42d
Diagnostic Tests	Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Exudative pharyngitis, symmetrical cervical lymphadenopathy, splenomegaly and hepatic dysfunction; atypical lymphocytes and positive serology appear after 10 to 14 days; acute illness resolves in 2 to 3 weeks, but malaise and weakness may persist for months.
Synonyms	EBV, EBV, Epstein-Barr, Febbre ghiandolare, Glandular fever, Infectious mononucleosis, Monocytic angina, Mononucleose, Mononucleosi, Mononucleosis - infectious, Mononukleose, Pfeiffer's disease. ICD9: 075 ICD10: B27.0

Clinical

Symptoms of Infectious Mononucleosis (IM) usually consist of fever, pharyngitis, and lymphadenopathy. [1]
• Patients usually do not recall a history of possible exposure.
• A prodrome consisting of 1 to 2 weeks of fatigue, malaise, and myalgia is common; however, abrupt presentations may occur.
• A low-grade fever is usually present and lasts for 1 to 2 weeks, occasionally up to 5 weeks.
• CMV / EBV co-infection may be associated with prolonged illness. [2]

Pharyngitis may be severe, particularly during the first week of illness. [3]
• Tonsillitis may be present, and lymphadenopathy is almost universal, lasting for 1 to 2 weeks.
• Posterior cervical nodes are often affected, and generalized adenopathy may occur.
• Periorbital edema and palatal petechiae are often present.
• Splenomegaly is found in most cases, and hepatomegaly in 25%.
• Asymptomatic pericardial effusions are common. [4]
• Patients often complain of headache.
• A morbilliform or papular erythematous eruption of the upper extremities or trunk is noted in 5% of cases.
• Lemmiere's syndrome has been reported as a complication of infectious mononucleosis. [5]
• Guillain-Barre syndrome and membranous glomerulonephritis have been reported following primary EBV infection. [6]

It is of note that a macular erythematous rash may occur in patients treated with ampicillin, usually appearing 5 to 9 days following the first dose.
• This phenomenon should not be misinterpreted as a penicillin allergy.
• Erythema nodosum and erythema multiforme have been associated with IM, as have petechiae and jaundice.
• The presence of severe abdominal pain may herald splenic rupture.

Other diseases ascribed to Epstein-Barr virus include nasopharyngeal carcinoma, Burkitt's lymphoma (African type) [7] , post-transfusion lymphoproliferative disorder (PTLD) [8] , hemophagocytic lymphohistiocytosis [9] and hemolytic anemia. [10]
• Epstein-Barr virus infection, like many other infectious diseases, is occasionally followed by Guillain-Barre syndrome.
• Gianotti-Crosti syndrome may be the only presenting manifestation of Epstein-Barr virus infection. [11]

A false positive serological reaction toward Epstein-Barr virus has been associated with a variety of conditions, including rheumatoid arthritis [12] [13] , Hepatitis E [14] , Hepatitis A [15] and Parvovirus B19 infection. [16]

This disease is endemic or potentially endemic to all countries.

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Infectious mononucleosis or EBV infection in Brazil

Prevalence surveys:

0.5% of CSF specimens from cases of suspected viral CNS infection (Sao Paulo, 2006 publication) [17]
2% of viruses associated with lympho-monocytic meningitis cases in Curitiba (2005 to 2006) [18]
Brazil - 12.7% of patients with fever and rash in Campinas (2003 to 2004) [19]

Seroprevalence surveys:

88.9% of urban children (2011 publication) [20]
95.8% of liver transplant recipients (1997 to 2007) [21]

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Influenza

Agent	VIRUS - RNA. Orthomyxoviridae, Orthomyxovirus: Influenza virus
Reservoir	Human Occasionally Ferret Bird Pig
Vector	None
Vehicle	Droplet
Incubation Period	1d - 3d
Diagnostic Tests	Viral culture (respiratory secretions). Serology. Nucleic acid amplification techniques are available.
Typical Adult Therapy	Respiratory precautions. Influenza A or B: Oseltamivir 75 mg PO BID X 5d OR Zanamavir 10 mg BID X 5 days
Typical Pediatric Therapy	Respiratory precautions. Influenza A or B: Oseltamivir 2 mg/kg (max 75 mg) PO BID X 5d OR Zanamavir (age > 5 years) 10 mg BID X 5 days
Vaccines	Influenza - inactivated Influenza - live
Clinical Hints	Myalgia, headache, cough, fever; pharyngitis and conjunctivitis often present; usually encountered in the setting of an outbreak; leucocytosis, chest pain and lobar infiltrate herald bacterial (pneumococcal or staphylococcal) pneumonia.
Synonyms	Asian flu, Aviaire influenza, Avian flu, Avian influenza, Bird flu, Epidemic catarrh, Grippe, H1N1, H2N2, H3N2, H5N1, Hong Kong flu, LPAI, Spanish influenza, Swine flu, Swine influenza. ICD9: 487 ICD10: J09,J10,J11

Clinical

Influenza is characterized by acute onset of fever, headache, myalgia, nonproductive cough, sore throat, and rhinitis. [1]
• The illness usually resolves in 2 to 7 days; however, symptoms often persist for up to two weeks.
• Severe illness or death may complicate the acute infection, notably in pregnant women [2] , the elderly and patients with underlying medical conditions. [3]
• Complications include primary viral pneumonia or bacterial pneumonia (most commonly pneumococcal) [4] ; myocarditis, myositis, Guillain-Barre syndrome [5] , encephalitis [6] , Gianotti-Crosti syndrome [7] and transverse myelitis. [8-10]

WHO Case definition for surveillance • Influenza:
Clinical case definition
A person with sudden onset of fever of >38°C and cough or sore throat in the absence of other diagnoses.
Laboratory criteria for diagnosis
• Virus isolation: Swab or aspirate from the suspected individual, or
• Direct detection of influenza viral antigen.
• Serology: Fourfold rise in antibody titer between early and late serum.
Case classification
• Suspected: A case that meets the clinical case definition.
• Confirmed: A case that meets the clinical case definition and is laboratory-confirmed (used mainly in epidemiological investigation rather than surveillance).

WHO definition for surveillance • Swine influenza (H1H1):
confirmed case • person with swine influenza A (H1N1) virus infection laboratory confirmed by
• real-time RT-PCR and/or
• viral culture and/or
• 4-fold rise in swine influenza A(H1N1) virus specific neutralizing antibodies
probable case • either
• person with influenza test positive for influenza A, but unsubtypable by reagents used to detect seasonal influenza virus infection , or
• person with clinically compatible illness or who died of unexplained acute respiratory illness who is considered to be epidemiologically linked to probable or confirmed case

CDC definition for surveillance • Swine influenza (H1H1):
confirmed case • person with acute respiratory illness with swine influenza A (H1N1) virus infection laboratory confirmed at

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CDC by
• real-time reverse transcriptase polymerase chain reaction (RT-PCR) and/or
• viral culture
probable case • person with acute febrile respiratory illness who is
• positive for influenza A, but negative for H1 and H3 by influenza RT-PCR
• positive for influenza A by influenza rapid test or influenza immunofluorescence assay (IFA) plus meets criteria for suspected case
suspected case • person with acute respiratory illness (defined as recent onset of >= 2 of rhinorrhea or nasal congestion, sore throat, or cough) plus
• close contact to confirmed case of swine influenza A (H1N1) virus infection during case's infectious period, or
• close contact defined as within about 6 feet of ill person
• infectious period defined as 1 day prior to illness onset to 7 days after onset
• travel to or residence in area with confirmed cases of swine influenza A (H1N1) virus infection

Avian influenza H5N1 infection:

Avian influenza H5N1 infection is characterized by fever greater than 38 C, shortness of breath and cough. [11] [12]
• The incubation period is 2 to 4 days.
• All patients reported to date have presented with significant lymphopenia and marked chest radiograph abnormalities consisting of diffuse, multifocal or patchy infiltrates.
• Some cases showed segmental or lobular consolidation with air bronchograms.
• Crackles were frequently heard on auscultation.
• Some of the patients reported sore throat, conjunctivitis, myalgia, rash or rhinorrhea.
• Watery diarrhea or loose stools was noted in approximately 50% of the cases.
• Myocardial dysfunction and hepatic dysfunction are also reported.
• Reactive hemophagocytic syndrome is the most characteristic pathological finding and may contribute to the lymphopenia, liver dysfunction, and abnormal clotting profiles observed among patients with severe infection.
• Approximately 90% of patients with H5N1 infection have been below age 40. [13]
• Approximately 60% of patients have died, on an average of 10 days after onset of symptoms.

Influenza virus H1N1 infection

• During the "Spanish flu" H1N1 pandemic of 1918 to 1919, illness was characterized by unusual severity, tendency to affect young healthy adults, rapid progression and overwhelming pneumonia.
• During the outbreak of A(H1N1)pdm09 virus outbreak of 2009 to 2010 [14] [15] , children [16] [17] and young adults accounted for a large proportion of cases. [18] [19] Severe cases were not necessarily associated with underlying disease. Obesity [20-29] , immune-compromise [30] (but not necessarily AIDS [31]) , pregnancy [32-53] , infection while hospitalized [54] , preexisting neurological disorders [55] , sickle cell disease [56] and asthma were identified as risk factors for complications. [57-63] Children below age 5 years, particularly those with neuro-developmental disorders, were also found to be at risk. [64-66]
• Most deaths were caused by primary viral pneumonia [67-77] , and bacterial co-infection was identified in as many as 29% of fatal cases. [78-83]
• Vomiting and diarrhea were reported in up to 25% of patients [84] , and as many as 6% were afebrile. [85] Case-fatality rates were not necessarily higher than those reported for other strains of Influenza virus. [86] [87]
• Additional complications included myopathy or rhabdomyolysis [88-94] , encephalitis or encephalopathy [95-120] , ischemic stroke [121] , aseptic meningitis [122] , acute disseminated or hemorrhagic leukoencephalitis [123-132] , deafness [133] , cerebellitis [134] , acute myelopathy [135] , Guillain-Barre syndrome [136-139] , parkinsonism [140] , narcolepsy [141] [142] , quadriplegia [143] , glomerulonephritis [144] [145] , tubulointerstitial nephritis [146] , renal failure [147-156] , hemolytic-uremic syndrome [157-161] , reactive thrombocytosis [162] , hemophagocytic lymphohistiocytosis [163-168] , myopathy [169] , cold agglutinin syndrome [170] , autoimmune [171] and thrombotic thrombocytopenic purpura [172] , myocarditis [173-193] or reversible myocardial dysfunction [194-196] , pericarditis [197] [198] , subacute thyroiditis [199] , rash [200] , pancreatitis [201] , vascular thrombosis [202] , plastic bronchitis [203] , hemorrhagic pneumonia [204] and Acute Respiratory Distress Syndrome (ARDS). [205-219]
• In some cases, the clinical features of leptospirosis suggested a diagnosis of H1N1 influenza. [220]

This disease is endemic or potentially endemic to all countries.

Influenza in Brazil

GIDEON does not follow routine country reports on human Influenza, since the scope and nature of these data are often diffuse, sporadic or inconsistent. See the "Worldwide" note for material regarding pandemic influenza, influenza vaccine, avian influenza in humans and other relevant subjects.

During the 1918 pandemic of H1N1 influenza, 116,771 cases (22.3% of the population) were reported in Sao Paulo, with 5,331 deaths. [221-223]

Notable outbreaks:

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2009 to 2010 - An outbreak (2,125 fatal cases) was reported. [224-234] ***Context:*** A pandemic of H1N1 Influenza virus [A (H1N1) pdm09 [235] infection occurred. [236-333] Over 600,000 cases had been officially-reported worldwide as of March, 2010. [334-336] 18,449 fatal cases were reported to August 1, 2010 (true number for first 12 months estimated at 293,500 [337] [338]). [339-363] Indigenous populations from Australia, Canada, the United States and New Zealand were found to have at least a 3-fold greater death rate than others in their countries. [364-383] Reporting of case-number summaries was suspended by WHO as of July 6 [384] ; and on August 10, the pandemic was declared to have ended. [385] [386] The pandemic began in Mexico, spreading rapidly to the United States and Canada. Swine were not implicated in the transmission of disease. [387-389] Human-to-swine transmission was confirmed in Argentina [390] , Cambodia [391] [392] , Vietnam [393] [394] , Italy [395] and Canada during the outbreak [396-407] ; and infected swine were identified in Argentina [408-410] , Australia [411-413] , Brazil [414] , Cameroon [415] [416] , China [417-420] , Denmark [421] , Finland [422] , Germany [423] [424] , Iceland [425] , India [426] , Indonesia [427] , Ireland [428] , Italy [429] [430] , Japan [431] [432] , England [433] , Mexico [434] , Northern Ireland [435] , Norway [436] [437] , Republic of Korea [438] [439] , Reunion Island [440] , Russian Federation [441] , Scotland [442] , Taiwan [443] , Thailand [444] [445] , the United Kingdom [446] [447] and the United States. [448-456] Infected turkeys were subsequently identified in Canada [457-459] , Chile [460-464] , France [465] the United Kingdom [466] [467] and the United States. [468] [469] Infection was reported in cats [470-482] , ferrets [483-488] , a dog [489] , a badger (*Taxidea taxus*) , a captive Bornean binturong (*Arctictis binturong penicillata*) [490] [491] and a cheetah [492] [493] in the United States [494-500] ; skunks in Canada [501] ; dogs in Italy [502] and China [503] ; farmed American mink ((*Neovison vison*) in the Netherlands [504] ; and in dogs and swine in Hong Kong. [505]

- Reporting dates vary by country. The following updates include incidence data as of December 31, 2010. [506] [507] : Afghanistan (17 fatal) [508] [509] , Albania (6 fatal), Algeria (57 fatal cases), American Samoa (94 - 0 fatal), Andorra (1), Angola (37) [510] , Anguilla (14), Antigua and Barbuda (0 fatal), Argentina (626 fatal) [511-532] , Armenia (3 fatal), Aruba (13), Australia (51,170 - 195 fatal) [533-579] , Austria (24 fatal) [580-582] , Azerbaijan (2), Bahamas (4 fatal), Bahrain (7 fatal), Bangladesh (7 fatal) [583] , Barbados (157 - 3 fatal) [584] , Belarus (20 fatal) [585] , Belgium (17 fatal) [586-589] , Belize (60), Bermuda (1 fatal), Bhutan (487) [590] , Bolivia (59 fatal) [591] , Bosnia and Herzegovina (10 fatal), Botswana (23), Brazil (2,125 fatal) [592-602] , British Virgin Islands (25), Brunei (850 - 1 fatal), Bulgaria (40 fatal) , Burundi (7), Cambodia (6 fatal) [603-605] , Cameroon (4) [606] , Canada (429 fatal) [607-638] , Cape Verde (118), Cayman Islands (130 - 1 fatal), Chad (1), Chile (156 fatal) [639-654] , Central African Republic [655] , China (724 fatal - including 56 in Hong Kong [656-680] and 2 in Macao) [681-736] , Colombia (254 fatal) [737] , Comoros (2 fatal in Mayotte) [738] , Congo (21) [739] , Cook Islands (106 - 1 fatal), Costa Rica (65 fatal), Croatia (25 fatal), Cuba (1,805 - 83 fatal) [740] , Cyprus (6 fatal) [741] , Czech Republic (98 fatal), Democratic Republic of Congo (222) [742] [743] , Democratic Republic of Korea (9) [744] , Denmark (32 fatal) [745-749] , Dominica (51), Dominican Republic (464 - 24 fatal), Ecuador (130 fatal) [750] , Egypt (281 fatal) [751-754] , El Salvador (34 fatal) [755] , Estonia (19 fatal), Ethiopia (12), Falkland Islands (7), Fiji (268 - 0 fatal), Finland (43 fatal) [756-759] , France (349 fatal) [760-792] , French Guiana (29 - 1 fatal) [793] [794] , French Polynesia (185 - 7 fatal) [795] [796] , Gabon (4), Georgia (20 fatal), Germany (253 fatal) [797-815] , Ghana (1 fatal) [816] , Gibraltar (16), Greece (141 fatal) [817-826] , Grenada (28), Guadeloupe (5 fatal) [827] , Guam (341 - 2 fatal) [828] , Guatemala (26 fatal) [829] [830] , Guyana (30), Haiti (95) [831] [832] , Honduras (18 fatal), Hong Kong (56 fatal) [833-857] , Hungary (133 fatal) [858] , Iceland (2 fatal) [859] [860] , India (44,958 - 2,703 fatal) [861-878] , Indonesia (691 - 10 fatal) [879] [880] , Iran (147 fatal) [881-889] , Iraq (42 fatal) [890] , Ireland (24 fatal) [891-893] , Israel (113 fatal, including 28 in Gaza and the West Bank) [894-907] , Italy (256 fatal) [908-920] , Ivory Coast (5) [921] , Jamaica (7 fatal), Japan (198 fatal) [922-950] , Jordan (19 fatal) [951] , Kazakhstan (17) [952] , Kenya (417) [953-956] , Kiribati (4 - 0 fatal), Kuwait (30 fatal), Laos (156 - 1 fatal) [957-959] , Latvia (34 fatal), Lebanon (5 fatal) [960] [961] , Lesotho (65), Libya (1 fatal), Liechtenstein (5), Lithuania (23 fatal) [962] , Luxembourg (3 fatal) [963] , Macao (2 fatal) [964] , Macedonia (23 fatal), Madagascar (3 fatal) [965-970] , Malaysia (1,780 - 77 fatal) [971-978] , Malawi (4), Maldives (1 fatal), Mali (12) [979] , Malta (5 fatal), Marshall Islands (115 - 1 fatal), Martinique (44 - 1 fatal) [980] , Mauritania (15), Mauritius (8 fatal), Mexico (1,316 fatal) [981-1023] , Micronesia (82 - 0 fatal), Moldova (35 fatal) [1024] , Monaco (1), Mongolia (29 fatal) [1025] [1026] , Montenegro (7 fatal), Montserrat (21), Morocco (64 fatal) [1027-1029] , Mozambique (2 fatal), Myanmar (137) [1030-1035] , Namibia (1 fatal), Nauru (8 - 0 fatal), Nepal (172 - 3 fatal) [1036-1039] , The Netherlands (62 fatal) [1040-1053] , Netherlands Antilles (128 cases - 59 in Curacao, including 3 on a cruise ship; 29 in St. Maarten and 38 on Bonaire), New Caledonia (508 - 7 fatal), New Zealand (4,974 - 51 fatal) [1054-1080] , Nicaragua (2,152 cases - 11 fatal) [1081] , Niger (12), Nigeria (2 fatal) [1082] [1083] , Niue (0), Northern Marianas (6 - 0 fatal), Norway (12,513 cases, 29 fatal) [1084-1087] , Oman (31 fatal) [1088] [1089] , Pakistan (14 fatal) [1090-1093] , Palau (47 - 0 fatal), Panama (12 fatal) [1094] [1095] , Papua New Guinea (29 - 0 fatal), Paraguay (47 fatal), Peru (238 fatal) [1096-1100] , Philippines (3,207 - 30 fatal), Pitcairn Island (0), Poland (148 fatal) [1101] [1102] , Portugal (83 fatal) [1103-1106] , Puerto Rico (20), Qatar (8 fatal) [1107] , Republic of Korea (171 fatal) [1108-1121] , Reunion (12 fatal) [1122-1136] , Romania (122 fatal) [1137] , Russian Federation (19 fatal) [1138-1147] , Rwanda (494 - 0 fatal) [1148-1150] , Saint Kitts and Nevis (2 fatal), Saint Lucia (1 fatal), Saint Vincent and the Grenadines (19), Samoa (173 - 2 fatal), Sao Tome and Principe (2 fatal), Saudi Arabia (124 fatal) [1151-1166] , Scotland (38 fatal) [1167-1180] , Senegal (184), Serbia (71 fatal) [1181] [1182] , Seychelles (33), Singapore (19 fatal) [1183-1203] , Slovakia (53 fatal), Slovenia (19 fatal) [1204] , Solomon Islands (4 - 0 fatal), South Africa (93 fatal) [1205-1209] , Spain (271 fatal) [1210-1247] , Sri Lanka (48 fatal), Sudan (5 fatal), Suriname (144 - 2 fatal) [1248] , Swaziland (2), Sweden (25 fatal) [1249-1252] , Switzerland (18 fatal) [1253-1256] , Syria (127 fatal) [1257] [1258] , Taiwan (44 fatal) [1259-1272] , Tanzania (1 fatal), Thailand (212 fatal) [1273-1291] , Tokelau (0), Tonga (20 - 1 fatal), Trinidad and Tobago (5 fatal), Tunisia

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(21 fatal), Turkey (627 fatal) [1292-1301] , Turks and Caicos Islands (45), Tuvalu (23 - 0 confirmed) [1302] , Uganda (263), Ukraine (282 fatal) [1303-1312] , United Arab Emirates (6 fatal) [1313-1316] , United Kingdom (474 fatal: at least 142 in England, 38 in Scotland - including the first fatal case in Europe [1317-1319] , 21 in Wales and 13 in Northern Ireland) [1320-1369] , United States (2,718 fatal) [1370-1446] , Uruguay (20 fatal), Vanuatu (3 - 0 fatal), Venezuela (137 fatal) [1447] [1448] , Vietnam (53 fatal) [1449-1455] , Virgin Islands, U.S. (49), Wallis and Futuna (55 - 0 fatal) [1456] , Yemen (28 fatal) , Zambia (90) [1457] and Zimbabwe (41). [1458-1516]

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Intestinal spirochetosis

Agent	BACTERIUM. Brachyspira pilosicoli and B. aalborgi Anaerobic gram-negative spirochetes
Reservoir	Human Fowl Pigs
Vector	None
Vehicle	Endogenous
Incubation Period	Unknown
Diagnostic Tests	Spirochetes resemble "brush border' on bowel biopsy; identification of Brachyspira by PCR
Typical Adult Therapy	Metronidazole appears to be effective in some cases.
Typical Pediatric Therapy	As for adult.
Clinical Hints	Chronic diarrhea and abdominal pain in the absence of other identifiable etiology
Synonyms	Human intestinal spirochetosis. ICD9: 009.1 ICD10: A04.8

Clinical

This diagnosis should be suspected in patients with persistent or chronic diarrhea lasting more than several weeks, in whom alternative etiologies are not identified.

- Abdominal pain, hematochezia, flatulence and intermittent constipation are also reported in some cases. [1-3]
- *Brachyspira* has been identified in the blood in some cases [4] , even in the absence of intestinal disease. [5]
- Asymptomatic infection is common. [6]
- Intestinal spirochetosis in children may mimic inflammatory bowel disease. [7]
- Although some patients improve following administration of Metronidazole, other cases resolve without specific therapy. [8]

Roentgenographic studies may reveal colonic mucosal edema and luminal narrowing. [9]

Standard H & E staining of colonic biopsies reveals a 'pseudo-brush border' consisting of tiny spirochetes [10-12] ; or free-floating spirochetes in the intestinal mucus. [13]

- Similar findings are often present in asymptomatic individuals. [14]
- The organism can be identified using specialized culture [15] or molecular methods. [16-18]

This disease is endemic or potentially endemic to all countries.

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Intra-abdominal abscess

Agent	BACTERIUM. Mixed anaerobic / aerobic, staphylococci, Neisseria gonorrhoeae, Chlamydia trachomatis, etc
Reservoir	Human
Vector	None
Vehicle	None
Incubation Period	Variable
Diagnostic Tests	Various imaging techniques (CT, Gallium scan, ultrasound, etc).
Typical Adult Therapy	Percutaneous or open drainage + antibiotics directed at known or suspected pathogen(s)
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, chills and localizing pain (e.g., chest pain in subphrenic abscess) - setting of prior surgery, biliary or colonic disease, appendicitis, vaginal discharge (PID); FUO, subdiaphragmatic gas or limited diaphragmatic motion may be present.
Synonyms	Abscess - Abdominal, Acute appendicitis, Appendicitis, Intraabdominal abscess, Intraperitoneal abscess, P.I.D., Pancreatic abscess, Pelvic abscess, Pelvic inflammatory disease, Pylephlebitis, Subhepatic abscess, Subphrenic abscess, Suppurative pancreatitis, Tuboovarian abscess. ICD9: 614,577.0 ICD10: K35,N73,K75.1,K85

Clinical

Intraabdominal abscesses often occur in the setting of prior abdominal trauma, surgery or infection.

Signs and symptoms may include fever, pain, tenderness and leucocytosis.
• In many cases, the sole presenting feature is prolonged fever, which may be accompanied by weight loss, lethargy and anemia.
• One or more localized masses may be detectable on palpation or through the use of imaging techniques.

Comprehensive reviews of clinical presentation:
• Pelvic Inflammatory Disease [1-6]
• Splenic Abscess [7] [8]
• Pancreatic Abscess [9] [10]
• Pylephlebitis. [11]

This disease is endemic or potentially endemic to all countries.

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Intracranial venous thrombosis

Agent	BACTERIUM. Oral anaerobes, streptococci, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture (blood, CSF if indicated). Ophthalmoscopy. Roentgenographic studies of skull & sinuses.
Typical Adult Therapy	Antibiotic(s) directed at known or suspected pathogens
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, seizures and fever; cranial nerve dysfunction may be present; usually occurs in the setting of facial, otic or sinus infection.
Synonyms	Cavernous sinus thrombosis, Cerebral sinus thrombosis, Cortical vein thrombosis, Internal cerebral vein thrombosis, Straight sinus thrombosis, Superior sinus thromobosis, Transverse sinus thrombosis. ICD9: 325 ICD10: G08

Clinical

Cortical vein thrombosis may occasionally be clinically silent, or produce only transient neurological findings. [1]
• Septic cortical vein or venous sinus occlusion may progress to subdural empyema, meningitis, brain abscess, systemic infection or pulmonary embolism.
• Severe headache is present in 90% of cases, and cerebral lesions with neurological signs in 50%. [2]
• If collateral flow is compromised, the resulting neurological may mimic brain abscess, with impairment of consciousness, focal or generalized seizures, and increased intracranial pressure.
• Depending on the site of the lesion, one may encounter hemiparesis, which involves the face and hand if veins; unilateral or bilateral leg weakness; aphasia; etc. [3]

Cavernous sinus thrombosis is characterized by diplopia, photophobia, orbital edema, and progressive exophthalmos.
• Involvement of cranial nerves III, IV, V, and VI is reflected by ophthalmoplegia, fixed pupil, a loss of the corneal reflex and diminished upper facial.
• Papilledema, retinal hemorrhages, and visual loss may also occur.

Anterior superior sagittal sinus thrombosis may produce intracranial hypertension without other signs.
• More extensive blockage of this sinus is associated with bilateral leg weakness followed by arm weakness and clouding of consciousness.

Lateral sinus thrombosis causes pain over the ear and mastoid, occasionally with edema over the mastoid itself (Griesinger's sign); or ipsilateral facial pain and lateral rectus weakness (Gradenigo's syndrome).

This disease is endemic or potentially endemic to all countries.

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Isosporiasis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Eimeriida: Isospora [Cystoisospora] belli
Reservoir	Human
Vector	None
Vehicle	Food Liquids Fecal-oral Sexual (homosexual) contact
Incubation Period	7d - 10d
Diagnostic Tests	Microscopy of stool or duodenal contents. Advise laboratory when this organism is suspected.
Typical Adult Therapy	Sulfamethoxazole/trimethoprim 800/160 mg BID X 10 days - Then BID X 3 weeks (may be indefinite in AIDS patient) Increase dosage / duration in immune-suppressed patients Pyrimethamine 50 to 75 mg per day + leucovorin if allergic to sulfa
Typical Pediatric Therapy	Sulfamethoxazole/trimethoprim 25/5 mg/kg BID X 10 days - Then BID X 3 weeks
Clinical Hints	Myalgia, watery diarrhea, nausea and leukocytosis; eosinophilia may be present; prolonged and severe in AIDS patients.
Synonyms	Isospora belli. ICD9: 007.2 ICD10: A07.3

Clinical

Isosporiasis is characterized by abdominal cramps, watery diarrhea, headache, weight loss and myalgias. [1]
- Fever and vomiting may also be present.
- A low-grade eosinophilia is present in 50% of patients
- Fecal leucocytes are not seen.

Infection in AIDS patients may cause significant weight loss and dehydration, requiring hospitalization. [2]
- Disease is also more severe among patients with lymphoma and leukemia. [3]
- Chronic and severe infection may occasionally affect immunocompetent patients as well, and infants and young children are most likely to suffer severe disease. [4]
- Paralysis related to severe potassium depletion has been reported in an AIDS patient with isosporiasis. [5]
- Biliary disease similar to primary sclerosing cholangitis has been reported. [6]
- Disseminated extraintestinal infection has rarely been reported.

This disease is endemic or potentially endemic to all countries.

Isosporiasis in Brazil

Prevalence surveys:

7% of AIDS-associated diarrhea (2005 publication) [7]
2% of AIDS-associated diarrhea (1999 publication) [8]
10.1% of AIDS-associated diarrhea (1989 publication) [9]
11.1% of fecal samples from HIV/AIDS patients in the Triangulo Mineiro region (1993 to 2003) [10]
4.2% of HIV-positive patients in northeastern Sao Paulo (1998 to 2008) [11]

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Kawasaki disease

Agent	UNKNOWN
Reservoir	Unknown
Vector	None
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Diagnosis is based on clinical criteria only.
Typical Adult Therapy	Intravenous gamma globulin 2.0 g/kg over 10 to 12h X 1 dose. Plus aspirin 100 mg/kg/day X 14d (or until defervescence) - then 5 to 10 mg/kg/day until normal ESR Infliximab 5 mg/kg has been successful in some studies.
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, conjunctivitis, stomatitis, erythematous rash which desquamates; occasional coronary artery occlusion; the disease is most common among children; case-fatality rates of 1% to 4% are reported.
Synonyms	Kawasaki's disease, Mucocutaneous lymph node syndrome. ICD9: 446.1 ICD10: M30.3

Clinical

Diagnostic criteria: [1] [2]

Fever for at least five days in addition to at least 4 of the following:

1. Changes in the oral mucosa (erythema, strawberry tongue, etc)
2. Changes in hands and feet (erythema, swelling, periungual desquamation, rarely gangrene [3])
3. Rash, primarily on trunk (maculopapular, scarlatiniform, erythema multiforme).
4. Cervical lymphadenopathy [4]
5. Absence of other etiology.

Kawasaki disease is encountered among adults [5-7] as well as children.
• The incidence of specific diagnostic criteria are roughly similar in both groups
• Cheilitis, meningitis, and thrombocytosis are more common in children. Rare instances of thrombocytopenia are also reported [8]
• Arthralgia is common, and may involve one or multiple joints [9]
• Arthralgia, adenopathy, and liver function abnormality [10] [11] are more common in adults. [12]
• Older children may have a more marked inflammatory response and worse outcome, as compared to young children. [13]
• Absence of fever [14] , acute hepatitis [15] , pleural effusion, disseminated intravascular coagulopathy [16] , pancreatitis [17] [18] and cholestasis have been reported in some cases. [19]
• Rare instances of recurrent Kawasaki disease have been reported. [20] [21]

There is no diagnostic test for Kawasaki disease.

Atypical or Incomplete Kawasaki Disease:

As many as 23% of patients may present with "incomplete (atypical) Kawasaki disease" characterized by fever >=5 days and the presence of <4 "classic signs." [22-25]
• The clinical picture in atypical Kawasaki disease may be dominated by one unusual finding: seizure, bloody diarrhea, nephrotic syndrome, hyponatremia or compressive cervical lymphadenopathy. [26]
• Of 232,263 cases reported in Japan during 2007 to 2008, 80% had classic clinical findings and 20% had "incomplete" Kawasaki disease. [27]
• Occasionally, the initial presentation of Kawasaki disease may be limited to erythema multiforme [28] or fever with cervical lymphadenopathy. [29]
• Patients with incomplete and atypical KS are more likely than those with other febrile diseases to present with mucosal changes, conjunctivitis, extremity abnormalities, perineal desquamation [30] , and later development of coronary artery abnormalities. [31]

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The appearance of redness or crusting at a BCG inoculation site is a valuable predictive sign for Kawasaki disease. [32]
• Orange-brown discoloration of nails (chromonychia) is a common finding in some series. [33]

Additional findings:
Infants below age 1 year have a relatively high incidence of cardiac involvement. [34]
• Cardiac involvement is present in 13.6% of cases (Japan, 2003 to 2004) [35]
• Coronary arteritis is common, and coronary artery aneurysms may rupture [36] [37] or persist into adulthood. [38-41]
• Meningoencephalitis, often with seizures, has been reported as a presenting feature of Kawasaki disease. [42] [43]
• Additional complications include oculomotor [44] or facial palsy [45], parotitis [46], large pleural effusions [47], retropharyngeal mass [48], cholestatic jaundice [49-51], colitis [52], nephrotic syndrome [53], sterile pyuria [54], sensorineural hearing loss [55] [56] and peripheral vascular gangrene [57] and necrotic lesions on the face. [58]
• 7% of affected children develop Kawasaki disease shock syndrome, with decreased systolic blood pressure or evidence of hypoperfusion. The shock syndrome is characterized by an increased rate of echocardiographic abnormalities and is less likely to respond to IVIG therapy [59] [60]
• Neutrophilia, anemia, thrombocytosis, hemophagocytic lymphohistiocytosis [61] [62], hepatic dysfunction [63] and sterile pyuria [64] are common. Syndrome of inappropriate ADH secretion has been reported. [65]

Diseases which may mimic Kawasaki disease include Chikungunya [66], meningococcal septicemia [67], Takayasu's arteritis [68], systemic onset juvenile idiopathic arthritis [69] and Q fever. [70]

This disease is endemic or potentially endemic to all countries.

Kawasaki disease in Brazil

70 cases were reported from a tertiary hospital in Brasilia during 2002 to 2007. [71]

A series of 125 cases was reported by two hospitals in Rio de Janeiro (2010 publication) [72]

115 cases were reported from a hospital in Federal district during 2002 to 2009. [73]

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Kikuchi's disease and Kimura disease

Agent	UNKNOWN
Reservoir	Unknown
Vector	None
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Biopsy.
Typical Adult Therapy	Supportive Hydroxychloroquine and corticosteroids have been successful for Kikuchi's disease in some cases.
Typical Pediatric Therapy	As for adult
Clinical Hints	Most patients of Asian origin. Kikuchi disease: prolonged (1 to 12 months) cervical lymphadenopathy (rubbery, non-matted - may be tender), fever (40%), weight loss, 'sweats', leukopenia. Salivary gland involvement, glomerulitis, painless subcutaneous masses and eosinophilia suggest Kimura disease.
Synonyms	Angiolymphoid hyperplasia, Angiolymphoid hyperplasia-eosinophia, Eosinophilic follicular lymphadenitis, Histiocytic necrotizing lymphadenitis, Kikuchi and Fujimato's disease, Kikuchi's disease, Kimura disease. ICD9: 289.3 ICD10: I89.8

Clinical

Kikuchi's disease:
Kikuchi's disease (histiocytic necrotizing lymphadenitis) is characterized by histiocytic necrotizing lymphadenitis, usually of the cervical region [1] [2] ; however, other anatomic regions may be involved. [3]
• Generalized lymphadenopathy is occasionally encountered [4]
• The disease is primarily seen in young Japanese women or women of Oriental descent in the third decade of life. [5]
• Pediatric [6] , male and elderly patients are occasionally encountered. [7]
• Leukopenia is present in 50% of cases, and atypical lymphocytes may be seen in the peripheral blood smear.
• Additional features may include aseptic meningitis [8] [9] , maculopapular or urticarial rash [10] , arthralgia, myalgia, hepatosplenomegaly, hepatic dysfunction, neuropathy, venous thrombosis [11] , orbital pseudotumor [12] , pericarditis, pulmonary infiltrates with pleural effusion [13] and pulmonary hemorrhage.
• Biopsy material reveals paracortical hyperplasia without granulocytic infiltration and a typical 'starry sky' pattern. [14] [15]
• Clinical features may mimic those of lupus erythematosus [16] , tuberculous meningitis [17] , or lymphoma. [18-21]
• The prognosis is good, and patients recover after a mean of 3 months.
• Relapse occurs in 20% of cases [22] and recurrence in 3% to 4%. [23]
• Hydroxychloroquine and corticosteroids have been advocated by some authorities.

Kimura disease:
Kimura disease (angiolymphoid hyperplasia with eosinophiles (eosinophilic follicular lymphadenitis) is also most common among Oriental males. [24]
• Most present as painless subcutaneous masses and lymphadenopathy of the cervical region.
• Cases of isolated Kimura disease of the earlobe [25] and eyelid have been reported [26] [27]
• In contrast to Kikuchi's disease, salivary gland involvement [28] [29] , glomerulitis, nephrotic syndrome [30] , elevated IgE and eosinophilia are often encountered.
• Hypercoagulability has been reported in some cases [31]

Angiolymphoid hyperplasia with eosinophilia is clinically similar to Kimura disease, but is histologically distinct from the latter. [32-38]
• The condition is characterized by characterized by reddish-brown nodules and plaques in the dermis and the subcutaneous tissues, typically occurring on the neck and head. [39]

This disease is endemic or potentially endemic to all countries.

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Kikuchi's disease and Kimura disease in Brazil

Sporadic cases of Kikuchi disease [40-44] and Kimura disease are reported. [45-47]

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Kingella infection

Agent	BACTERIUM. Kingella kingae, et al A facultative gram-negative coccobacillus
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Unknown
Diagnostic Tests	Culture of blood, joint fluid, CSF, etc. Alert laboratory if these organisms are suspected.
Typical Adult Therapy	Penicillin G or Penicillin V usually effective - dosage per severity/site
Typical Pediatric Therapy	As for adult
Clinical Hints	A relatively rare cause of septic arthritis, endocarditis, meningitis and other infections; most infections have been in young children.
Synonyms	

Clinical

Kingella kingae, *K. (Suttonella) indologenes*, *K. denitrificans* and *K. oralis* are found in the normal respiratory tract, and occasionally associated with bacteremia, bone and joint infection (notably in young children) [1] [2] and endocarditis (the 'K' in the HACEK group). [3]

• *Kingella potus* has been isolated from a kinkajou wound in a zookeeper. [4]

This disease is endemic or potentially endemic to all countries.

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Lagochilascariasis

Agent	PARASITE - Nematoda. Phasmidea: Lagochilascaris minor
Reservoir	Unknown
Vector	None
Vehicle	Ingestion of ova - details not known
Incubation Period	>30d
Diagnostic Tests	Identification of ova or adult parasites in tissue and exudates.
Typical Adult Therapy	No proven therapy. Albendazole has been used with some success
Typical Pediatric Therapy	As for adult
Clinical Hints	Tender subcutaneous mass (usually limited to scalp and neck, occasionally pharynx or paranasal sinuses) with suppuration and fistulae; eosinophilia may be present.
Synonyms	Lagochilascaris minor. ICD9: 128.8 ICD10: A81.8

Clinical

Lagochilascariasis is characterized by slowly-developing soft tissue masses of the head, neck, nasopharynx or tonsils. [1-3]
- Initially, the patients may complain of a sensation of 'something crawling in the back of the mouth."
- Lesions become fluctuant, forming sinuses and fistulae [4] which discharge pus which contains worms, larvae and eggs.
- Worms may also exit through the nose or mouth, or involve the paranasal sinuses [5] , and rarely the brain [6] or lungs. [7]

This disease is endemic or potentially endemic to 10 countries.

Lagochilascariasis in Brazil

Ten cases of lagochilascariasis had been reported in Brazil as of 1992 - from Sao Paulo State, Federal District and Para State. [8-13]

- A subsequent case report was published in 1993. [14]
- A case of human infection was reported in Para State (2000 publication). [15]

Ova of *Lagochilascaris* sp. were identified in soil samples from a public park in Pelotas city, Rio Grande do Sul State (2011 publication) [16]

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Laryngotracheobronchitis

Agent	VIRUS OR BACTERIUM. Parainfluenza virus, Influenza virus, Mycoplasma, et al
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	3d - 8d
Diagnostic Tests	Viral culture (respiratory secretions). Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Usually encountered in the setting of bronchiolitis, laryngitis or croup following a minor upper respiratory infection in young children.
Synonyms	Bronchitis, Croup, Laringitis, Laryngite, Laryngitis, Laryngotracheitis. ICD9: 464,466 ICD10: J04,J05,J20,J21

Clinical

Laryngotracheobronchitis is a self-defined syndrome consisting of hacking cough, often with an 'itching' or 'foreign body' sensation in the airways, and hoarseness. [1]
- Viral croup and epiglottitis are two major inflammatory causes of airway obstruction in children.
- Spasmodic croup and membranous laryngotracheobronchitis may be associated with obstruction. [2]

Bacterial tracheitis is an uncommon (>200 cases reported worldwide) severe condition usually affecting children that manifests as cough, stridor, mucopurulent tracheal secretions and lack of response to therapeutic modalities used for treating viral croup. [3]
- Fever may be low-grade or even absent.
- 75% of patients require intubation and mechanical ventilation.
- The case/fatality rate is approximately 2%.
- Causative pathogens include *Staphylococcus aureus* (50% of cases) and *S. pneumoniae*, *H. influenzae*, *M. catarrhalis* and *S. pyogenes*. Gram-negative bacilli are also reported in some cases.
- Occasionally, co-infection with viral croup agents is found.

This disease is endemic or potentially endemic to all countries.

Laryngotracheobronchitis in Brazil

Mycobacterium tuberculosis accounts for 41.7% of infectious granulomatous laryngitis cases, *Paracoccidioides braziliensis* 41.7%, *Leishmania brasiliensis* 16.7% (2007 publication) [4] (2007 publication) [5]

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Legionellosis

Agent	BACTERIUM. Legionella pneumophila, et al An aerobic gram-negative bacillus
Reservoir	Water
Vector	None
Vehicle	Water Aerosols
Incubation Period	5- 6d (range 2-12d); Pontiac fever = 1-2d
Diagnostic Tests	Serology. Culture. Urine antigen (certain types). Nucleic acid amplification. Alert lab if organism suspected.
Typical Adult Therapy	Fluoroquinolone (Levofloxacin, Trovafloxacin, Pefloxacin, Sparfloxacin or Moxifloxacin). OR Azithromycin. OR Erythromycin + Rifampin
Typical Pediatric Therapy	Azithromycin. OR Erythromycin + Rifampin
Clinical Hints	Respiratory illness with extrapulmonary manifestations (diarrhea, confusion, renal or hepatic dysfunction, relative bradycardia, etc.); most cases reported during summer in temperate areas; case-fatality rates of 5% to 25% are reported.
Synonyms	Doenca dos legionarios, Legionarsjuka, Legionarssjuka, Legionella, Legionellose, Legionellosi, Legionnaire's disease, Pontiac fever. ICD9: 482.84 ICD10: A48.1,A48.2

Clinical

WHO Case definition for surveillance:
Clinical description
An illness characterized by an acute lower respiratory infection with focal signs of pneumonia on clinical examination and/or radiological evidence of pneumonia.
Laboratory criteria for diagnosis
Presumptive: one or more of the following:
• Detection of specific *Legionella* antigen in respiratory secretions or urine
• Direct fluorescent antibody (DFA) staining of the organism in respiratory secretions or lung tissue, using evaluated monoclonal reagents
• A fourfold or greater rise in specific serum antibody titer to *Legionella* species other than *Legionella pneumophila* serogroup 1, using a locally validated serological test
Confirmative: one or more of the following:
• Isolation of *Legionella* from respiratory secretions, lung tissue, pleural fluid, or blood
• A fourfold or greater rise in specific serum antibody titer to *L. pneumophila* serogroup 1 by indirect immunofluorescence antibody test or microagglutination
• Most European countries and others such as the United States now include the detection of *L. pneumophila* serogroup 1 antigen in urine as a confirmatory test.
Case classification
• Suspected: Not applicable.
• Probable: A case compatible with the clinical description, with presumptive laboratory results.
• Confirmed: A case compatible with the clinical description, with confirmative laboratory results.

Pneumonia associated with extrapulmonary findings should suggest the possibility of Legionnaire's disease.
• Q-fever may be mistaken for Legionnaires' disease [1]
• The most common clinical manifestation is pneumonia, ranging from mild • to severe, with respiratory failure and death.
• Risk factors for overt disease include advanced age, smoking, chronic obstructive pulmonary disease, immunosuppression, and recent surgery.
• Person-to-person transmission has not been demonstrated.

Legionnaire's disease vs. Pontiac fever:
There are 2 currently recognized distinct clinicoepidemiological manifestations of legionellosis:
• Both forms are characterized initially by anorexia, vomiting, myalgia and headache, followed within a day by rising fevers and chills.
• Legionnaires. disease. (pneumonic form) and
• Pontiac fever (non-pneumonic Legionnaires disease)

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Legionnaires disease [2] [3]

• In the pneumonic form, non-productive cough, abdominal pain / diarrhea, confusion / delirium are common.
• It is not possible, clinically, to distinguish *Legionella* pneumonia from other pneumonias [4] ; suspicion should be raised in any pneumonia connected with epidemiological information (e.g., recent traveling, hospitalization, gatherings, immunosuppression).
• In addition, age (>50), sex (M), smoking, alcohol consumption have been shown to be risk factors.

Pontiac fever:

Pontiac fever is a self-limited, influenza-like illness lasting 2 to 5 days, often in healthy persons following exposure to contaminated whirlpools or spas. [5-7]
• Pontiac fever is not associated with pneumonia. It is thought to represent a reaction to inhaled antigen, rather than to bacteria.
• Proposed case definition for Pontiac fever include occurrence of at least one symptom among headache, myalgia, fever and rigors, beginning 2 to 8 days following exposure. [8]

Complications:

Reported complications of legionellosis have included empyema, pleural effusion, lung abscess, renal failure (in 10% to 50% of cases), endocarditis [9] [10] , peritonitis, cerebellar ataxia [11] , cutaneous and visceral abscesses [12] , arteriovenous fistula infection, pericarditis and myocarditis.
• Case-fatality rates may approach 40%, particularly among patients with underlying disease or immunosuppression.
• Additional risk factors for fatal infection include heart disease, malignancy, alcoholism and renal disease. [13]

This disease is endemic or potentially endemic to all countries.

Legionellosis in Brazil

Prevalence surveys:

5.1% of community-acquired pneumonia (Rio Grande do Sul, 2000 to 2001) [14]
25.7% of pneumonia cases on a renal transplant unit (Sao Paulo, 1988 to 1993) [15]

Notable outbreaks:

1989 to 1990 - An outbreak (8 cases) of legionellosis in a renal transplant unit in Sao Paulo was ascribed to a contaminated potable water system. [16-19]

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Leishmaniasis - cutaneous

Agent	PARASITE - Protozoa. Neozoa, Euglenozoa, Kinetoplastea. Flagellate: Leishmania tropica, et al
Reservoir	Human Hyrax Rodent Marsupial Dog Sloth Anteater Armadillo
Vector	Fly (sandfly = Phlebotomus for old world; Lutzomyia or Psychodopygus for new world)
Vehicle	None
Incubation Period	2w - 8w (range 1w - months)
Diagnostic Tests	Identification of organism on smear or specialized culture. Nucleic acid amplification
Typical Adult Therapy	Pentavalent antimonials 20 mg/kg/d IV or IM X 21d & / or topical paromomycin. Alternatives: L. major - Fluconazole or Azithromycin, PO L. mexicana or L. panamensis - Ketoconazole, PO L. braziliensis - Azithromycin, PO
Typical Pediatric Therapy	As for adult
Clinical Hints	Chronic ulcerating skin nodule; painless (*Leishmania tropica*) or painful (*L. major*); diffuse infection or regional lymphadenopathy occasionally encountered.
Synonyms	Aleppo button, Antep boil, Baghdad boil, Bay sore, Bejuco, Biskra button, Bolho, Bosjaws, Bush yaws, Busi-yasi, Chiclero ulcer, Cutaneous leishmaniasis, Delhi ulcer, Domal, El-Mohtafura, Forest yaws, Gafsa boil, Granuloma endemicum, Hashara, Jericho boil, Kaal Daana, Kandahar sore, Leishmania major, Leishmania tropica, Leishmaniasis, Leishmaniose: Kutane, Leishmaniosi cutanea, Lepra de montana, Liana, Okhet, One-year boil, Oriental sore, Pendjeh sore, Pian bois, Saldana, Ulcera de Bejuco, Urfa boil, Uta, Yatevi, Year boil. ICD9: 085.1,085.2,085.3,085.4 ICD10: B55.1

Clinical

WHO Case definition for surveillance:
Clinical description
• Appearance of one or more lesions, typically on uncovered parts of the body.
• The face, neck, arms and legs are the most common sites.
• At the site of inoculation a nodule appears, and may enlarge to become an indolent ulcer.
• The sore remains in this stage for a variable time before healing, and typically leaves a depressed scar.
• Other atypical forms may occur.
• In some individuals, certain strains can disseminate and cause mucosal lesions. These sequelae involve nasopharyngeal tissues and can be very disfiguring.
Laboratory criteria for diagnosis
• positive parasitology (stained smear or culture from the lesion)
• mucocutaneous leishmaniasis only: positive serology (IFA, ELISA)
Case classification
WHO operational definition:
• A case of cutaneous leishmaniasis is a person showing clinical signs (skin or mucosal lesions) with parasitological confirmation of the diagnosis (positive smear or culture) and/or, for mucocutaneous leishmaniasis only, serological diagnosis.

Typically, a nodule develops at the site of a sandfly bite following a few days to several months. [1] [2]
• The lesion may be erythematous, or covered by a thin yellow crust.
• The nodule reaches a diameter of 1 to 5 cm over a period of weeks or months, and is not painful. [3]
• The crust may thicken, and even replace the nodule; or may fall away to reveal an ulcer with a raised edge.
• Panniculitis [4] and satellite papules are common.
• The lesion may heal over a period of months or even years, leaving a depressed scar.
• Secondary infection is not prominent, and the major residua are scarring and disability.
• Rare instances of late scar carcinoma have been reported. [5]

Lesions caused by ***Leishmania major*** evolve and heal most rapidly, and are often inflamed or exudative ("wet sore" or "rural sore").
• Lesions caused by *L. tropica* are less inflamed ("dry sore" or "urban sore").
• Lesions due to *L. infantum* appear only after several months, and are small, nodular, and persist for years.

Lesions of ***L. aethiopica*** are typically single, and often involve the face.

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• Satellite papules evolve to produce a slowly growing, shiny tumor or plaque that may not crust nor ulcerate.
• If the site borders an area of mucosa, mucocutaneous leishmaniasis may develop, with swelling of the lips and enlargement of the nose over many years.

Leishmania brasiliensis produces deep, usually single, ulcers with a granulomatous base.
• 15 per cent of patients will relapse after spontaneous recovery or therapeutic improvement.

The lesions of ***L. guyanensis*** are multiple, fleshy and protuberant, and involve the limbs.
• Unlike other *Leishmania* species, ***L. braziliensis*** and ***L. panamensis*** are commonly associated with metastatic lesions along the path of draining lymphatics.
• Nodular lymphadenitis occurs, and may mimic nocardiosis. [6]

The lesions of ***L. mexicana*** ('chiclero ulcer') are commonly located on the side of the face or behind the ears.
• The lesion consists of a single ulcerative lesion, most commonly involving the ear pinna, without a tendency for cutaneous metastasis, lymphatic or mucosal involvement. [7]
• Destruction of the pinna is common.

Skin lesions with regional adenopathy may also occur in visceral leishmaniasis, and suggest a diagnosis of cutaneous leishmaniasis. [8]
• Facial lesions of chromomycosis may be misdiagnosed as cutaneous leishmaniasis. [9]

Other clinical forms:
Three forms of cutaneous leishmaniasis do not heal spontaneously: **Disseminated cutaneous leishmaniasis**, **Leishmaniasis recidivans** and **American mucosal leishmaniasis**.
• Diffuse cutaneous leishmaniasis is often seen with *L. amazoensis* infections, and also occurs in about 0.01% of *L. aethiopica* infections.
• The nodule spreads locally without ulceration, while secondary hematogenous lesions appear on other body sites.
• These are often symmetrical, and involve the face and extensor surfaces of the limbs.
• The external genitalia may also be affected, but the eye, mucosae and peripheral nerves are not infected (in contrast to lepromatous leprosy).
• The infection evolves gradually over many years.

Cases of erysipeloid, recidiva cutis (LRC), and disseminated leishmaniasis (DL) have been ascribed to *L. panamensis* infection. [10]

Leishmaniasis recidivans (lupoid leishmaniasis) is a rare complication of *L. tropica* infection.
• The lesion presents as a spreading nodule, leading to a plaque formation simulating discoid lupus erythematosus. [11] [12]
• After initial healing, papules reappear in the edge of the scar and the lesion spreads slowly over many years.
• The condition most commonly involves the face, and may be quite disfiguring.

Sporotrichoid cutaneous leishmaniasis may mimic cutaneous sporotrichosis. [13]
• Lesions of cutaneous leishmaniasis may mimic those of erysipeloid [14] or carcinoma. [15]
• Diffuse cutaneous leishmaniasis may mimic lepromatous leprosy [16] The lesions of both cutaneous and mucocutaneous leishmaniasis could be mistaken for those of borderline tuberculoid leprosy. [17]

Atypical, non-ulcerating nodular granulomatous lesions caused by *L. mexicana* and *L. chagasi* have been described in Central America.
• Most cases have involved exposed areas on the body, and most patients have been children.

In rare cases, leishmaniasis of the nose may present as rhinophyma [18] or mimic erysipelas. [19]

This disease is endemic or potentially endemic to 85 countries.

Leishmaniasis - cutaneous in Brazil

Time and Place:
Disseminated cutaneous disease due to *Leishmania amazonensis* [20] is endemic to Amapa, Amazonas, Bahia, Ceara, Goias, Minas Gerais [21], Mato Grosso do Norte, Para, Piaui and Roraima.
- *L. guyanensis* [22] [23], *L. lainsoni* [24] [25], *L. naiffi* [26], *L. mexicana* [27] and *L. shawi* [28] are also encountered. [29]
- *Leishmania brasiliensis* causes a distinct form of disseminated leishmaniasis, almost exclusively in Northern and Northeastern Brazil.
- The condition is characterized by 10 to 300 papular, acneiform, nodular or ulcerative lesions containing few parasites on microscopic examination. [30] [31]
- Disseminated leishmaniasis accounted for 0.2% of cutaneous infection in these areas during 1978 to 1984; 1.9% during

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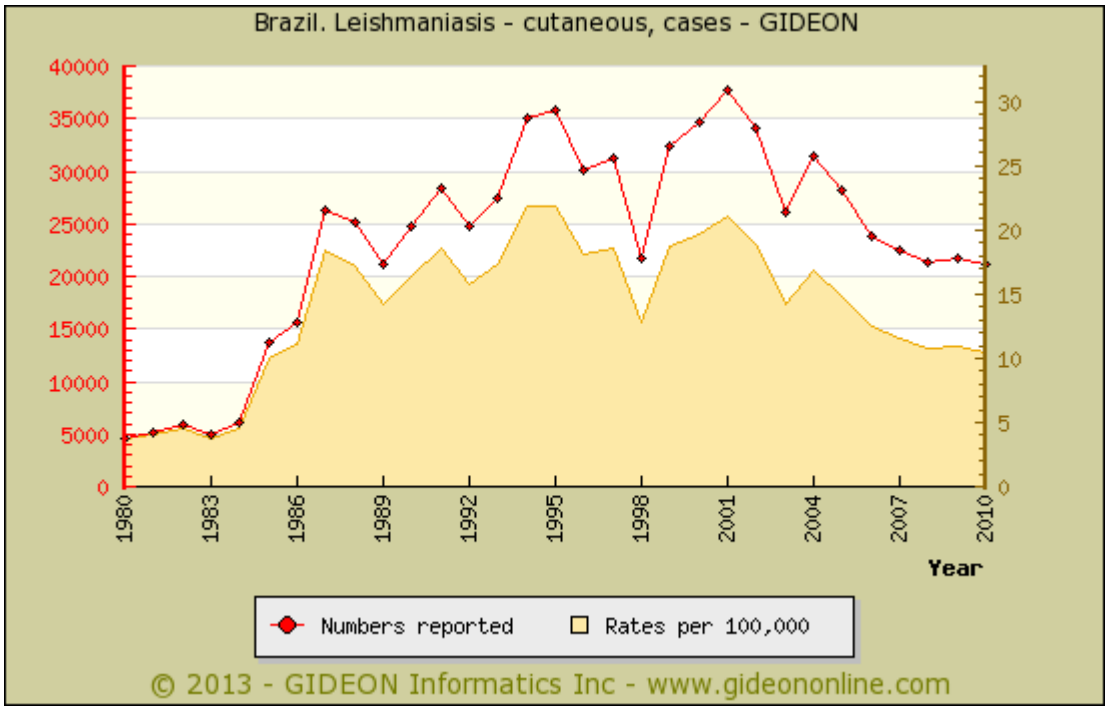
1992 to 1998.



Graph: Brazil. Leishmaniasis - cutaneous, cases

Notes:
1. 63,078 cases were reported during 1971 to 1980 (44.6% from Ceara); 455,007 during 1980 to 2000.

Disease rates per 100,000 in the Amazon region: 79.9 in 2001; 81.8 in 2002; 97.3 in 2003; 88.9 in 2004; 75.7 in 2005; 60.7 in 2006. [32]

Prevalence surveys:

17.46% of tissue samples from rats (*Rattus norvegicus*) in Belo Horizonte, Minas Gerais (*Leishmania braziliensis* PCR, 2011 publication) [33]

0.22% of sandflies (*Nyssomyia neivai*) in Doutor Camargo, Parana State (*Leishmania* spp, 2008) [34]

0.4% of *Lutzomyia whitmani* in Maranhao (2006 publication) [35]

0.77% of sandflies in Espirito Santo State - including *Lutzomyia intermedia*, *Lu. whitmani*, *Lu. fischeri* and *Lu. ferreirana* (2004 to 2008). [36]

18.2% of *L. whitmani* in Divinopolis - 13.2% of *L. neivai*, 3.1% of *L. christenseni*, 1.9% of *L. pessoai*, 0.6% of *L. aragaoi*, 0.6% of *L. fischeri*, 0.6% of *L. lenti*, 0.6% of *L. lutziana* and 0.6% of *L. monticula* (2010 publication) [37]

Seroprevalence surveys:

4.0% of inhabitants of 15 villages were seropositive to *L. braziliensis*. (Paco do Lumiar County) [38]

16.7% of dogs in northwestern Parana State (2005) [39]

6.2% of dogs in Itapema and Belneario Camboriu, Santa Catarina State (ELISA, 2010 publication) [40]

13.9% of dogs from 3 regions of Southern Brazil were seropositive and 2.4% had cutaneous lesions, from which *L. braziliensis* was identified. (Parana State, 2007 publication) [41]

10.7% of dogs and 2.4% of cats in Santa Rita de Cassia, municipality of Barra Mansa, Rio de Janeiro (2009 publication) [42]

8.1% to 21.1% of opossums (*Didelphis marsupialis*) in metropolitan areas were seropositive to *Leishmania* species, including *L. braziliensis* complex. (Belo Horizonte, Minas Gerais, 2007 publication) [43]

Regional data:

Acre: Infection in Rio Branco, Acre is caused by *Leishmania (Viannia) braziliensis*, *Leishmania (Viannia) lainsoni*, *Leishmania (Viannia) guyanensis*, *Leishmania (Viannia) naiffi* and *L. (V.) lainsoni*. [44]

Amazonas State: 715 cases (28.4 per 100,000) were in 1998; 2,072 (80.3 per 100,000) in 1999.

- *Leishmania naiffi* has been isolated in Para, Amazonas and Rondonia - from both humans and armadillos (*Dasypus novemcintcus*).

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- 2,557 cases of cutaneous and mucocutaneous leishmaniasis were registered in Acre State during 1992 to 1997, with rates of 2,310 per 100,000 in Brasilia.
Bahia: The principal agent is *Leishmania (Viannia) braziliensis*; and vectors *Lutzomyia (Nyssomyia) intermedia* [45] , *Lu. whitmani* and *Lu. migonei*.
- 48,138 cases of American cutaneous leishmaniasis were reported in Bahia during 1985 to 1999.
- 13% of municipalities in Bahia were endemic in 1985; 27% in 1990; 34% in 1996.
- 1,396 cases were reported from a single institution (2005 to 2006) [46]
Bahia, Mato Grosso Santa Catarina: *Leishmania amazoensis* is reported. [47]
Manaus region: Most cases of cutaneous leishmaniasis in the Manaus region are caused by *Leishmania guyanensis* - *L. brazilensis* is rare. *L. amazonensis* and *L. naiffii* are also reported in the area. [48]
Mato Grosso: The principal agent in is *L. (V.) brasiliensis*. [49]
- 2,283 cases (27.1 per 100,000) were reported in Matto Grosso State in 1998; 4,895 (50.5 per 100,000) in 1999.
Northeastern Brazil reported 2,000 cases in 1980; 9,000 in 1993; 13,919 in 2000.
Pernambuco: Transmission rates increased ten-fold during 1990 to 1995. 6,306 cases were reported in Pernambuco during 1990 to 1999.
Para State: [50] 2,177 cases (37.7 per 100,000) were reported in 1998; 5,051 (86.2 per 100,000) in 1999.
Parana State: An endemic focus reported in Prudentopolis (100 cases) (2009 publication) [51] In one series, *Leishmania braziliensis* accounted for 100% of cases in this state. [52]
Rio de Janeiro State: *L. amazonensis* infection was first reported in 2007, as a cause of diffuse cutaneous leishmaniasis. [53]
Rondonia State: Local agents include *Leishmania braziliensis*, *L. lainsoni* and *L. guyanensis*. [54]

Infecting species:
The agents of localized cutaneous leishmaniasis in Brazil are *Leishmania amazoensis* [55] , *L. brasiliensis*, *L. guyanensis*, *L. lainsoni*, *L. lindenbergi* [56] , *L. naiffi* [57] [58] and *L. shawi*. [59]
- Disseminated cutaneous disease is caused by *Leishmania amazonensis* [60] , *L. guyanensis* [61] , *L. lainsoni* [62] [63] , *L. naiffi* [64] , *L. mexicana* [65] and *L. shawi* [66-68]

Reservoirs:
The mammalian reservoirs for *Leishmania amazoensis* in Brazil and *Didelphis marsupialis*, *Philander opossum*, Marmosa cinerea, *Metachirus nudicaudatus*, *Cerdocyon thous*, *Oryzomys megacephalus*, *Dasyprocta leporina* and *Proechimys guyannensis*. [69]
- The reservoirs for *Leishmania guyanensis* are *Didelphis marsupialis*, *Choloepus didactylus*, *Dasypus novemcinctus*, *Tamandua tetradactyla* and *Proechimys guyannensis*.
- *Leishmania amazonensis* and *Leishmania braziliensis* have been identified in free-ranging marsupials (*Micoureus paraguayanus* and *Didelphis albiventris*) in the Pontal do Paranapanema region of Sao Paulo State [70]
- *Leishmania amazoensis* infection was identified in a captive spider monkey (*Ateles paniscus*) in Bauru, Sao Paulo. [71]
- The reservoirs for *Leishmania lainsoni* are *Agouti paca*, and possibly *Cebus apella*.
- The reservoir for *Leishmania naiffi* is *Dasypus novemcinctus*.
- There is evidence for infection by *Leishmania (Leishmania) amazonensis* and *Leishmania (Leishmania) infantum chagasi* in Brazilian bats. [72]
- Natural infection of cats has been reported in Rio de Janeiro (2004 publication). [73]
- Natural *L. braziliensis* infection has been reported in horses [74] , dogs, water rats (*Nectomys squamipes*), black rats (*Rattus rattus*), mice (*Mus musculus*) [75] , grass mice (*Bolomys lasiurus*), march mice (*Holochilus scieurus*), field mice (*Akodon arviculoides*), wooly opossums (*Marmosa* sp) and common opossums (*Didelphis albiventris*).
- *Leishmania braziliensis* was identified in 17.46% of tissue samples from rats (*Rattus norvegicus*) in Belo Horizonte, Minas Gerais (PCR, 2011 publication) [76]

Vectors:
The principal Brazilian vector for *L. (V.) braziliensis* is *Lutzomyia (Psychodopygus) wellcomei*. *Lutzomyia (Psychodopygus) complexa* [77] and *Lutzomyia neivai* [78] [79] are also implicated.
- *Lu. (Nyssomyia) anduzei*, *Lu. umbritilis* [80] and *Lu. whitmani* are the local vectors for *L. guyanensis*. *Lu. whitmani* also serves as vector for *L. braziliensis* in Parana State.
Leishmania spp. were found in 0.22% of sandflies (*Nyssomyia neivai*) in Doutor Camargo, Parana State (2008) [81]
Nyssomyia neivai predominated among sandflies captured in Itambaraca, Porto Almeida and Sao Joaquim do Pontal, Parana State - followed by *Pintomyia pessoai*, *Migonemyia migonei*, *Nyssomyia whitmani* and *Pintomyia fischeri* (2004 to 2006) [82]
- *Lu. (N.) umbratilis* is the vector for *Leishmania guyanensis* in northern Para State and Manaus. [83] [84] *Lu. (N.) anduzei* also acts as a vector for this species in Manaus.
Lutzomyia (Nyssomyia) flaviscutellata is implicated as a vector for *Leishmania amazoensis* in Para State; and *Lu. (N.) olmeca vociva* in the area of Manaus. *Lutzomyia anduzei* and *Lutzomyia claustrei* are also found in the region. [85]
Additional potential vectors have been identified in the urban periphery of Manaus: *Nyssomyia umbratilis* and *Nyssomyia anduzei* (2001 to 2002) [86]
- Additional vectors for *L. guyanensis* include *Lu. spathotricha*. *Psathyromyia dendrophyla*, and possibly *Ps. shannoni*. [87]

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- Vectors in Rio de Janeiro State include *Lu. migonei*, *L, intermedia* [88] , *Lu. longipalpis* [89] and *Migonemyia migonei* [90]
- 0.4% of *Lutzomyia whitmani* in Maranhao are infested (2006 publication) [91] Additional vectors in Maranhao include *L. longipalpis*, *L. evandroi*, *L. lenti*, *L. termitophila*, *L. flaviscutellata*, *L. migonei*, *L. infraspinosa*, *L. sordellii*, *L. wellcomei*, *L. antunesi*, and *L. trinidadensis*. [92]
- Sand fly species identified in Minas Gerais state include: *Brumptomyia brumpti*, *Lutzomyia bacula*, *Lutzomyia cortelezzii*, *Lutzomyia lent*i, *Lutzomyia sallesi*, *Lutzomyia longipalpis*, *Lutzomyia migonei*, *Lutzomyia intermedia*, *Lutzomyia neivai*, *Lutzomyia whitmani*, *Lutzomyia christenseni*, *Lutzomyia monticola*, *Lutzomyia pessoai*, *Lutzomyia aragaoi*, *Lutzomyia brasiliensis*, *Lutzomyia lutziana and Lutzomyia sordellii*. [93]

Lutzomyia whitmani [94] and *Lu. intermedia* are implicated in transmission in Minas Gerais. [95]

Lutzomyia whitmani is the predominant vector in wooded areas of Belo Horizonte, Minas Gerais; while *L. longipalpis* is the predominant vector in urban areas (2010 publication) [96]
- *Lutzomyia (Nyssomyia) whitmani*, *L. (N.) antunesi*, *L. (L.) spathotrichia*, *L. (P.) c. carrerai*, *L. (P.) complexa*, *L. (P.) lainsoni* and *L. (N.) umbratilis* comprise 92.4% of sandfly species in Matto Grosso (2002 publication). [97]
- *Bichromomyia flaviscutellata*, *Brumptomyia avellari*, *Brumptomyia brumpti*, *Brumptomyia sp*, *Evandromyia aldafalcaoae*, *Evandromyia cortelezzii*, *Evandromyia evandroi*, *Evandromyia lenti*, *Evandromyia teratodes*, *Evandromyia termitophila*, *Lutzomyia longipalpis*, *Nyssomyia whitmani*, *Pintomyia christenseni*, *Psathyromyia aragaoi*, *Psathyromyia campograndensis*, *Psathyromyia punctigeniculata*, *Psathyromyia shannoni* and *Sciopemyia sordellii* have been identified in Matto Grosso do Sul. [98]

Lu. longipalpis has been identified as a vector for *Leishmania amazonensis* in Sierra da Bodoquina, Matto Grosso do Sul [99]
- *Lutzomyia intermedia* and *Lutzomyia choti* are implicated as vectors in Espirito Santo State (2008 publication). [100]
- *Nyssomyia neivai* has been identified as a natural vector for *Leishmania braziliensis* in Santa Catarina (2006) [101]
- *Lutzomyia choti* has been implicated in transmission in Pernambuco. [102]
- Phlebotomines identified in an undisturbed region of the Amazon region included *Lutzomyia flaviscutellata* (64.5% of strains), *Lutzomyia georgii*, *Lutzomyia olmeca nociva*, *Lutzomyia furcata*, *Lutzomyia monstruosa* and *Lutzomyia umbratilis* (2010 publication) [103]
- 0.77% of sandflies in Espirito Santo State are infested - including *Lutzomyia intermedia*, *Lu. whitmani*, *Lu. fischeri* and *Lu. ferreirana* (2004 to 2008). [104]
- Vectors identified in Espirito Santo do Pinhal include *Nyssomyia whitmani* and *Pintomyia pessoai* in the rural area, *Lutzomyia longipalpis* and *Ny. whitmani* in the periurban area and *Lu. longipalpis* in the urban area. [105]
- *Lutzomyia (Pintomyia) fischeri* has been identified as a probable vector in Porto Alegre. [106]
- 18.2% of *L. whitmani* in Divinopolis were found to be infested, 13.2% of *L. neivai*, 3.1% of *L. christenseni*, 1.9% of *L. pessoai*, 0.6% of *L. aragaoi*, 0.6% of *L. fischeri*, 0.6% of *L. lenti*, 0.6% of *L. lutziana* and 0.6% of *L. monticula* (2010 publication) [107]

Notable outbreaks:

1978 - Outbreaks cutaneous leishmaniasis were reported in Maranhao [108] and the Ribeira Valley, Sao Paulo [109]

1984 to 1986 - An outbreak (105 cases) of *Leishmania braziliensis braziliensis* infection was reported in an urbanized area of Rio de Janeiro. [110]

1984 to 1989 - An outbreak of *Leishmania (Viannia) braziliensis* infection was reported in Corte de Pedra, Bahia. [111]

1992 - An outbreak was reported in the Northeast region of Sao Paulo state. [112]

1993 (publication year) - An outbreak of *Leishmania (Viannia) braziliensis* infection was reported in Itaporanga, Sao Paulo. [113]

1993 - An outbreak An outbreak (29 cases) of *Leishmania braziliensis* infection was reported in the village of Canoa, Santo Amaro, Bahia. [114]

1993 to 1994 - An outbreak (25 cases) was reported in Campinas, Sao Paulo State. [115]

1998 (publication year) - An outbreak (26 cases) of American cutaneous leishmaniasis was reported in military training unit in "Zona da Mata," Pernambuco State. [116]

2002 (publication year) - An outbreak (8 cases) of *Leishmania (V.) lindenbergi* infection was reported among soldiers stationed in Belem, Para. [117]

2002 (publication year) - An outbreak (72 cases) was reported in the Jequitinhonha River Valley, Minas Gerais. [118]

2002 - An outbreak (38 human and 24 canine cases) of *Leishmania braziliensis* infection was reported in Parana State. [119]

2005 (publication year) - An outbreak was reported in Ceara State. [120]

2009 (publication year) - An outbreak (71 cases) of American tegumentary leishmaniasis due to *Leishmania (Viannia) braziliensis* was reported among military personnel in Pernambuco. [121]

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Leishmaniasis - mucocutaneous

Agent	PARASITE - Protozoa. Neozoa, Euglenozoa, Kinetoplastea. Flagellate: Leishmania braziliensis, et al
Reservoir	Rodent Human Sloth Marsupial
Vector	Fly (sandfly = Lutzomyia or Psychodopygus)
Vehicle	None
Incubation Period	2w - 8w (range 1w - 6m)
Diagnostic Tests	Microscopy (culture in specialized laboratories). Serology. Nucleic acid amplification.
Typical Adult Therapy	Pentavalent antimonials (Stibogluconate) 20 mg/kg/d IV/IM X 28d. OR Amphotericin B 0.5 mg/kg/d X 4 to 8w High dose (8 mg/kg/day) fluconazole has been used against Leishmania braziliensis
Typical Pediatric Therapy	As for adult
Clinical Hints	Skin ulceration or nasopharyngitis associated with purulent, mucoid exudate; the process may extend to underlying soft tissues; metastatic lesions often involve the palate and pharynx.
Synonyms	Agla, Espundia, Mucocutaneous leishmaniasis. ICD9: 085.5 ICD10: B55.2

Clinical

As many as 40 per cent of patients with cutaneous ulcers due to *Leishmania brasiliensis* will develop mucosal involvement, 50% of these within two years of the original infection, and 90% within ten years (cases appearing after as many as 35 years have been reported).
• 15% of patients give no history of a previous primary skin lesion.
• In virtually all cases the nasal mucosa is affected, and in 33% a second site is also involved • the pharynx, palate, larynx, or upper lip, in order of frequency.
• Lesions may be subtle, and a thorough otorhinolarygologic examination should be performed in suspect cases. [1]
• Computed tomographic (CT) scans reveal involvement of the paranasal sinuses in most cases. [2]

The initial presentation is typically nasal obstruction associated with a nodule or polyp on the inferior turbinate. [3] [4]
• The process evolves slowly to perforate the nasal septum, and may ultimately destroy the entire mouth and nose.
• Death may result from secondary sepsis, starvation or laryngeal obstruction.
• Extensive lesions are less common in infection by *Leishmania panamensis* and *Leishmania guyanensis*.

A form of muco-cutaneous disease (Sudanese mucosal leishmaniasis) has been reported in Africa.
• The disease is not preceded or accompanied by cutaneous lesions.
• Infection presents as either: nasal obstruction, discharge and bleeding; oral 'fullness', loss of teeth and bleeding gums; or perforation of the hard palate.
• The muco-cutaneous form is found almost exclusively in Sudanese adult males. [5]

The clinical features of mucosal leishmaniasis may suggest a misdiagnosis of Wegener's granulomatosis. [6]
• The lesions of both cutaneous and mucocutaneous leishmaniasis could be mistaken for those of borderline tuberculoid leprosy. [7]

This disease is endemic or potentially endemic to 17 countries.

Leishmaniasis - mucocutaneous in Brazil

Mucocutaneous leishmaniasis was first described in Brazil in 1911, and is most common in Amapa, Amazonas, Bahia (notably Corte de Pedra [8]), Mato Grosso do Norte, Minas Gerais, Para, Roraima and Sao Paulo. [9]
- Mucosal leishmaniasis due to *Leishmania guyanensis* has been reported in Para, Acre, and Rondonia States; and cases caused by *Leishmania braziliensis* in Rondonia State. [10]

Disseminated forms of *Leishmania braziliensis* infection are increasingly reported. [11] [12]

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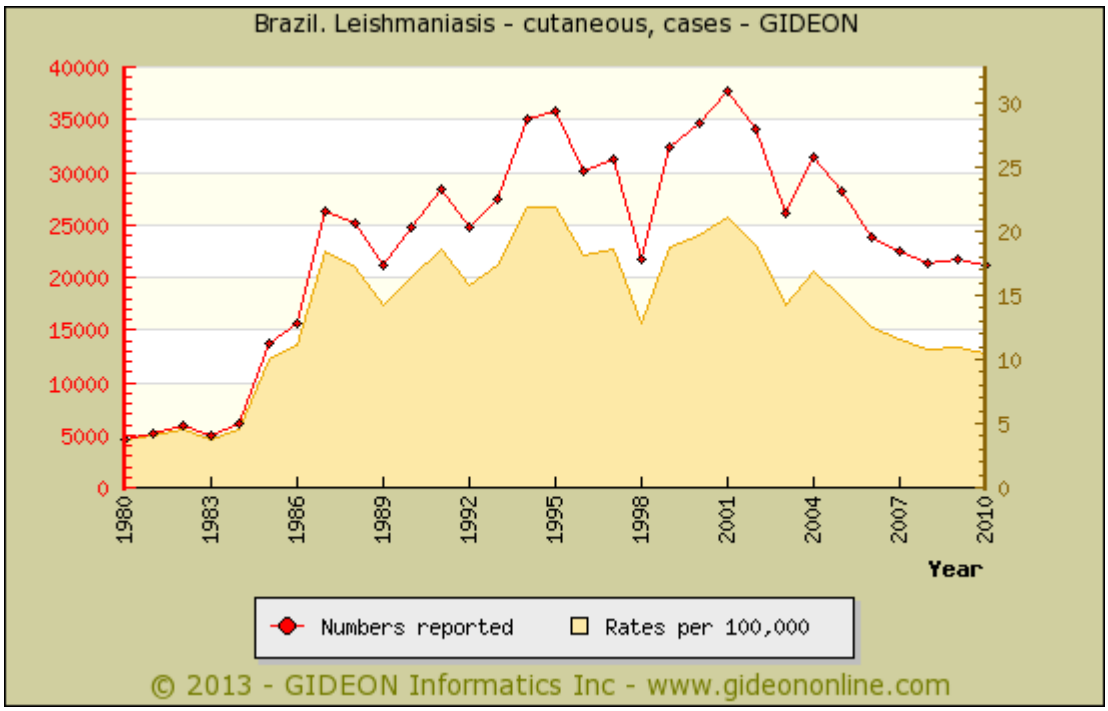
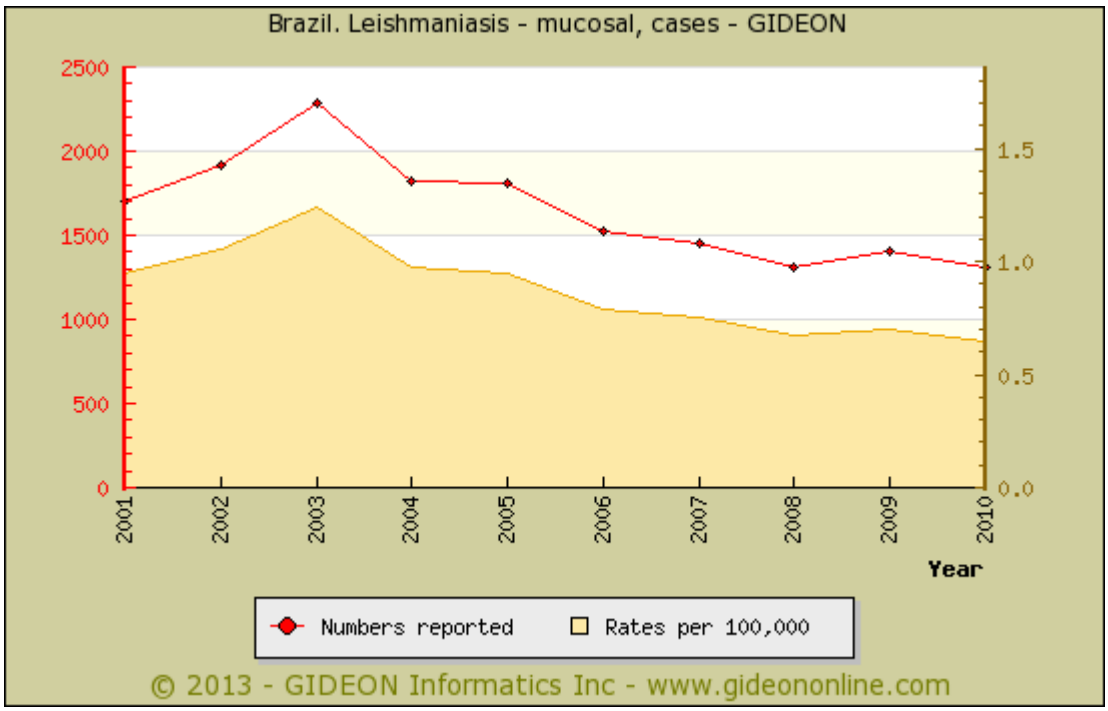
Young males are more often affected by the mucosal form of leishmaniasis than other segments of the population. (Gujara-Mirim, Rondonia, 2007 publication) [13]



Graph: Brazil. Leishmaniasis - cutaneous, cases

Notes:
1. Data include both cutaneous and mucocutaneous leishmaniasis.
2. 63,078 cases were reported during 1971 to 1980 (44.6% from Ceara); 455,007 during 1980 to 2000.



Graph: Brazil. Leishmaniasis - mucosal, cases

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Leishmania brasiliensis accounts for 16.7% of infectious granulomatous laryngitis cases (2007 publication) [14]

Reservoirs: (see complete listing in note for Leishmaniasis-cutaneous)
- Natural infection of sloths (*Bradypus variegatus*) and cats (*Felis catus*) has been demonstrated in Rio de Janeiro State.
- Natural *L. braziliensis* infection of horses, dogs, water rats (*Nectomys squamipes*), black rats (*Rattus rattus*), grass mice (*Bolomys lasiurus*), march mice (*Holochilus scieurus*), field mice (*Akodon arviculoides*), wooly opossums (*Marmosa* sp) and common opossums (*Didelphis albiventris*) has been demonstrated in Pernambuco State.
- 13.9% of dogs from 3 regions of Southern Brazil were seropositive and 2.4% had cutaneous lesions, from which *L. braziliensis* was identified. (Parana State, 2007 publication) [15]
- 8.1% to 21.1% of opossums (*Didelphis marsupialis*) in metropolitan areas were seropositive to *Leishmania* species, including *L. braziliensis* complex. (Belo Horizonte, Minas Gerais, 2007 publication) [16]
- There is evidence for infection by *Leishmania (Leishmania) amazonensis* and *Leishmania (Leishmania) infantum chagasi* in Brazilian bats. [17]

Vectors: (see complete listing in note for Leishmaniasis-cutaneous)
The principal vector for *Leishmania braziliensis* in Brazil is *Lutzomyia (Psychodopygus) wellcomei*. [18]
- *Lu. pessoai* (Sao Paulo, *Lu. whitmani* and *Ps. complexus* (in Para), *Lutzomyia neivai* (in Porto Alegre) [19] and *L. (N.) intermedia* (in Sao Paulo) are also implicated.
- Additional suspect vectors in Brazil include *Lu. migonei*, *Lu. (Ps.) paraensis* and *Lu. (Ps.) amazoensis*. [20]
- *Lu. (Ps.) whitmani* also serves as vector for *Leishmania braziliensis* in Parana and Bahia States.
- Infected *Lutzomyia intermedia* and *Lutzomyia migonei* have been identified in Rio de Janeiro. [21]
- Potential vectors identified in the urban periphery of Manaus include *Nyssomyia umbratilis* and *Nyssomyia anduzei* (2001 to 2002) [22]
- Sand fly species identified in Minas Gerais state include: *Brumptomyia brumpti*, *Lutzomyia bacula*, *Lutzomyia cortelezzii*, *Lutzomyia lenti*, *Lutzomyia sallesi*, *Lutzomyia longipalpis*, *Lutzomyia migonei*, *Lutzomyia intermedia*, *Lutzomyia neivai*, *Lutzomyia whitmani*, *Lutzomyia christenseni*, *Lutzomyia monticola*, *Lutzomyia pessoai*, *Lutzomyia aragaoi*, *Lutzomyia brasiliensis*, *Lutzomyia lutziana and Lutzomyia sordellii*. [23]
- *Nyssomyia neivai* has been identified as a natural vector for *Leishmania braziliensis* in Santa Catarina (2006) [24]

Notable outbreaks:

1978 - Outbreaks cutaneous leishmaniasis were reported in Maranhao [25] and the Ribeira Valley, Sao Paulo [26]

1984 to 1986 - An outbreak (105 cases) of *Leishmania braziliensis braziliensis* infection was reported in an urbanized area of Rio de Janeiro. [27]

1984 to 1989 - An outbreak of *Leishmania (Viannia) braziliensis* infection was reported in Corte de Pedra, Bahia. [28]

1993 (publication year) - An outbreak of *Leishmania (Viannia) braziliensis* infection was reported in Itaporanga, Sao Paulo. [29]

2002 - An outbreak (38 human and 24 canine cases) of *Leishmania braziliensis* infection was reported in Parana State. [30]

2009 (publication year) - An outbreak (71 cases) of American tegumentary leishmaniasis due to *Leishmania (Viannia) braziliensis* was reported among military personnel in Pernambuco. [31]

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31. Rev Soc Bras Med Trop 2009 Sep-Oct;42(5):594-6.

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Leishmaniasis - visceral

Agent	PARASITE - Protozoa. Neozoa, Euglenozoa, Kinetoplastea. Flagellate: Leishmania donovani, L. infantum, L. cruzi; rarely, L. tropica
Reservoir	Human Rodent Dog Fox Hares
Vector	Fly (sandfly = Phlebotomus for old world; Lutzomyia for new world)
Vehicle	Blood
Incubation Period	2m - 6m (10d - 12m)
Diagnostic Tests	Smear / culture of bone marrow, splenic aspirate, lymph nodes. Serology. Nucleic acid amplification.
Typical Adult Therapy	Pentavalent antimonials (Stibogluconate) 20 mg/kg/d X 28d. OR Amphotericin B 1 mg/kg/QOD X 8w (or lipid complex 3 mg/kg/d X 5d) OR Paromomycin 11 mg/kg IM QD X 21 days OR Miltefosine 50 to 150 mg PO daily X 4 to 6 weeks.
Typical Pediatric Therapy	Pentavalent antimonials (Stibogluconate) 20 mg/kg/d X 28d. OR Amphotericin B 1 mg/kg/QOD X 8w (or lipid complex 3 mg/kg/d X 5d) OR Paromomycin 11 mg/kg IM QD X 21 days OR Miltefosine 2.5 mg/kg daily (maximum 150 mg) X 28d
Clinical Hints	Chronic fever, weight loss, diaphoresis, hepatosplenomegaly, lymphadenopathy and pancytopenia; grey pigmentation (Kala Azar = 'black disease') may appear late in severe illness; case-fatality rate = 5% (treated) to 90% (untreated).
Synonyms	Burdwan fever, Cachectic fever, Dum Dum fever, Kala azar, Leishmania donovani, Leishmania infantum, Leishmania siamensis, Leishmaniose: Viszerale, Leishmaniosi viscerale, Ponos, Visceral leishmaniasis. ICD9: 085.0 ICD10: B55.0

Clinical

WHO Case definition for surveillance:
Clinical description
• An illness with prolonged irregular fever, splenomegaly and weight loss as its main symptoms.
Laboratory criteria for diagnosis
• positive parasitology (stained smears from bone marrow, spleen, liver, lymph node, blood or culture of the organism from a biopsy or aspirated material)
• positive serology (IFA, ELISA)
Case classification
WHO operational definition:
• A case of visceral leishmaniasis is a person showing clinical signs (mainly prolonged irregular fever, splenomegaly and weight loss) with serological (at geographical area level) and/or parasitological confirmation (when feasible at central level) of the diagnosis.
• In endemic malarious areas, visceral leishmaniasis should be suspected when fever lasts for more than two weeks and no response has been achieved with anti-malaria drugs (assuming drug resistant malaria has also been considered).

Following an incubation period of two to eight months, the patient develops chronic fever, abdominal pain (from an enlarged spleen) and swelling, weight loss, cough and occasionally, diarrhea.
• The classical fever rises twice daily, without rigors; however, single 'spikes,' irregular or undulant fevers are common.
• Caucasians may experience an abrupt onset of high fever, with rapid progression of illness, toxemia, weakness, dyspnea, and anemia.
• Visceral leishmaniasis in HIV-positive patients is characterized by short incubation period, high incidence of multi-organ disease, and tendency to relapse. [1]

Physical signs may be limited to splenomegaly; but chronically-ill patients are typically pale and cachetic.
• Hyperpigmentation of face, extremities and abdomen ('Kala azar) may be present in advanced cases.
• The spleen is non-tender, and may be massively enlarged, reaching the left or even right iliac fossa.
• Moderate hepatomegaly is present in one-third of cases.
• Rare instances of granulomatous hepatitis are reported. [2]
• Generalized lymphadenopathy is found in 50% of African patients, and a smaller percentage of Indian and European cases. In some cases, localized lymphadenopathy may be the only sign of infection. [3]
• Jaundice, mucosal and retinal hemorrhage, laryngeal lesions [4] , uveitis, chronic diarrhea with malabsorption [5] , interstitial

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nephritis [6] , pericardial effusion [7] and panniculitis [8] are occasionally encountered.
• Skin lesions with regional adenopathy may suggest a diagnosis of cutaneous leishmaniasis. [9]
• A chronic rash (Post kala-azar dermal leishmaniasis = PKDL) resembling leprosy, and involving primarily the extremities and face often appears months to years following infection. [10]
• Other rare manifestations include the hemophagocytic syndrome [11] , laryngeal infection [12] , leukemoid changes or myelodysplasia or pyothorax [13]
• The clinical features of visceral leishmaniasis may mimic those of autoimmune hepatitis, primary biliary cirrhosis, and systemic lupus erythematosus. [14]

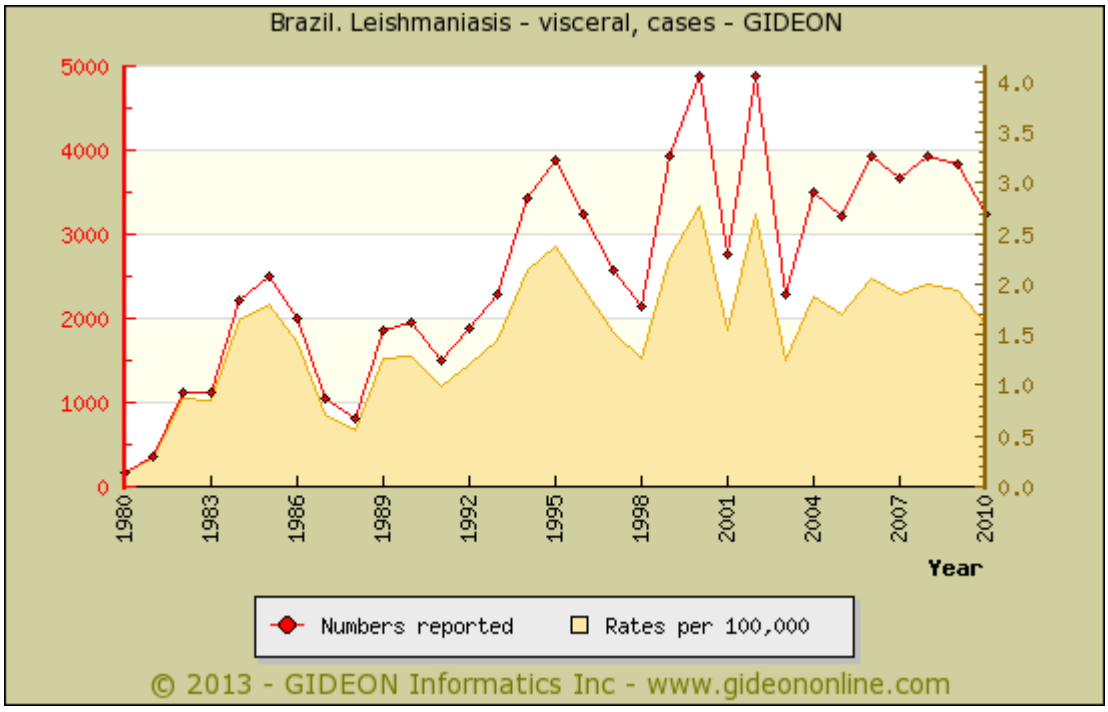
Laboratory studies reveal pancytopenia, hypoalbuminemia, hyperglobulinemia, and only mild hepatic dysfunction.
• In endemic countries, leishmaniasis is the principal cause of febrile pancytopenia among children without hematologic malignancy. [15]
• Intercurrent infections are common notably pneumococcal disease (otitis, pneumonia or septicemia), tuberculosis and measles.
• Evidence of primary adrenal insufficiency, hypoparathyoridism and hypothyroidism are common. [16]

The case/fatality rate without treatment is 80% to 90%.

This disease is endemic or potentially endemic to 108 countries.

Leishmaniasis - visceral in Brazil

Time and Place:
- Visceral leishmaniasis was first described in Brazil in 1934 and first reported from Rio de Janeiro state in 1977. [17]
- 90% of cases are reported from the north (mouth of Amazon) and eastern region.
- The disease is endemic to Alagoas, Bahia [18] , Ceara, Espirito Santo, Goias, Maranhao [19] , Mato Grosso do Sul, Minas Gerais [20] , Para, Paraiba, Pernambuco, Piaui [21] , Rio Grande do Norte, Roraima and Sergipe.
- Urban transmission was first reported in 1981, from Teresina (Piaui State).
- A large focus extends from Roraima into Venezuela and Guyana. [22]



Graph: Brazil. Leishmaniasis - visceral, cases

Notes:
1. 44,289 cases were reported during 1980 to 2000 - 39,823 of these from the northeast.
2. Brazil accounts for over 90% of cases in the New World.
3. Federal District reported 15 cases during 2005 to 2008 - including 3 in 2007 and 4 in 2008. [23]

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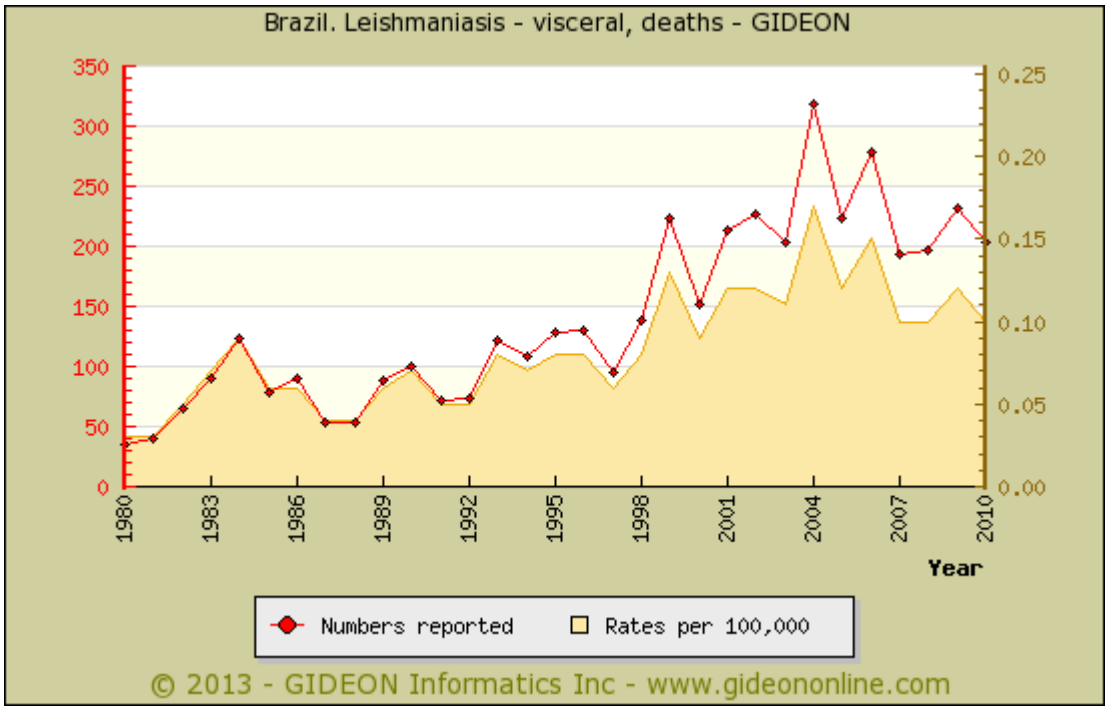

Graph: Brazil. Leishmaniasis - visceral, deaths

Incidence by district and state:
- Aracatuba (Sao Paulo) reported 43 cases (4 fatal) in 2002.
- Bahia reported 12,413 cases during 1985 to 1999. 7% of municipalities were endemic in 1985; 18% in 1990; 30% in 1996.
- Belo Horizonte reported only one case prior to 1994; 35 during an epidemic in 1994.
- Brasilia, Federal District reported 21 autochthonous cases during 2005 to 2009. [24]
- Ceara: Fortaleza registered 1,379 cases during 2001 to 2006.
- Ipanema, Minas Gerais reported six cases during September 2009 to February 2010. [25]
- Mato Grosso reported 123 cases of canine infection in 2007; 301 (and 6 cases of human infection) in 2008. [26]
- Mato Grosso do Sul reported 114 cases (9 fatal) in 2001; 25 (6 fatal) during January to April 2002; 149 (12 fatal) during 2000 to 2003. [27] 577 cases were reported in Campo Grande in 2001. [28]
- Natal reported 316 cases Natal during 1989 to 1993.
- Pernambuco reported 1,310 cases during 1990 to 1999; 427 during 2000 to 2001. [29]
- Piaui and Maranhao reported 1,400 cases in 1994 (over 50% of Brazilian cases).
- Rio de Janeiro reported 87 cases during 1977 to 2006. [30]
- Roraima reported 82 cases among Indians during 1989 to 1993. 10.3% of dogs in the region are infested.
- Teresina reported almost 1,000 cases during 1981 to 1985; 1,200 during 1993 to 1996. [31]
- Tres Lagoas, Mato Grosso do Sul State reported 149 cases during 2000 to 2003. [32]

Prevalence surveys:

0.3% of solid organ transplant recipients (2001 to 2006) [33]
1% of renal transplant recipients in Fortaleza, Ceara State (2008 publication) [34] [35]

Seroprevalence surveys:

4.5% of inhabitants of 15 villages (*L. infantum*, Paco do Lumiar County, 2007 publication) [36]
4.8% to 20% of asymptomatic inhabitants of northeastern Sao Paulo (2007 publication) [37]
20.6% of persons in rural Rio Grande do Norte (2008 publication) [38]
24.6% of asymptomatic persons in Natal (2011 publication) [39]
47% in Pancas, Espirito Santo (2003 to 2004) [40]
18.9% of persons in Raposa, Maranhao State (2011 publication) [41]
12.6% of persons in Barcarena, PA (2003) [42]
24.4% of persons in an endemic region of Para State (2008 publication) [43]

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13.9% of children in an urban endemic area (2012 publication) [44]
22.3% of hemodialysis patients in Natal (2009 publication) [45]

The rate of infection among children ages 1 to 11 years in rural Ceara is 4.6% per year (1987 to 1989).
- 61.7% of children age <15 years in Sao Jose de Ribamar, Maranhao are skin test-positive. [46]
- Acquisition through blood transfusion has been reported. Nine percent of volunteer blood donors and 37% of multiply-transfused hemodialysis patients from Natal (Rio Grande do Norte) are seropositive (1994 to 1996).

160 deaths were ascribed to 'leishmaniasis' (unspecified) in 1996; 117 in 1997; 138 in 1998.

HIV-AIDS and visceral leishmaniasis: [47]
25 cases of HIV-*Leishmania* coinfection were reported to November 1995; 100 to June 2003.
- An additional 15 cases were reported in a 2009 publication. [48]
- The estimated rate of visceral leishmaniasis among HIV-positive patients treated at a tertiary medical center in Brasilia was 16% (2009 publication) [49]
- HIV-positive patients with visceral leishmaniasis were found to have a mean age of 37.3 years, vs. 12.5 years among HIV-negative leishmaniasis patients; and were more likely to be male (88% vs. 65%, Rio Grande do Norte, 1990 to 2009). [50]
- Asymptomatic leishmaniasis was identified in 10.8% of HIV-positive patients (ELISA, Minas Gerais, 1999 to 2001) [51]

Reservoirs:
- Dogs are key reservoirs in this country; in addition to foxes (*Cerdocyon thous*) in northeastern Brazil. [52]
- The role of additional fox species (*Dusicyon vetulus*) in the north; and of opossums (*Didelphis albiventris* and *D. marsupialis* [53]) is unclear.
- *Leishmania chagasi* infection has been documented in a bush dog (*Speothos venaticus*) in Mato Grosso State (2007 publication) [54]
- The first case in the Americas of natural infection in a cat (*Felis catus*) was reported from Sao Paulo State.
An infected cat was subsequently identified in Belo Horizonte City. [55]
5.8% of cats in Andradina Municipality, Sao Paulo State were found to be infected (2010 publication) [56] [57]
21.85% of cats in an endemic area of Sao Paulo State were found to be infected (2012 publication) [58]
23.0% of cats in Aracatuba, Sao Paulo State were found to be seropositive (CAG-ELISA, 2011 publication) [59]
49.1% of cats with dermatological problems were found to be infected by *Leishmania chagasi* (Aracatuba, 2008 to 2009) [60]
- An opossum (*Didelphis albiventris*) from Jacobina, Bahia State, was found naturally infected with *Leishmania donovani* [61] Natural infection has also been demonstrated in *Didelphis aurita* in the region of Rio de Janeiro [62] and in 20.4% of *Didelphis albiventris* in Mato Grosso do Sul. [63]
- Infection has been identified in wild canids in Belo Horizonte: bush dog (*Speothos venaticus*), hoary zorro (*Lycalopex vetulus*), crab-eating fox (*Cerdocyon thous*) [64] and maned wolf (*Chrysocyon brachyurus*). [65]
- Infection has also been identified in wild and captive felids (jaguars and pumas). [66]
- There is evidence for infection by *Leishmania (Leishmania) amazonensis* and *Leishmania (Leishmania) infantum chagasi* in Brazilian bats. [67]
- There is no evidence for infection of capybaras (*Hydrochoerus hydrochaeris*) in Sao Paulo (2009 publication) [68] ; and anteaters (*Tamandua tetradactyla*) in the Amazon basin. [69]
- Evidence for natural infection has been found in a variety of captive primates in Belo Horizonte: a black-fronted titi (*Callicebus nigrifrons*), a howler monkey (*Alouatta guariba*), golden-bellied capuchins (*Cebus xanthosternos*), a golden-headed lion tamarin (*Leontopithecus crysomelas*), a black-headed owl monkey (*Aotus nigriceps*), Rio Tapajos sakis (*Pithecia irrorata*) and emperor tamarins (*Saguinus imperator*). [70]
- 23% of fleas and 50% of ticks from dogs infected by *Leishmania chagasi* were found to carry the parasite (2011 publication) [71]

Canine infection rates:
- 21.4% of stray dogs and 26.2% of in domestic dogs in Fortaleza, Ceara State (2008 publication) [72]
- 36% in Jacobina (1993 publication) [73]
- 47.8% in Raposa, Maranhao State (2011 publication) [74]
- 42.5% in Anastacio, Mato Grosso do Sul (2004 publication) [75]
- 11.2% in Cuiaba, Mato Grosso (2011 publication) [76] [77]
- 22.1% in Cuiaba, Mato Grosso (2012 publication) [78]
- 2% to 5% in Belo Horizonte; 5% in Montes Claros, Minas Gerais (2002 and 2007) [79-81]
- 24.7% in Belo Horizonte, Minas Gerais (2011 publication) [82]
- 7.8% in Belo Horizonte, Minas Gerais (2010 publication) [83]
- 23.47% in northern Minas Gerais (positive by at least one test, 2008 to 2009) [84]
- 77.0% to 77.5% of asymptomatic and 87.5% to 95.0% of symptomatic dogs in Minas Gerais (conjunctival PCR, 2012

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publication) [85]
- 46% in the Krenak indigenous community, Minas Gerais (2007) [86]
- 23.3% in Para State (2010 publication) [87]
- 78% in Piaui (2011 publication) [88] Also see reference [89]
- 4.2% of domiciled dogs in Paracatu (2010 publication) [90]
- 0.0027% (a single dog) in Parana State (2010 publication) [91]
- 6% to 20% in various areas of Parana (2007 publication) [92]
- 40.3% in Pernambuco (2006 publication) [93]
- 16% in Garanhuns, Pernambuco (2010 publication) [94]
- 4.7% (*L. infantum*) in Sao Paulo (2007 publication) [95]
- 1% of sick dogs in Sao Paulo State (2007 publication) [96]
- 0% in rural Botucatu, Sao Paulo State (2011 publication) [97]
- 3.79% in Sao Paulo (1999 publication) [98]
- 64.7% of euthanized dogs in Ilha Solteira, Sao Paulo (2009 publication) [99]
- 57% in Pancas, Espirito Santo (2003 to 2004) [100]
- 13% in Vitoria, Espirito Santo (2012 publication) [101]
- 58.5% in the rural northeast (2010 publication) [102]
- 18% in Lago Norte, Federal District (2008) [103]
- 4.6% in Rio de Janeiro in 1984; 1.6% in 2008. [104]
- 2.9% in Rio de Janeiro (2011 publication) [105]
- 27.9% in Monte Negro, Rondonia (2010 publication) [106]
- 40% in Salvador (spleen PRC, 2011 publication) [107]

Vectors:
The etiologic agent of visceral leishmaniasis is *Leishmania chagasi* (*L. infantum*), and the local vector is *Lutzomyia longipalpis*. [108-114]
- *Lu. forattinii* has also been implicated in Mato Grosso do Sul; and *Lu. whitmani* in Parana state; and *Lu. corelezzi* in Santa Luzia, Minas Gerais state [115]
- *Lutzomyia longipalpis* (95%) was the most common vector identified in Mato Grosso do Sul State; trypanosomoids were detected in 10.39%. *Brumptomyia avellari*, *Evandromyia cortelezzii*, *Evandromyia lenti* and *Nyssomyia whitmani* were also found. (2003) [116]
- *Lutzomyia longipalpis* was identified in a coastal area of Rio de Janeiro (2008 to 2009) [117]
- 1.1% of female sandflies (all *Lu. longipalpis*) carry *Leishmania* species (Teresina, Piaui, 2004 to 2005) [118]
- *Lutzomyia complexa* is the most common sandfly species in Sao Vicente Ferrer, followed by *Lu. migonei*. (northern Zona da Mata, Pernambuco State, 2002 to 2003) [119]
- 1.5% of *Lu. cruzi* and 0.7% of *Lu. forattinii* sandflies were infected with *L. chagasi* (*L. infantum*). (Corumba, Mato Grosso do Sul, 2006) [120] *Lu. almerioi* has also been identified as a vector in the area. [121]
- 1.6% of *Lutzomyia longipalpis* in Raposa, Maranhao State were found to be infected (2011 publication) [122]
- Vectors in Maranhao include *L. longipalpis*, *L. evandroi*, *L. lenti*, *L. termitophila*, *L. flaviscutellata*, *L. migonei*, *L. infraspinosa*, *L. sordellii*, *L. wellcomei*, *L. antunesi*, and *L. trinidadensis*. [123]
- Vectors in Manaus include *Lutzomyia umbritalis*, *Lutzomyia anduzei* and *Lutzomyia claustrei*. [124]
- Sand fly species identified in Minas Gerais state include: *Brumptomyia brumpti*, *Lutzomyia bacula*, *Lutzomyia cortelezzii*, *Lutzomyia lenti*, *Lutzomyia sallesi*, *Lutzomyia longipalpis*, *Lutzomyia migonei*, *Lutzomyia intermedia*, *Lutzomyia neivai*, *Lutzomyia whitmani*, *Lutzomyia christenseni*, *Lutzomyia monticola*, *Lutzomyia pessoai*, *Lutzomyia aragaoi*, *Lutzomyia brasiliensis*, *Lutzomyia lutziana and Lutzomyia sordellii*. [125]
- Vectors identified in Espirito Santo do Pinhal include *Nyssomyia whitmani* and *Pintomyia pessoai* in the rural area, *Lutzomyia longipalpis* and *Ny. whitmani* in the periurban area and *Lu. longipalpis* in the urban area. [126]
- 3.9% of *Lu. longipalpis* in Janauba, Minas Gerais were found to carry *Leishmania infantum chagasi* (2011 publication) [127]
- *Lutzomyia cruzi* has been identified as a vector in Jaciara, Mato Grosso. [128]
- *Migonemyia migonei* has been implicated as a vector in Pernambuco. [129]
- 1.9% of phlebotomine sandflies (99.15% *Lu. longipalpis*) carry *Leishmania chagasi*. (Mato Grosso do Sul, 2005 to 2006) [130]
- *Viannamya furcata*, *Lutzomyia neivai* and *Lutzomyia sallesi* have also been implicated. [131]
- *Leishmania infantum* was found in 19% of *Lutzomyia longipalpis*, 3.8% of *Nyssomyia whitmani*, 33.3% of *Evandromyia termitophila* and *Nyssomyia intermedia* (southeastern Brazil, 2006 to 2007) [132]
- *Leishmania infantum / chagasi* has been identified in ticks *Rhipicephalus sanguineus* in Italy and Brazil. [133] [134]

Notable outbreaks:
1964 (publication year) - An outbreak of visceral leishmaniasis was reported in Bahia. [135]
1980 to 1986 - An outbreak was reported in Piaui. [136]

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1981 to 1985 - An outbreak (1,000 cases) was reported in Terasina. [137]
1986 (publication year) - An outbreak was reported in Santarem, Para State. [138]
1993 to 1996 - An outbreak (1,200 cases) was reported in Terasina. [139]
1994 (publication year) - An outbreak (316 cases) was reported in Natal. [140]
2008 - An outbreak (17 cases, 6 fatal) was reported in Rondonopolis, Mato Grosso. [141]

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Leprosy

Agent	BACTERIUM. Mycobacterium leprae An acid-fast bacillus
Reservoir	Human ? Armadillo
Vector	None
Vehicle	Patient secretions
Incubation Period	3y - 5y (range 3m - 40y)
Diagnostic Tests	Visualization of organisms in exudate, scrapings or biopsy. Nucleic acid amplification.
Typical Adult Therapy	Multibacillary: One year therapy Dapsone 100 mg + Clofazimine 50 mg daily; and, Rifampin 600 mg + Clofazimine 300 mg once monthly Paucibacillary: Six month therapy Dapsone 100 mg daily; and Rifampin 600 mg once monthly
Typical Pediatric Therapy	Multibacillary: One year therapy Dapsone 1 to 2 mg/kg + Clofazimine 1 mg/kg daily; and, Rifampin 10 mg/kg + Clofazimine 1 mg/kg once monthly Paucibacillary: Six month therapy Dapsone 1 to 2 mg/kg daily; and Rifampin 10 mg/kg once monthly
Clinical Hints	Anesthetic, circinate hypopigmented skin lesions and thickened peripheral nerves (tuberculoid leprosy); or diffuse, destructive papulonodular infection (lepromatous leprosy); or combined/ intermediate forms.
Synonyms	Aussatz, Doence de Hansen, Hansen's disease, Lebbra, Lepra, Mycobacterium leprae, Mycobacterium lepromatosis. ICD9: 030 ICD10: A30

Clinical

WHO Case definition for surveillance:
Clinical description
• The clinical manifestations of the disease vary in a continuous spectrum between the two polar forms, lepromatous and tuberculoid leprosy:
• In lepromatous (multibacillary) leprosy, nodules, papules, macules and diffuse infiltrations are bilateral symmetrical and usually numerous and extensive; involvement of the nasal mucosa may lead to crusting, obstructed breathing and epistaxis; ocular involvement leads to iritis and keratitis
• In tuberculoid (paucibacillary) leprosy, skin lesions are single or few, sharply demarcated, anesthetic or hypoesthesic, and bilateral asymmetrical, involvement of peripheral nerves tends to be severe
• Borderline leprosy has features of both polar forms and is more labile
• Indeterminate leprosy is characterized by hypopigmented maculae with ill-defined borders; if untreated, it may progress to tuberculoid, borderline
or lepromatous disease
Laboratory criteria for confirmation
• Alcohol-acid-fast bacilli in skin smears (made by the scrape-incision method).
• In the paucibacillary form the bacilli may be so few that they are not demonstrable.
• In view of the increasing prevalence of HIV and hepatitis B infection in many countries where leprosy remains endemic, the number of skin smear sites and the frequency of smear collection should be limited to the
minimum necessary.

Case classification:
WHO operational definition:
A case of leprosy is defined as a person showing one or more of the following features, and who as yet has to complete a full course of treatment:
• hypopigmented or reddish skin lesions with definite loss of sensation
• involvement of the peripheral nerves, as demonstrated by definite thickening with loss of sensation
• skin smear positive for acid-fast bacilli
Classification (microbiological):
Paucibacillary (PB): includes all smear-negative cases
Multibacillary (MB): includes all smear-positive cases.
Classification (clinical):
Paucibacillary single lesion leprosy: 1 skin lesion.
Paucibacillary leprosy: 2 to 5 patches or lesions on the skin.
Multibacillary leprosy: >5 patches or lesions on the skin.

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The major forms of leprosy are as follows: [1] [2]

Tuberculoid • one or a few well-demarcated, hypopigmented, and anesthetic skin lesions, frequently with active, spreading edges and a clearing center; peripheral nerve swelling or thickening also may occur.

Lepromatous • a number of erythematous papules and nodules or an infiltration of the face (including oral mucosa [3] , hands, and feet with lesions in a bilateral and symmetrical distribution that progress to thickening of the skin.

Borderline (dimorphous) • skin lesions characteristic of both the tuberculoid and lepromatous forms.

Indeterminate • early lesions, usually hypopigmented macules, without developed tuberculoid or lepromatous features.

Relapsing disease may manifest as lymphadenopathy mimicking tuberculosis [4]

• Relapses may follow effective antimicrobial therapy. [5-7]

The skin lesions of paracoccidioidomycosis may mimic those of tuberculoid leprosy. [8]

• Lepromatous leprosy may mimic sarcoidosis. [9] [10]

• Lupus vulgaris may mimic actinomycosis or mycetoma. [11]

• Diffuse cutaneous leishmaniasis may mimic lepromatous leprosy [12] The lesions of both cutaneous and mucocutaneous leishmaniasis could be mistaken for those of borderline tuberculoid leprosy. [13]

• Leprosy may be initially misdiagnosed as adult stills disease or an auto-immune disorder. [14]

Leprosy may be associated with endocrine dysfunction including hypogonadism, sterility and osteoporosis [15] [16]

Six percent of leprosy patients exhibit rheumatological manifestations, most commonly resembling rheumatoid arthritis [17] [18]

• Rare instances of spondylodiscitis have been reported. [19]

Lucio's phenomenon is a rare and aggressive necrotizing variant of erythema nodosum leprosum that classically occur in patients with undiagnosed, diffuse non-nodular lepromatous leprosy. [20]

Erythema multiforme is occasionally encountered among patients with leprosy. [21]

Chronic skin lesions may undergo malignant transformation. [22]

Cranial nerve involvement, most often trigeminal and facial, is not uncommon. [23]

• Segmental necrotizing granulomatous neuritis is reported in some cases. [24]

This disease is endemic or potentially endemic to all countries.

Leprosy in Brazil

As of 2003, Brazil has the second highest number of leprosy cases (after India), and accounts for 94% of all cases in Latin America. [25]

- Although India continues to account for over 50 per cent of the global leprosy burden, disease rates per 100,000 population in Brazil have exceeded those in India since 2004. [26]

Highest rates of leprosy occur in the Amazon [27] , midwest, southeast, south and northeast.

- The rate of leprosy in Mossoro, Rio Grande do Norte state increased from 27.8 per 100,000 population in 1998, to 51.4 per 100,000 in 2004 . [28]

- As of 2009, A highly endemic region was identified in the area of Maranhao, Para, Tocantins and Piaui - accounting for only 5.2% of the Brazilian population, but 20% of new leprosy cases. [29]

For every 10 new cases of leprosy, there are at least 4 undiagnosed cases in household contacts. (Mato Grosso state, 1999) [30]

- 2% of household contacts develop leprosy over a 5-year period. (Uberlandia, 2007 publication) [31]

- 20.5% of leprosy contacts in Minas Gerais are found to have leprosy (2009 publication) [32]

- Contact with armadillos has been identified as a risk factor for infection in Brazil. [33]

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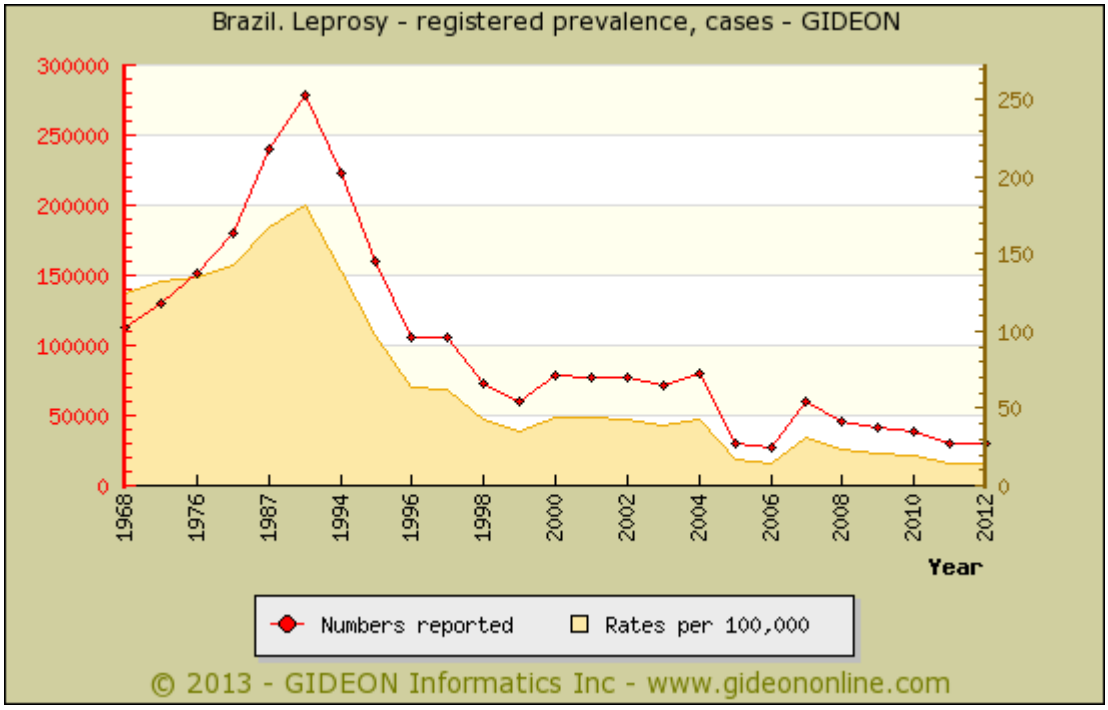

Graph: Brazil. Leprosy - registered prevalence, cases

Notes:

1. Additional references: 1997 [34]
Individual years:
1976 - True number estimated at 242,273 cases.
1981 - True number estimated at 315,665 cases (260 per 100,000).
1993 - True number estimated at 283,000 cases (181.6 per 100,000.
1996 - 46.75% of cases were multibacillary. Rates in Amazonas were 312 per 100,000.
1997 - 42.7% of cases were multibacillary.
1998 - Brazil accounted for 27% of the world's leprosy cases outside of India.
2000 - 147.6 per 100,000 in Mato Grosso, 107.8 per 100,000 in Para, 83.1 per 100,000 in Rondonia and 83.0 per 100,000 in Maranao.

Disease detection rates per 100,000 in the Amazon region: 84.8 in 2001; 87.9 in 2002; 88.0 in 2003; 83.8 in 2004; 78.0 in 2005; 61.9 in 2006. [35] [36]

- Leprosy rates in Amazonas State have declined by 1% (age group 60 to 69) to 9% (age group 0 to 4 years) during 19890 to 2009. [37]

- Detection rate per 10,000 in Autazes, Eirunepe, and Sao Gabriel da Cachoeira provinces) were 3.55, 14.94, and 2.13/ 10,000 (among non-indigenous peoples); and 10.95, 1.93, and 0.78/10,000 (among indigenous peoples) (2000 to 2005) [38]

- In 2003, the rate in Paracatu district, Minas Gerais was 68 per 100,000. [39]

- In 2012 (publication year) 4% of school children in hyperendemic Amazon communities were found to have leprosy. [40]

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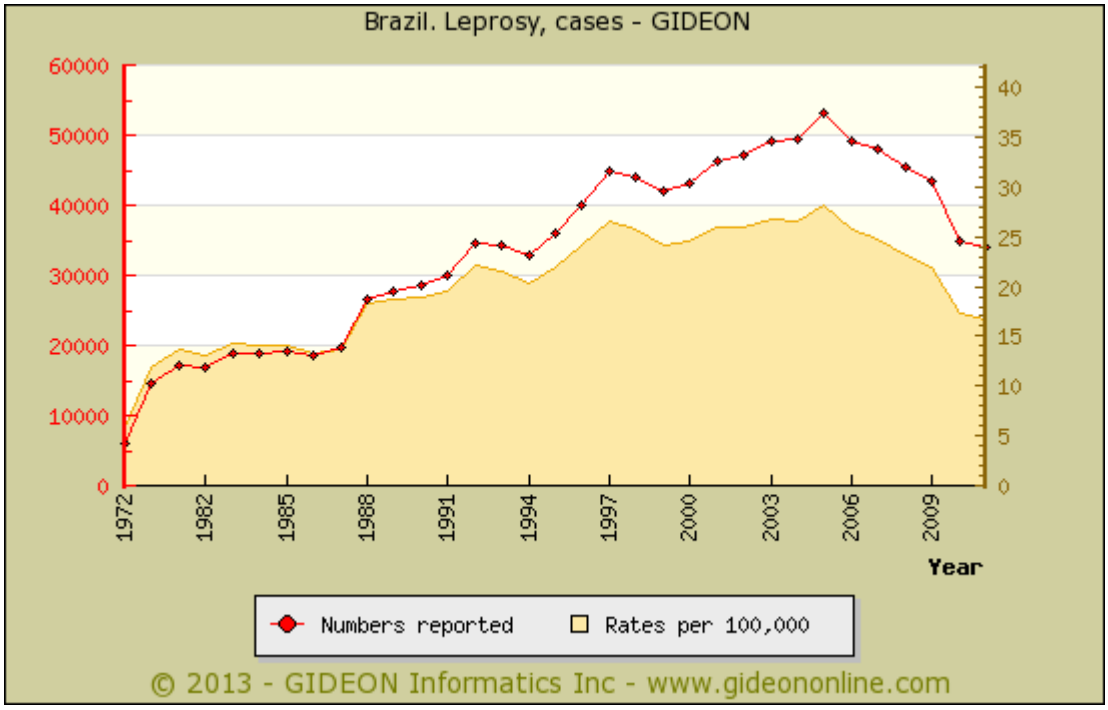

Graph: Brazil. Leprosy, cases

Notes:
1. 604,073 new cases were reported during 1980 to 2000.
2. 1996 - Highest rate in Rondonia (194 per 100,000).

Geographic notes:

- 105 cases (106.06 per 100,000) were detected in 12 municipalities in Tocantins State through active case finding in 1999.
- 6 cases were reported among stem-cell transplant recipients in Sao Paulo (2007 publication) [41]
  Leprosy was detected in 5.2% of persons examined in Sao Paulo during 2010. [42]
  The leprosy rate in Sao Jose do Rio Preto was 10.4 per 100,000 during 1998 to 2007. [43]
- 20 cases (136 per 100,000) were detected among school students in Buriticupu, Maranhao State, using active case finding. (2007 publication) [44]
  A screening study reported that leprosy was present in 0.4023% of the population of Maranhao (2005 to 2007). [45]
  A screening study reported that leprosy was present in 0.21984% of the population of Buriticupu, Maranhao (2008 to 2010) [46]
- 474 cases were detected in Metropolitan Manaus during 1998 to 2005 - 10.4% in children below age 15. [47]
- 455 cases were reported in Uberaba, Minas Gerais, during 2000 to 2006. [48] 19,283 patients with leprosy were registered in Minas Gerais during 2000 to 2005. [49]
- 1,124 cases were treated at a university hospital in Brasilia during 1985 to 2005. [50]
- 595 cases of disabling leprosy were reported from Belo Horizonte during 1993 to 2003 [51]
- 12,238 cases (54 per 100,000) were reported in Piaui State during 2003 to 2008 - 85% of these classified as new cases. [52]
  During 2001 to 2008, Teresina reported a rate of new cases of 96.21 per 100,000, and a disease prevalence of 80 to 110 per 100,000. [53]
- Analysis of 1,353 cases treated in Rio de Janeiro and 336 in Duque de Caxia during 1986 to 2008 - see reference [54]

Candida albicans was found in the mouths of 65.8% of patients with lepromatous leprosy, vs. 47.4% of controls (Sao Paulo, 2009 publication) [55]

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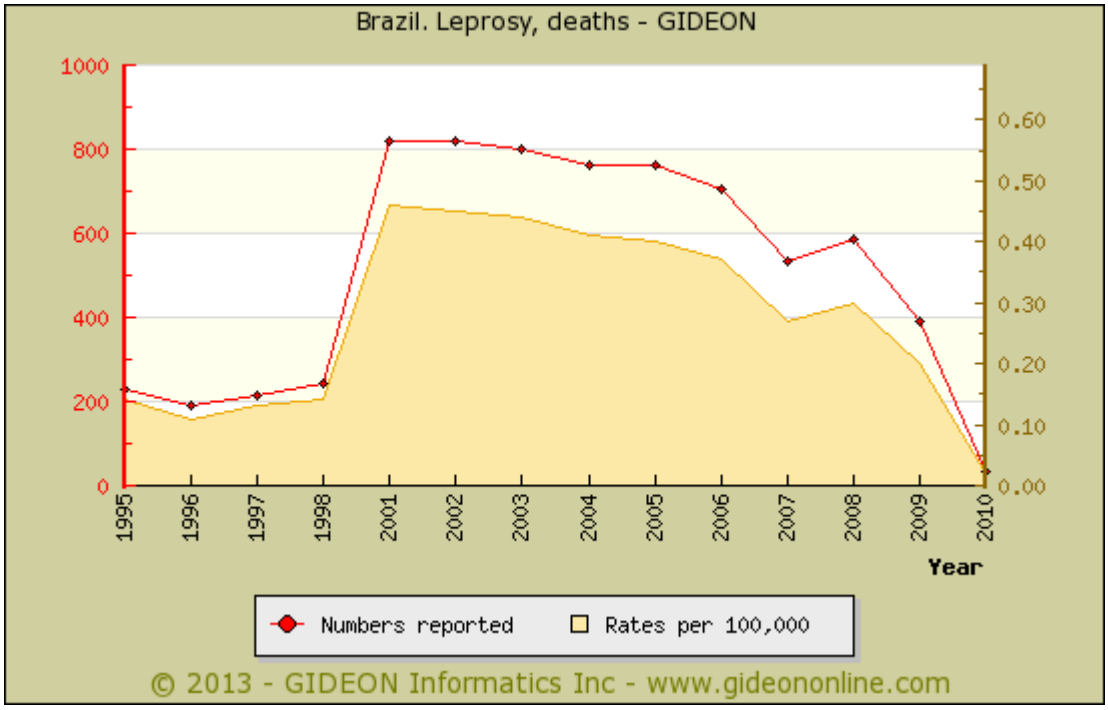

Graph: Brazil. Leprosy, deaths

Notes:
1. 877 deaths were ascribed to leprosy during 1991 to 1994.

Mycobacterium leprae was identified in 21% of armadillos (*Dasypus novemcinctus* and *Euphractus sexcinctus*) in Ceara (2012 publication) [56]

MDT coverage is 100% (1998).

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Leptospirosis

Agent	BACTERIUM. Leptospira interrogans An aerobic non-gram staining spirochete
Reservoir	Cattle Dog Horse Deer Rodent Fox Marine mammal Cat Marsupial Frog
Vector	None
Vehicle	Water Soil urine contact
Incubation Period	7d - 12d (range 2d - 26d)
Diagnostic Tests	Culture on specialized media. Dark field microscopy of urine, CSF. Serology.
Typical Adult Therapy	Doxycycline 100 mg BID X 5 to 7d
Typical Pediatric Therapy	Age >= 8y: Doxycycline 2.2 mg/kg BID X 5 to 7d. Age < 8y: IV Penicillin G 50,000u/kg q6h X 5 to 7d
Clinical Hints	"Sterile" meningitis, nephritis, hepatitis, myositis and conjunctivitis; often follows recent skin contact with fresh water in rural or rodent-infested areas; case-fatality rates of 5% to 40% are reported.
Synonyms	Andaman hemorrhagic fever, Canefield fever, Canicola fever, Field fever, Fish handler's disease, Fort Bragg fever, Japanese autumnal fever, Leptospira, Leptospirose, Leptospirosen, Leptospirosi, Mud fever, Pre-tibial fever, Rat fever, Rice field fever, Swamp fever, Swineherd disease, Weil's disease. ICD9: 100 ICD10: A27

Clinical

WHO Case definition for surveillance:
Clinical description
Acute febrile illness with headache, myalgia and prostration associated with any of the following symptoms:
• conjunctival suffusion
• meningeal irritation
• anuria or oliguria and/or proteinuria
• jaundice
• hemorrhages (from the intestines; lung bleeding is notorious in some areas)
• cardiac arrhythmia or failure
• skin rash
and a history of exposure to infected animals or an environment contaminated with animal urine.
Other common symptoms include nausea, vomiting, abdominal pain, diarrhea, arthralgia.
Laboratory criteria for diagnosis
• Isolation (and typing) from blood or other clinical materials through culture of pathogenic leptospires
• Positive serology, preferably Microscopic Agglutination Test (MAT), using a range of *Leptospira* strains for antigens that should be representative of local strains
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: Not applicable.
• Confirmed: A suspect case that is confirmed in a competent laboratory.
Note: Leptospirosis is difficult to diagnose clinically in areas where diseases with symptoms similar to those of leptospirosis occur frequently.
SPECIAL ASPECTS
• Serology by Microscopic Agglutination Test (MAT) may provide presumptive information on causative serogroups.
• Attempts should be made to isolate leptospires, and isolates should be typed to assess locally circulating serovars.
• Questioning the patient may provide clues to infection source and transmission conditions.
• Animal serology may give presumptive information on serogroup status of the infection Isolation followed by typing gives definite information on serovar.

Disease due to *Leptospira interrogans* serovar. *icterohaemorrhagiae* is usually overt, and often manifest as hepatitis, meningitis and nephritis. [1]
• Canicola fever is due to serovar. *canicola* (occasionally *L. interrogans* serovar. *pomona*) and characterized by a milder lymphocytic meningitis, without hepatic or renal involvement.
• Serovar. *autumnalis* (occasionally *L. interrogans* serovar. *pomona*) produces Fort Bragg fever, a febrile illness associated with raised, erythematous, and mildly tender pretibial skin lesions.

Acute phase

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Subclinical infection is common.
• Overt leptospirosis (90% of cases) is characterized by a self-limited, systemic illness.
• Patients are at risk for severe and potentially fatal illness which may present with renal failure, liver failure, pneumonia [2] [3] or hemorrhagic diathesis. [4]
• Illness begins abruptly with such symptoms as fever (38 to 40 C), headache (over 95% of cases), rigors, myalgia (over 80%), conjunctivitis (30 to 40%), abdominal pain (30%), vomiting (30 to 60%), diarrhea (15 to 30%), cough, muscular (calf) tenderness, pharyngitis (20%) and a pretibial maculopapular rash (fewer than 10%).
• Additional findings may include lymphadenopathy, splenomegaly, atypical lymphocytosis [5] , thrombocytopenia, transitory paraparesis [6] , hepatomegaly or pancreatitis. [7]
• During the acute illness, bacteria can be recovered from or seen in blood, CSF, or tissue using specialized techniques.
• Organisms are demonstrated in urine after the 5th to 7th days. Pyuria, hematuria and proteinuria may be evident as well.
• Severe hypomagnesemia has been reported during the acute phase of infection. [8-10]

Latency and relapse:
The acute phase is followed by an asymptomatic period of 4 to 30 days.
• At this point, illness reappears, with conjunctival suffusion, photophobia, eye pain, myalgia, lymphadenopathy and hepatosplenomegaly.
• Additional findings may lymphocytic meningitis (70 to 80% of patients) with normal glucose levels; pretibial purpura, uveitis [11] [12] , iridocyclitis or chorioretinitis, facial nerve palsy [13] , thrombocytopenia, hypotension, myopericarditis [14] and pancreatitis. [15]
• Weil's disease is characterized by hepatic and renal function which may progress to severe and even fatal hepatorenal failure which carries a case-fatality rate of 5 to 40%.
• Renal involvement, principally interstitial nephritis and tubular necrosis [16] may be severe, even in the absence of jaundice. [17]
• Pulmonary infiltrates [18] , severe hemorrhagic pneumonia [19] [20] and acute pulmonary distress syndrome may be encountered, even in the absence of hepatic and renal failure. [21-23]
• Congestive heart failure is rare; however, cardiac arrhythmias may occur and result in sudden deaths.
• Acute disseminated encephalomyelitis has been reported as a complication of leptospirosis. [24]
• Relatively severe infection is reported among pregnant women. [25]

Persistent, asymptomatic renal colonization by Leptospirae may follow infection in humans. [26]

The clinical features of dengue [27] , influenza pneumonia [28] and pyomyositis may mimic those of leptospirosis. [29]

This disease is endemic or potentially endemic to all countries.

Leptospirosis in Brazil

Time and Place:
- The first cases of leptospirosis in South America were reported in Para, in 1911. [30]
- Urban epidemics have been reported during the rainy season since the 1970's.

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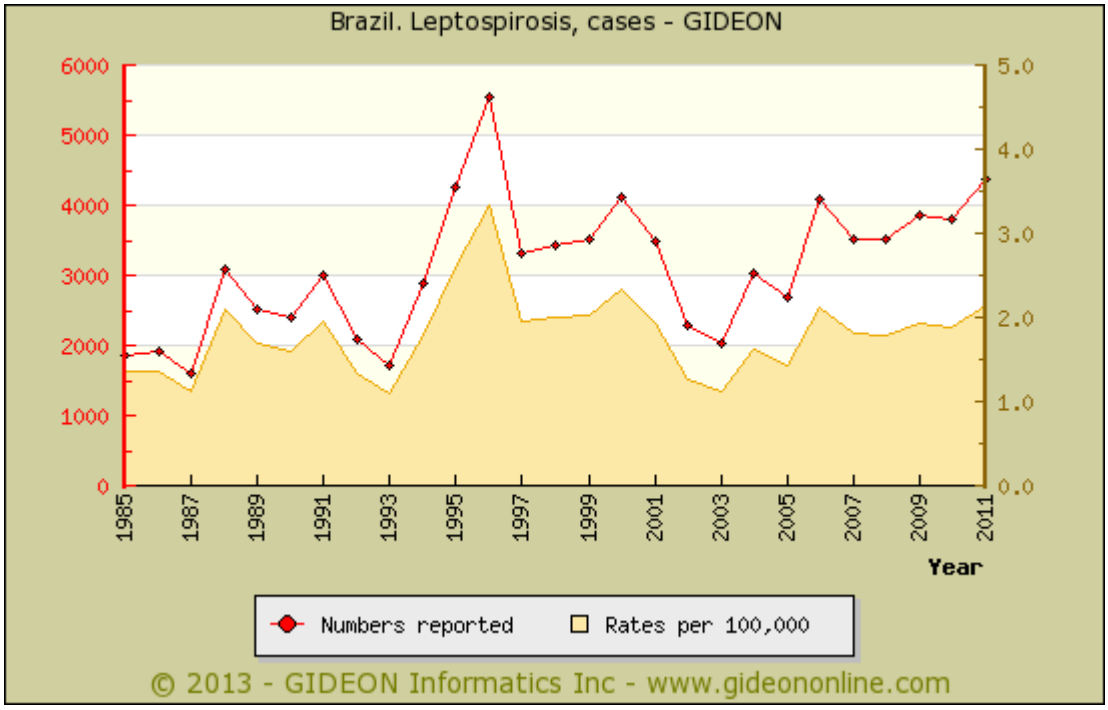

Graph: Brazil. Leptospirosis, cases

Notes:
1. 42,652 cases were reported during 1987 to 2000 - including 8,446 in Sao Paulo and 7,278 in Rio de Janeiro.

2,237 cases were confirmed in Sao Paulo City during 1947 to 1972 - 86.5% due to serovar. *icterohaemorrhagiae*.
- 9,335 cases were confirmed in Sao Paulo during 1969 to 1997 (mean 0.53 per 100,000 per year), with peak incidence during January to April. [31]
- 285 cases (42 fatal) were reported in 2004; 262 (28) in 2005; 167 (31) in 2006

279 cases were reported in Recife in 1966 and 1970, following flooding - 92.5% of these due to serovar. *icterohaemorrhagiae*.

665 cases(35 fatal) were reported in Manaus, Amazonas during 2000 to 2010. [32]

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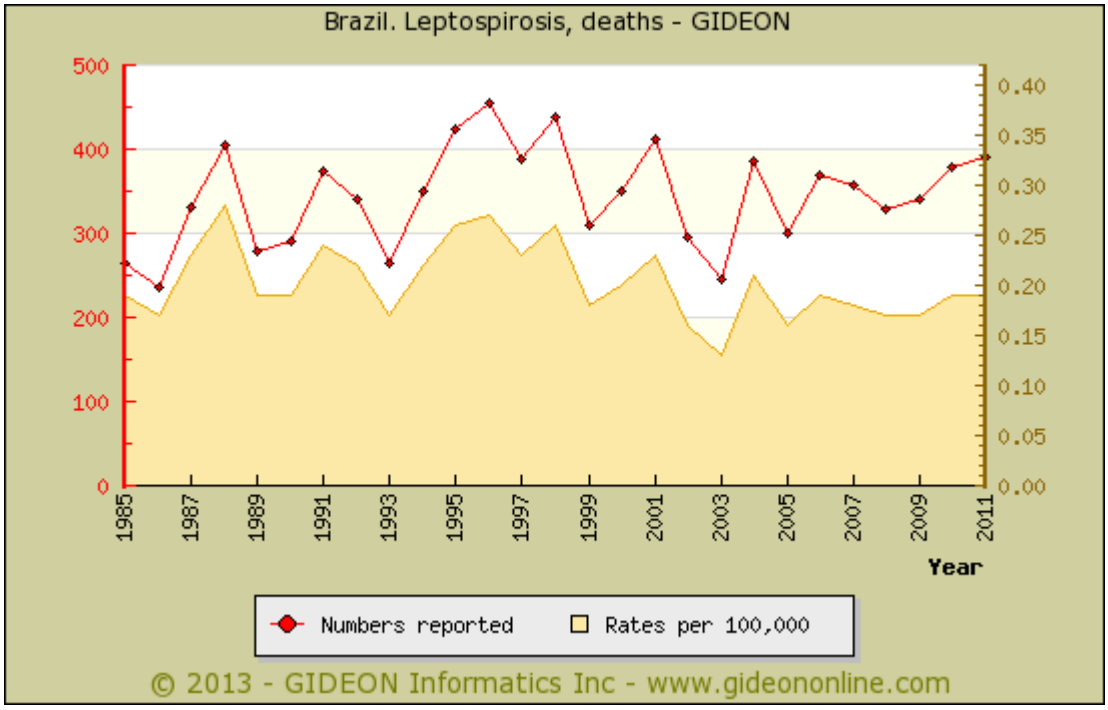

Graph: Brazil. Leptospirosis, deaths

Notes:
1. 2,966 fatal cases were reported during 1986 to 1995.

Leptospirosis-associated severe pulmonary hemorrhagic syndrome (SPHS) emerged in the slum communities of Salvador, with 47 cases (74% fatal) during 2003 through 2005. [33]
- By 2005, SPHS was responsible for 55% of the deaths due to leptospirosis in the area.

Prevalence surveys:

13.9% of rubella-negative dengue-like illness (Federal District, 2008) [34]
33.33% of CSF samples from aseptic meningitis cases, which were negative by routine microbiological methods (2011 publication) [35]
4.5% of sheep at slaughter (kidney and liver biopsies, 2012 publication) [36]
39% of rats in Rio de Janeiro

Seroprevalence surveys:

15.9% of patients with suspected dengue, and 9% of patients with suspected hepatitis (Mato Grosso do Sul, 2007 publication) [37]
4.0% of slaughterhouse workers in northern Para (2006 publication) [38]
15.2% of individuals in a farming community (Rio Grande do Norte, 2001) [39]
12.4% of persons tested in a random survey (Salvador, 2007 publication) [40]
15.4% of slum residents (Salvador, 2008 publication) [41]
10.5% of stray dogs in Itapema, Santa Catarina state (2005 publication) [42]
7.7% of dogs (RMAT, 1970 to 1973) [43]
7.1% of dogs in Ilheus, Bahia (2012 publication) [44]
16.1% of *Cebus apella* (serovars *pomona*, brasiliensis, mini, szwajizak, *grippotyphosa*, sarmin, fluminense, autumnalis, *hebdomadis*, guaratuba, javanica and *icterohaemorrhagiae*) in Tocantins (2002) [45]
2.4% of Alouatta caraya (serovars mangus and fluminense) in Tocantins; 12.9% of *Nasua nasua* (serovars fluminense and javanica); 20% of *Cerdocyon thous* (serovars fluminense and brasiliensis) (2002)
56.8% of captive nonhuman primates in Salvador (2011 publication) [46]
20.8% of adult dairy goats (Rio de Janeiro region, 2007 publication) [47]
43.6% of dairy goat flocks in northeastern Brazil (2012 publication) [48]
25.9% of goats and 47.4% of sheep in Rio de Janeiro (2011 publication) [49]
3% of ewes in the Federal District (2010 publication) [50]
66.9% of chronically ill urban dray horses (Londrina municipality, 1996 to 2005) [51]
16.1% of farmed pigs in Alagoas (2012 publication) [52]

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68.1% of domestic rats in Salvador - 80.3% are culture-positive (2008 publication) [53]

9.68% of nine-banded armadillos (*Dasypus novemcinctus*) and 33.3% of six-banded armadillos (*Euphractus sexcinctus*) (Sao Paulo, 2008 publication) [54]

36.17% of rats (*Rattus norvegicus* in Duque de Caxias, Rio de Janeiro State - most toward *Leptospira interrogans* serovar icterohaemorrhagiae (1993 publication) [55]

0% of bats in Sao Paulo (2008) [56]

7.8% of vampire bats (*Desmodus rotundus*) in Botucatu region, Sao Paulo (2009 publication)

42.7% of wild carnivores, 17.5% of domestic dogs and 74.1% of horses in the northern Pantanal (2002 to 2006) [57]

3.5% of captive neotropical felids in Foz do Iguacu, Parana (2012 publication) [58]

70% of wild white-lipped peccaries (*Tayassu pecari*) in the Brazilian Pantanal (2003 to 2005) [59]

0% of free-ranging Brazilian tapirs (*Tapirus terrestris*) in Goias state (2010 publication) [60]

6% of pampas deer (*Ozotoceros bezoarticus*) in the Pantanal wetlands of Mato Grosso do Sul, Brazil (2011 publication) [61]

31.1% of captive manatees (*Trichechus inunguis*) in northern Brazil (2012 publication) [62]

Reservoirs:

- The principal hosts in Brazil are rodents, pigs and dogs.
- Infection is also reported in dogs, monkeys, goats, horses, pigs, armadillos, bats, peccaries, tapirs, deer and manatees (see above).
- *Leptospira interrogans* serovar. *icterohaemorrhagiae* has been identified in capybara (*Hydrochaeris hydrochaeris*). [63]
- *Leptospira borgpetersenii* has been identified in white-eared opossums (*Didelphis albiventris*) in southern Brazil. [64]

Infecting serovars:

- The principal pathogens in this country are *Leptospira* serovars *icterohaemorrhagiae*, *grippotyphosa*, *canicola*, javanica, *Ballum* and *pomona*.
- Serovars *wolffi*, *andamana* and *alexi* were identified during the 1970's.
- Serovar. Hutstbridge predominates in Mato Grosso do Sol (2007 publication) [65]
- Serovar. *hardjo* is most common among cattle in the eastern Amazon area, followed by serovar. *bratislava* and serovar. *shermani*. 32.8% of exposed humans in this region are seropositive for these same species.
- Serovar. Autumnalis accounted for 34.2% of infections among dogs in Uberlandia, Minas Gerais and serovar Tarassovi 23.7%; while serovar Tarassovi predominated among humans (2008) [66]
- Seropositivity toward *L. interrogans* serovar. *hardjo*, wolffi and mini has been found among Pampas deer (*Ozotocerus bezoarticus*) from the Pantanal Matogrossense, and Parque Nacional de Emas. [67]
- Seropositivity toward serovars Copenhageni, Bratislava, Canicola and Grippotyphosa was found among dogs in Bahia. [68]
- Sporadic cases of human infection by *Leptospira noguchii* are reported. [69]

Notable outbreaks:

1987 - An outbreak (23 cases) in Sao Paulo followed swimming in a pool filled with river water. [70]

1996 - An outbreak (1,425 cases, 22 fatal) was reported in Rio de Janeiro. [71] [72]

1996 - An outbreak (326 cases, 50 fatal) was reported in Salvador through active case finding during January to August 1996 - for a local rate of 12.5 per 100,000. [73] *Leptospira interrogans* serovar. *copenhageni* was identified in 87% of cases.

2000 - An outbreak (157 cases, 13 fatal) was reported in Salvador - for a local rate of 6.8 per 100,000. [74]

2009 to 2010 - An outbreak was reported in a goat herd with reproductive failure. [75]

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Listeriosis

Agent	BACTERIUM. Listeria monocytogenes A facultative gram-positive bacillus
Reservoir	Mammal Human Bird Soil Water
Vector	None
Vehicle	Transplacental Dairy products (eg, soft cheeses), Infected secretions Vegetables Poultry Water
Incubation Period	3d - 21d (-60d post-ingestion)
Diagnostic Tests	Culture of blood or CSF.
Typical Adult Therapy	Ampicillin 2g IV q6h X 2w (higher dosage in meningitis) + Gentamicin. Sulfamethoxazole/ trimethoprim recommended for Penicillin-allergic patients
Typical Pediatric Therapy	Ampicillin 50 mg/kg IV Q6h X 2w (higher dosage in meningitis). Sulfamethoxazole/trimethoprim recommended for Penicillin-allergic patients
Clinical Hints	Meningitis or sepsis, often immune-suppressed patients (lymphoma, AIDS, etc); gastroenteritis - may follow ingestion of 'over-the-counter' foods; neonatal septicemia occasionally encountered.
Synonyms	Listeria monocytogenes, Listeriose, Listeriosi. ICD9: 027.0 ICD10: A32

Clinical

Major risk factors for invasive Listeriosis reflect T-cell mediated immune compromise, including old age, pregnancy, hematological malignancy, chemotherapy, corticosteroid therapy and anti-TNF-alpha agents

Signs of *Listeria* meningitis are often atypical: [1]
- brain stem and cerebellar involvement (rhombencephalitis) occurs in 11% of cases [2] [3]
- nuchal rigidity in only 80% to 85%
- movement disorders (ataxia, myoclonus) in 15% to 20% [4]
- seizures in 25%. [5]

The blood culture is positive in 75% of meningitis cases; and the cerebrospinal fluid gram stain is positive in only 40%.

Symptoms of food-borne listeriosis develop between one day and three months after ingestion the bacteria in food. [6]
- Most cases are characterized by diarrhea and fever [7] [8]
- Headache, myalgia and arthralgia are common. [9]
- The bacteria may be excreted in stool for several months.

Other forms of listeriosis:
- Hepatic listeriosis may present as single or multiple abscesses, or diffuse granulomatous hepatitis. [10]
- Numerous cases of *Listeria* endocarditis of both native and prosthetic valves have been reported. [11-25] Instances of pericarditis [26] , cardiac pseudotumor [27] , and aortitis with aortic dissection have also been reported. [28]
- Sporadic cases of prosthetic joint infection [29-32] , renal failure, brain abscess [33] , cutaneous infection [34] , mycotic aortic aneurysm [35] [36] , pericarditis [37] , uveitis [38] , cholecystitis [39] [40] and rhabdomyolysis have been reported. [41]
- *Listeria* peritonitis has been reported in a patient undergoing peritoneal dialysis [42] and in a patient with biliary cirrhosis. [43] Spontaneous bacterial peritonitis due to *Listeria monocytogenes* [44-48] and infection of a ventriculo-peritoneal shunt have also been reported. [49]

This disease is endemic or potentially endemic to all countries.

Listeriosis in Brazil

Ten cases were reported in 1990.

Prevalence surveys:

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10.68% of retail cheeses and 41.2% of homemade (Minas Frescal) cheeses in Rio de Janeiro (1998 publication) [50]

13.3% and 9.6% of samples from two cheese manufacturing plants (Sao Paulo, 2008 to 2009) [51]

0% of pasteurized milk samples and 18% of Minas Frescal cheese products; all positive cheese samples were of the same brand. (Juiz de For a, Minas Gerais, 2008 publication) [52]

0% of raw milk samples from 4 important milk producing areas of Brazil (2008 publication) [53]

0% of raw cow's milk samples in Minas Gerais (2012 population) [54]

35.6% of poultry samples at a processing plant (southern Brazil, 2005 publication) [55]

42.5% of fully-cooked ham sold in supermarkets (*Listeria monocytogenes*, Forteleza, 2011 publication) [56]

0.6% of minimally processed leafy salads collected from retailers (Sao Paulo, 2007 publication) [57]

3.1% of ready-to-eat vegetables marketed in Sao Paulo (2012 publication) [58]

42% of linguica - a pork product (*Listeria monocytogenes*, 2009 publication) [59]

Notable outbreaks:

2006 to 2007 - An outbreak (6 cases) of listeriosis was reported in a hospital in Rio de Janeiro. [60]

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Liver abscess - bacterial

Agent	BACTERIUM. Various species from portal (Bacteroides, mixed aerobe-anaerobe) or biliary (Escherichia coli, etc) source
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Ultrasonography, CT or radionucleotide scan. If amoebic abscess suspected, perform Entamoeba serology
Typical Adult Therapy	Intravenous antibiotic(s) directed at likely or suspected pathogens. Percutaneous or open drainage
Typical Pediatric Therapy	As for adult
Clinical Hints	Tender liver, and prolonged fever in a patient with history of diverticulosis, cholecystitis, appendicitis, etc; clinically similar to amoebic abscess, but often multiple.
Synonyms	Ascesso fegato, Bacterial liver abscess, Hepatic abscess - bacterial, Liver abscess. ICD9: 572.0 ICD10: K75.0

Clinical

Symptoms of pyogenic hepatic abscess include fever and rigors of several days' to several weeks' duration.
• Dull right upper quadrant pain may be associated with cough and pleuritic pain with radiation to the right shoulder and an associated pleural rub. [1]
• Tender hepatomegaly is present in 50 to 70% of the patients.
• Jaundice is uncommon, unless the abscess is extensive or associated with ascending.
• In some cases, the sole presentation may be fever of unknown origin.

Serological studies, a history of diarrhea, edema of the right chest wall, and limitation to a single abscess in the posterior, superior right hepatic lobe may be suggestive of amoebic abscess. [2] [3]

Alkaline phosphatase is the most consistently elevated serum enzyme in patients with liver abscess.
• Blood cultures are positive in 50% of cases.

This disease is endemic or potentially endemic to all countries.

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Lobomycosis

Agent	FUNGUS. Lacazia (Loboa) loboi
Reservoir	Human Dolphin (Tursiops truncatus and Sotalia guianensis)
Vector	None
Vehicle	? Skin trauma
Incubation Period	1y - 2y
Diagnostic Tests	Biopsy. Note: Organism does not grow under laboratory conditions.
Typical Adult Therapy	Antifungal agents not of proven value; excision as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Spreading skin nodules and regional lymphadenopathy; may follow animal (dolphin) contact; infestation may persist for decades.
Synonyms	Keloidal blastomycosis, Lacazia loboi, Lacaziasis, Lobo's disease, Loboa loboi. ICD9: 116.2 ICD10: B48.0

Clinical

Small painless nodules develop in the skin, most commonly of the face, legs, arms and ears. [1]
• Lesions are characterized as lenticular, plaque-like or keloidal lesions slowly spread peripherally, with regional lymphadenopathy. [2]
• Older skin lesions become verrucous and may ulcerate.
• Underlying structures and viscera are not involved.
• Disseminated skin disease lasting for several decades has been reported. [3]

This disease is endemic or potentially endemic to 15 countries.

Lobomycosis in Brazil

Time and Place:
The world's first case of Lobomycosis was reported from the Amazon Valley in 1931.
- Brazil accounts for 70% of the world's cases.
- Most are reported from Amazonas (notably among the Cayabi Tribe), Mato Grosso, Para and Acre. [4]
- 135 cases were reported from the Amazon region to 1986.

A case of disseminated lobomycosis was reported in an 86 year-old Brazilian woman with a 55-year history cutaneous lesions. (2008 publication) [5]
- A case followed travel to the Amazon region (2009 publication) [6]

Prevalence surveys:
12% of bottlenose dolphins (*Tursiops truncatus*) in Laguna (1993) [7]

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Loiasis

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Loa loa
Reservoir	Human
Vector	Fly (deer fly = Chrysops)
Vehicle	None
Incubation Period	4m - 3y
Diagnostic Tests	Microfilariae in blood (take during daylight hours). Adult worm recovered. Serology. Nucleic acid amplification.
Typical Adult Therapy	Diethylcarbamazine: 50 mg PO day 1 50 mg PO TID day 2 100 mg PO TID day 3 3 mg/kg PO TID days 4 to 21. Notes: Ivermectin may cause encephalopathy if dual infection with Onchocerca. Albendazole = alternative to DEC in some cases
Typical Pediatric Therapy	As for adult
Clinical Hints	Migrating pruritic or painful subcutaneous nodules (Calibar swellings), fever and eosinophilia; adult worms may exit to subconjunctival space.
Synonyms	Calabar swellings, Filaria lacrimalis, Filaria loa, Filaria oculi humani, Filaria subconjunctivalis, Fugitive swellings, Loa loa, Microfilaria diurna. ICD9: 125.2 ICD10: B74.3

Clinical

In many cases, infected patients are asymptomatic, with eosinophilia as their sole clinical finding. [1]
• Transient areas of localized subcutaneous edema (Calabar swellings) are preceded by overlying urticaria, pain and itching for several hours. [2]
• The lesions are nonerythematous, measure 10 to 20 cm, and last for days to weeks.
• Calabar swellings are most common around joints and may be painful (74%) or painless (26%). [3]
• Often, an adult worm will pass beneath the conjunctiva, resulting in local swelling, pain and inflammation. [4]

Complications include endomyocardial fibrosis, retinopathy, encephalopathy [5] , peripheral neuropathy [6] , arthritis, pleural or peritoneal effusions [7] , hypoechogenic splenic lesions and breast calcification.

This disease is endemic or potentially endemic to 17 countries. Although Loiasis is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Loiasis in Brazil

2011 (publication year) - A Brazilian traveler acquired loiasis in Uganda. [8]
2012 (publication year) - A Brazilian traveler acquired loiasis in Cameroon. [9]

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Lyme disease

Agent	BACTERIUM. Borrelia spp. :Borrelia burgdorferi; B. afzelii and B. garinii are also encountered (in Eurasia) A microaerophilic spirochete
Reservoir	Tick Deer Rodent Bird
Vector	Tick (Ixodes, Amblyomma)
Vehicle	None
Incubation Period	7d - 14d (range 2d - 180d)
Diagnostic Tests	Serology. Nucleic acid amplification. Culture of blood and body fluids available in some laboratories.
Typical Adult Therapy	Doxycycline, Ceftriaxone, Amoxicillin or Cefuroxime Dosage, route and duration according to nature and severity of disease
Typical Pediatric Therapy	>= Age 8 years: As for adult < Age 8 years: Ceftriaxone, Cefuroxime or Amoxicillin. Dosage, route and duration according to nature and severity of disease
Vaccine	Lyme disease
Clinical Hints	Fever, circular erythematous skin lesion, arthralgia and lymphadenopathy; later meningitis or myocarditis, and eventual destructive polyarthritis; patient may recall recent tick bite.
Synonyms	Arcodermatitis chronica atrophicans, Baggio-Yoshinari syndrome, Borrelia A 14S, Borrelia afzelii, Borrelia americana, Borrelia bissettii, Borrelia burgdorferi, Borrelia carolinensis, Borrelia garinii, Borrelia lonestari, Borrelia lusitaniae, Borrelia spielmanii, Borrelia valaisiana, Borrelial lymphocytoma, Doenca de Lyme, Erythema chronicum migrans, Erythema migrans, Garin-Bujadoux-Bannwarth syndrome, LD imitator syndrome, LD-like syndrome, Lyme borreliose, Lyme borreliosis, Master's disease, Neuroborreliosis, Southern tick-associated rash illness, STARI, TAPOS, Tick-associated poly-organic syndrome. ICD9: 088.81 ICD10: A69.2

Clinical

CDC case definition for surveillance:
For surveillance purposes, the CDC (The United States Centers for Disease Control) defines Lyme disease as '..erythema migrans…>=5 cm in diameter or laboratory confirmation of infection with *Borrelia burgdorferi* and at least one objective sign of musculoskeletal, neurological or cardiovascular disease.'

European Union case definition for surveillance: [1]
In view of a wider range of clinical and bacteriological presentations, the European Union (EUCALB) definition requires preceding risk of tick exposure in addition to one or more of the following:
• erythema migrans [2]
• borrelial lymphocytoma (lymphadenosis benigna cutis • a painless bluish red nodule or plaque, usually of the ear lobe, nipple or scrotum) [3-5]
• acrodermatitis chronica atrophicans (long standing red or bluish red lesions, usually of the extensor surfaces of the extremities)
• early neurological disease (meningo-radiculoneuritis, with or without cranial nerve palsy)
• chronic neuroborreliosis (encephalitis, encephalomyelitis, radiculomyelitis)
• arthritis [6]
• carditis.

Probable late Lyme disease is defined as serologic evidence for exposure and physician-diagnosed disease in the absence of objective signs. [7]

There appears to be a specific association between *Borrelia afzelii* and acrodermatitis chronica atrophicans; and between *B. garinii* and nervous system manifestations.
• *B. garinii* infection appears to be more likely than *B. afzelii* infection to affect older patients, produce skin lesions on the trunk rather than extremities, and be associated with shorter incubation period, rapid clinical course and abnormal liver function.
• Cases of European Lyme disease are characterized by a slower-spreading and less intense erythema migrans than that seen in the American cases. Banworth's syndrome is more common in European cases than the classic form of meningitis associated with American Lyme disease. [8]

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Only 25% of patients recall a preceding tick bite.
• Co-infection by *Anaplasma phagocytophilum* is common. [9]

The clinical features of anaplasmosis are similar to those of Lyme disease; however, patients with anaplasmosis are more likely to exhibit headache, dizziness, myalgias, abdominal pain, anorexia, leukopenia, lymphopenia, thrombocytopenia, and elevated liver enzymes. [10]

Acute illness:
Typical features include low-grade fever, fatigue, headache, conjunctivitis, myalgia and arthralgia.
• The typical rash of erythema migrans [11] is present in 75% of cases and usually neither pruritic nor painful
• Multiple skin lesions may occur in 20% to 50% of cases. [12] [13]
• A nodule in the nipple or ear lobe (borrelial lymphocytoma) may be present, and appears to be more common in Europe. [14]
• Acrodermatitis chronicum atrophicans, typically seen on the hands and feet, is also more common in the European variety.
• Acropapular dermatitis has also been reported in children with Lyme disease. [15]
• Erythema annulare centrifugum is an inflammatory skin disease with incoherent conglomeration of figurate or gyrate erythemas. Lesions associated with borreliosis are positive for *Borrelia* by PCR [16]

Systemic manifestations of Lyme disease: [17]
• A wide variety of **neurological diseases** have been associated with advancing Lyme disease. [18-25]
• Some patients present with a typical Bell's palsy, which may be bilateral in 25% of cases. [26-29]
• Parsonage-Turner syndrome (acute brachial neuritis or neuralgic amyotrophy) [30] [31], Guillain-Barre and Banworth's syndrome (radiculitis with lymphocytic pleocytosis) , Horner's syndrome [32], sensorineural hearing loss [33], paralytic strabismus [34], acquired nystagmus [35], recurrent laryngeal nerve paralysis [36] [37], lower motor neuron disease [38], postural orthostatic tachycardia [39], peripheral neuropathy, personality changes and sleep disturbances are also encountered. [40]
• Persistent symptoms of pain, fatigue, and/or concentration and memory disturbances may be encountered following optimal antibiotic treatment. [41-44]

The range of **joint involvement** includes tendonitis, myositis and bursitis which wax and wane. [45]
• Ultimately, true arthritis ensues, characterized by erythema, pain, swelling and erosion of one or more large joints • most commonly the knees.

Cardiac disease is characterized by arrhythmia, heart block, chest pain or myo-pericarditis. [46-50]
• Rare instances of endocarditis have been reported. [51]
• The rate of carditis among children with early disseminated Lyme disease is 16%. [52]

Other findings associated with Lyme disease include eye disease (keratitis [53], iritis, papilledema, optic neuritis [54-56], papillitis [57], panophthalmitis, panuveitis [58-60]), splenomegaly, hepatomegaly, acute glomerulonephritis [61], vasculitis syndrome (*Borrelia lusitaniae* infection) [62], diffuse reversible alopecia [63] and regional lymphadenopathy

Lyme reinfection has been well-documented after successfully-treated early infection. [64] Repeat episodes of Lyme disease appear to represent re-infection rather than relapse. [65]

In one series, 33% of patients referred to an Infectious Diseases clinic for suspected Lyme disease were found to have Chronic Fatigue Syndrome, and only 23% Lyme disease. [66]

A distinct *Borrelia* species (*Borrelia lonestari*) is found in *Amblyomma americanum* ticks, and has been implicated in human infections in the United States.
• The condition has been referred to as, "southern tick-associated rash illness (STARI)" or Master's disease. [67]
• Erythema migrans has been described in patients with STARI. [68]

Baggio-Yoshinari Syndrome (BYS), a Lyme disease variant reported in Brazil, is characterized by fever, lymphadenopathy, skin lesions, arthralgia or arthritis and neurological findings. [69]

This disease is endemic or potentially endemic to 65 countries.

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Lyme disease in Brazil

Time and Place:
Lyme disease was first described in Brazil in 1991. [70] [71]
- 25 cases were reported to 1997 [72]
- Approximately 40 cases were reported to 2002 - most in Sao Paulo and Mato Grosso do Sul. [73]
- The first description of Lyme disease (3 cases) in Tocantins State was published in 2012. [74]

A unique form of the disease (Baggio-Yoshinari syndrome, BYS) associated with atypical spirochetal forms has been reported in Brazil [75]
- 19 cases of BYS were treated at a hospital in Sao Paulo during 1990 to 2006. [76]

Ixodes (Ixodes) pararicinus [77] , *Ixodes didelphidis* and *I. loricatus* have been identified as potential vectors in Brazil. [78]

Seropositive dogs have been reported in Sao Paulo State. [79]

A similar syndrome (LD-like syndrome or LD imitator syndrome) caused by an unidentified spirochete transmitted by *Amblyomma* has been described in Brazil. [80]
- 6.2% of children with unexplained fever and rash from Sao Paulo State had a Lyme-like illness (1998 to 2000) [81]

Seroprevalence surveys:

7.2% of patients with non-Lyme related skin diseases (Amazon region, 2010 publication) [82]
6.4% of technicians working with wild animals (Sao Paulo, 2006 publication)
1% of dogs in Bahia (2007 publication) [83]
54.9% of cattle in northeastern Para State (*B. burgdorferi*, 2008 publication) [84]

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Lymphocytic choriomeningitis

Agent	VIRUS - RNA. Arenaviridae, Arenavirus: Lymphocytic choriomeningitis virus
Reservoir	House mouse Guinea pig Hamster Monkey
Vector	None
Vehicle	Urine Saliva Feces Food Dust
Incubation Period	8d - 12d (range 6d - 14d)
Diagnostic Tests	Viral culture (blood, throat, CSF). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, myalgia, meningitis and encephalitis; photophobia or pharyngitis may be present; prior exposure to rodents; infection resolves within 2 weeks, however convalescence may require an additional 2 months.
Synonyms	

Clinical

Acute infection:
35% of Lymphocytic choriomeningitis virus infections are asymptomatic and 50% are characterized by a nonspecific flu-like illness.
• Overt infections are characterized by fever, headache, nausea and systemic symptoms, leukopenia and thrombocytopenia. [1] [2]
• Patients may also exhibit lymphadenopathy and a maculopapular rash (12% to 15% of patients have rash and/or meningitis or encephalitis).
• Relapses characterized by a more severe headache with meningitis may occur after initial improvement.
• Papilledema may be noted

The CSF protein concentration ranges from 50 to 300 mg/dl.
• A pleocytosis of several hundred lymphocytes/mm3 is commonly observed.
• Decreases in CSF glucose concentration are documented in over 20% of cases.

Complications:
Complications include encephalitis, psychosis, paraplegia, transitory aqueductal stenosis, and disturbances of cranial, sensory, or autonomic nervous function.
• Occasionally, orchitis, myocarditis, arthritis, or alopecia are encountered.
• Lymphocytic choriomeningitis is increasingly recognized as a cause of hydrocephalus, psychomotor retardation, congenital chorioretinitis and blindness, most often when acquired during the first or second trimesters of pregnancy. [3] [4]
• Congenital infection is also associated with microencephaly, periventricular calcifications, ventriculomegaly, pachygyria, cerebellar hypoplasia, porencephalic and periventricular cysts. [5]

The case-fatality rate for Lymphocytic choriomeningitis is less than one percent; however, patients with sustained viremia lacking an inflammatory response seem to be at risk for fatal outcome. [6]

This disease is endemic or potentially endemic to all countries.

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Lymphogranuloma venereum

Agent	BACTERIUM. Chlamydiaceae, Chlamydiae, Chlamydia trachomatis, types L1, L2, L3
Reservoir	Human
Vector	None
Vehicle	Sexual contact
Incubation Period	7d - 12d (range 3d - 30d)
Diagnostic Tests	Serology. Culture of pus performed in specialized laboratories.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3w. OR Erythromycin 500 mg QID X 3w
Typical Pediatric Therapy	Age < 8 years: Erythromycin 10 mg/kg PO QID X 2 to 4w. Age >= 8 years: Doxycycline 2 mg/kg PO BID X 2 to 4w
Clinical Hints	Genital nodule or vesicle with large, suppurating regional nodes; generalized lymphadenopathy or proctitis may be present; late complications include genital edema, rectal strictures and perianal abscesses.
Synonyms	Bubonulus, Durand-Nicolas-Favre disease, Linfogranuloma venereo, Lymphogranuloma inguinale, Lymphopathia venereum, Maladie de Nicolas et Favre, Tropical bubo, Venereal bubo, Venerisk lymfogranulom. ICD9: 099.1 ICD10: A55

Clinical

Acute illness:
The first stage of Lymphogranuloma venereum (LGV) is characterized by a papule or ulcer on the genital or anal mucosa, or of the adjacent skin. [1-4]
• Occasionally, the lesion is intraurethral or cervical, producing urethritis or cervicitis.
• The secondary stage occurs days to weeks after the primary lesion and is characterized by lymphadenopathy and systemic illness.
• Cervical lymphadenopathy may occur if infection is acquired through oro-genital contact. [5]

Lymphadenitis:
The inguinal lymph nodes are most often affected, and are unilateral in two thirds of patients.
• The obturator and iliac nodes are occasionally affected in women.
• Initially the lymph nodes are discrete and tender with overlying erythema.
• A characteristic "groove" may be evident between the femoral and inguinal lymph nodes.
• In some cases, patients may present with a "bubonulus": penile adenopathy and secondary local acute lymphedema. [6]
• Later, the nodes may suppurate and coalesce, forming a bubo that may rupture spontaneously (30% of cases) to produce fistulae [7] or sinus tracts which may drain for months.

Inguinal lymphadenopathy in cat-scratch disease may suggest a diagnosis of lymphogranuloma venereum. [8]
• Rectal involvement may suggest a diagnosis of inflammatory bowel disease. [9] [10]

Systemic manifestations at this stage include fever, headache, and myalgia.
• Meningitis may also occur.
• LGV is increasingly recognized as a cause of hemorrhagic proctitis in men who have sex. [11]
• Reactive arthritis has been reported following LGV proctitis [12] [13]

Relapse occurs in 20% of untreated patients.

Only 25% of women present with inguinal lymphadenopathy.
• Women and homosexual men may present with proctitis or pain in the lower abdomen and back pain related to involvement of pelvic and lumbar lymph nodes.
• Late complications include esthiomene (chronic hypertrophic and ulceration of the vulva, scrotum or penis), and elephantiasis of the male or female genitalia.
• Major lower leg involvement may suggest a diagnosis of deep-vein thrombosis. [14]

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This disease is endemic or potentially endemic to all countries.

Lymphogranuloma venereum in Brazil

Approximately 50,000 cases were reported during 1987 to 1996.

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Malaria

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Haemosporida: Plasmodium spp.
Reservoir	Human Primate (Plasmodium knowlesi)
Vector	Mosquito (Anopheles)
Vehicle	Blood
Incubation Period	12d -30d
Diagnostic Tests	Examination of blood smear. Serology, antigen & microscopic techniques. Nucleic acid amplification.
Typical Adult Therapy	Resistant falcip: Lumefantrine/Artemether OR Quinine + Doxycycline or Clindamycin OR Atovaquone/proguanil OR Artesunate IV if severe malaria If sens., Chloroquine 1g, then 500 mg at 6, 24 & 48 hrs. If P. ovale or P. vivax - follow with Primaquine
Typical Pediatric Therapy	Resistant falcip: Lumefantrine/Artemether OR Quinine + Clindamycin OR Atovaquone/proguanil OR Artesunate (>age 8) IV (severe malaria) If sens, Chloroquine 10 mg/kg, then 5 mg/kg at 6, 24, & 48 hrs. If P. ovale or P. vivax - follow with Primaquine
Clinical Hints	Fever, headache, rigors ("shaking chills"), vomiting, myalgia, diaphoresis and hemolytic anemia; fever pattern (every other or every third day) and splenomegaly may be present; clinical disease may relapse after 7 (ovale and vivax) to 40 (malariae) years.
Synonyms	Ague, Bilious remittent fever, Chagres fever, Estiautumnal fever, March fever, Marsh fever, Paludism, Paludismo, Plasmodium falciparum, Plasmodium knowlesi, Plasmodium malariae, Plasmodium ovale, Plasmodium vivax. ICD9: 084 ICD10: B50,B51,B52,B53,B54

Clinical

WHO Case definition for surveillance (For use in endemic areas and people exposed to malaria, e.g., a history of visit to endemic area).
• Malaria must be defined in association with clinical disease symptoms.
• The case definition for malaria cannot be uniform: it will vary according to how malaria is perceived in a given country, local patterns of transmission, and disease consequences.
• The suggested definitions are deliberately broad.
• Each national malaria control program must adapt the definition and introduce additional indicators to make it more applicable to local epidemiology and control targets.
Clinical description
• Signs and symptoms vary; most patients experience fever.
• Splenomegaly and anemia are commonly associated signs.
• Common but non-specific symptoms include otherwise unexplained headache, back pain, chills, sweating, myalgia, nausea, vomiting.
• Untreated *Plasmodium falciparum* infection can lead to coma, generalized convulsions, hyperparasitemia, normocytic anemia, disturbances of fluid, electrolyte, and acid-base balance, renal failure, hypoglycemia, hyperpyrexia, hemoglobinuria, circulatory collapse / shock, spontaneous bleeding (disseminated intravascular coagulation), pulmonary edema, and death.
Laboratory criteria for diagnosis
Demonstration of malaria parasites in blood films (mainly asexual forms).
Case classification
In areas without access to laboratory-based diagnosis.
• Probable uncomplicated malaria: A person with symptoms and/or signs of malaria who receives anti-malarial treatment.
• Probable severe malaria: A patient who requires hospitalization for symptoms and signs of severe malaria and receives anti-malarial treatment.
• Probable malaria death: death of a patient diagnosed with probable severe malaria.
In areas with access to laboratory-based diagnosis.
• Asymptomatic malaria: A person with no recent history of symptoms and/or signs of malaria who shows laboratory confirmation of parasitemia.
• Confirmed uncomplicated malaria: A patient with symptoms and/or signs of malaria who received anti-malarial treatment, with laboratory confirmation of diagnosis.

Acute infection:

Most cases present with non-specific signs suggestive of 'sepsis,' such as fever, rigors, headache and myalgia.
• Clinical findings include cough, fatigue, malaise, arthralgia, myalgia, headache, and diaphoresis.

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• In Africa, tickborne relapsing fever [1] and rabies are often mis-diagnosed as malaria. [2]

The typical malarial paroxysm begins with rigors lasting 1 to 2 hours, followed by high fever.
• This is followed by marked diaphoresis and a fall in temperature.
• Tertian (fever every other day) fever may occur in infection by *P. falciparum*, *P. vivax* and *P. ovale*; quartan (every third day) fever with *P. malariae* infection; and daily fever with *P. knowlesi* infection. [3] [4]
• *P. knowlesi* malaria appears to be more severe than *P. malariae* malaria, with higher rates of parasitemia and fatality. [5-9]
• 'Classical' fever patterns are rarely helpful, and anemia and splenomegaly develop only after several attacks.
• Less common findings include anorexia, vomiting, diarrhea and hypotension.
• In some cases, malaria may present as fever accompanied by an urticarial rash. [10]

Complications:
Complications include pulmonary disease (ARDS) [11] [12], encephalopathy (cerebral malaria) [13-15], nephropathy, retinopathy [16-19], cranial nerve palsy, cerebellar ataxia [20], acute disseminated encephalomyelitis [21], hypocalcemia with tetany [22], shock ('algid malaria'), purpura fulminans [23], massive diarrhea, pancreatitis [24], myocarditis [25] and dysfunction of other organs. [26] [27]
• Patients with *falciparum* malaria are at increased risk for bacteremia. [28]
• Occasionally, patients experience Post-malaria Neurological Syndrome: acute confusion, cerebellar ataxia, diffuse cerebral demyelination, seizures, hearing loss [29], cognitive dysfunction [30] or other neuropsychiatric findings several days to weeks following successful treatment of falciparum malaria. [31-36]
• *Plasmodium falciparum* infection accounts for most complications and deaths from malaria [37]; however, severe disease may occasionally complicate infection by other species. [38-43]
• The presence of malarial retinopathy is associated with a poor prognosis. [44]
• *P. falciparum* is also responsible for most malarial drug resistance.
• Maternal infection is associated with vertical transmission to the newborn [45], fetal loss and low birth weight in infants. [46-50]
• 5% of African children with severe malaria were found to have concomitant bacteremia [51]
• Severe and fatal disease associated with *Plasmodium vivax* infection is increasingly reported in recent years. [52-65] Instances of acute glomerulonephritis [66-68], IgA nephropathy [69], renal cortical necrosis [70], acalculous cholecystitis [71], jaundice, pancreatitis [72], thrombocytopenia [73] [74], shock, peripheral gangrene [75], cerebral malaria [76], hypoglycemia and acute respiratory distress syndrome have been reported with *Plasmodium vivax* infections. [77-80]
• *Plasmodium malariae* infection is rarely associated with severe illness [81]; and may lead to renal glomerular damage and nephrotic syndrome. [82-84]
• Pericarditis [85] and acute respiratory distress syndrome have been reported in *Plasmodium ovale* infection. [86]

Malaria and HIV infection:
HIV infection increases the incidence and severity of clinical malaria; however, in severe malaria the level of parasitemia is similar in HIV-positive and HIV-negative patients. [87-95]
• During pregnancy, HIV infection increases the incidence of clinical malaria, maternal morbidity, and fetal and neonatal morbi-mortality.
• HIV infection increases the risk for malaria treatment failure, and for cerebral malaria in children. [96]
• Some antimalarial drugs may inhibit HIV, while certain anti-retroviral drugs are effective against *Plasmodium* species. [97]

Relapse:
Relapse may occur months to years following the initial episode.
• Relapses of *P. vivax* and *P. ovale* infection result from release of parasites which had remained dormant in the liver.
• As such, treatment of infection by either of these two species should include a drug (eg, primaquine) active against intrahepatic parasites.
• Although infections caused by *P. falciparum* and *P. knowelsi* do not relapse, reinfection may occur. [98]

Plasmodium malariae persists without symptoms in the blood, rather than the liver.
• Relapse has been reported as long as 40 to 50 years following exit from an endemic area. [99]

This disease is endemic or potentially endemic to 186 countries. Chloroquine resistant falciparum malaria endemic to 81 countries. Chloroquine-sensitive malaria endemic to 29 countries.

Malaria in Brazil

Time and Place:
Brazil accounts for 60% of malaria cases in the Americas region (2011 publication). [100]
- Approximately 19 million people (12% of the population) live in risk areas.
- Risk exists year-round.

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- There is no risk along the East coast from Rio Grande do Sul to Ceara (including Foz do Iguacu).
- Most cases of malaria are reported from rural Amapa, Amazonas, Goias, Maranhao, Mato Grosso, Rondonia, Roraima and Para (except in Belem).
- Cases are also reported from Parana [101] , Tocantins [102] and Acre.
- Rondonia accounts for 50% of cases.

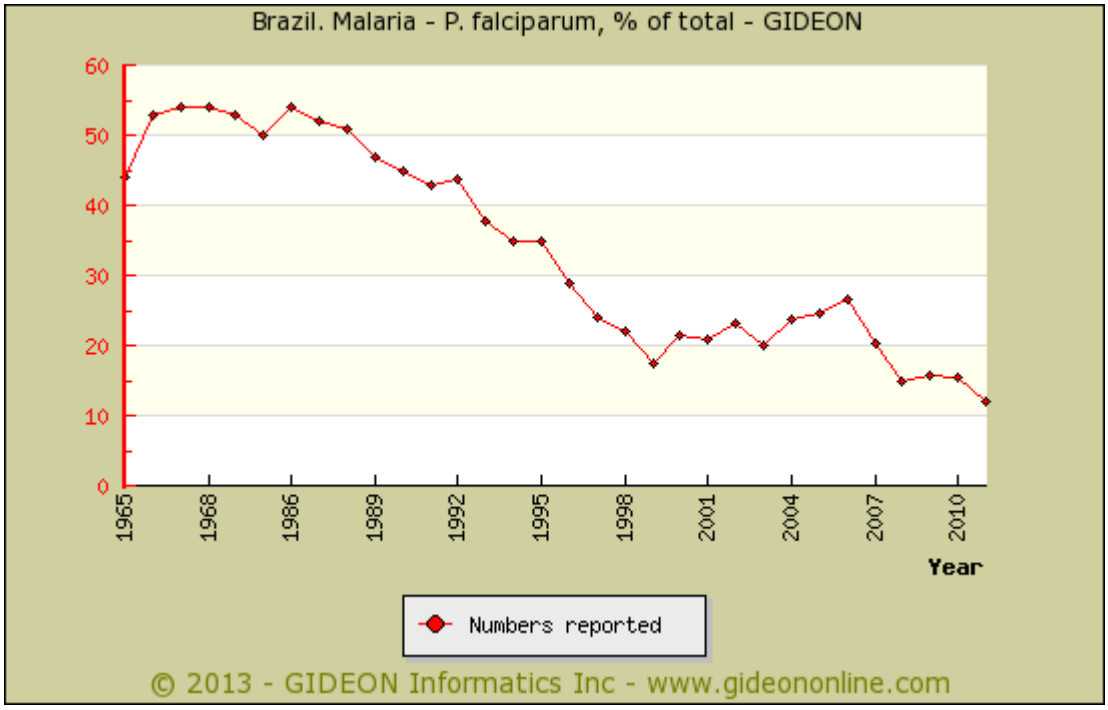
Malaria is present in most forested areas below 900 m within the nine states of the "Legal Amazonia" region (Acre, Amapa, Amazonas, Maranhao (western part), Mato Grosso (northern part), Para (except Belem City), Rondonia, Roraima and Tocantins. [103]
- Transmission intensity varies from municipality to municipality, but is higher in jungle areas of mining, lumbering and agricultural settlements less than 5 years old, than in the urban areas, including in large cities such as Porto Velho, Boa Vista, Macapa, Manaus [104] , Santarem and Maraba, where the transmission occurs on the periphery of these cities.
- In the states outside "Legal Amazonia", malaria transmission risk is negligible or non-existent.
- An autochthonous case was reported in Cananeia (southern coast of Sao Paulo) in 2002 - the area had been malaria-free for 60 years. [105]
- The incidence of malaria in Santa Isabel de Rio Negro increased sixteen-fold during 2003 to 2007. [106]
- Increasing transmission of *vivax* malaria was reported in eastern Mato Grosso during 2010. [107] [108]

Although malaria was eradicated from Rio de Janeiro since 1968, sporadic autochthonous cases are reported from the state (3 in 1997).
- The State of Sao Paulo experienced 177 autochthonous cases during 1951 to 1960; 160 during 1961 to 1970; 52 during 1971 to 1980; 72 during 1981 to 1990.

An outbreak (11 cases) was reported the non-endemic region of Monte Alegre de Minas, State of Minas Gerais in 2005. [109]

The prevalence of malaria in Vale do Amanhecer settlement, Juruena, Mato Grosso was 33% in 2005. [110]

Infecting species:
- Chloroquine-resistant *P. falciparum* **IS** reported since 1961. [111]
- Chloroquine-resistant *P. vivax* may be present. [112]
- *Plasmodium malariae* may account for as many as 11.9% of malaria cases in Apiacas (Mato Grosso State).
- The relative rarity of *P. vivax* infection in West Africa is related to lack of Duffy antigen (necessary for erythrocyte adherence) in the local population [113] ; however, infection is reported among Duffy antigen-negative populations in Brazil. [114-116]



Graph: Brazil. Malaria - *P. falciparum*, % of total

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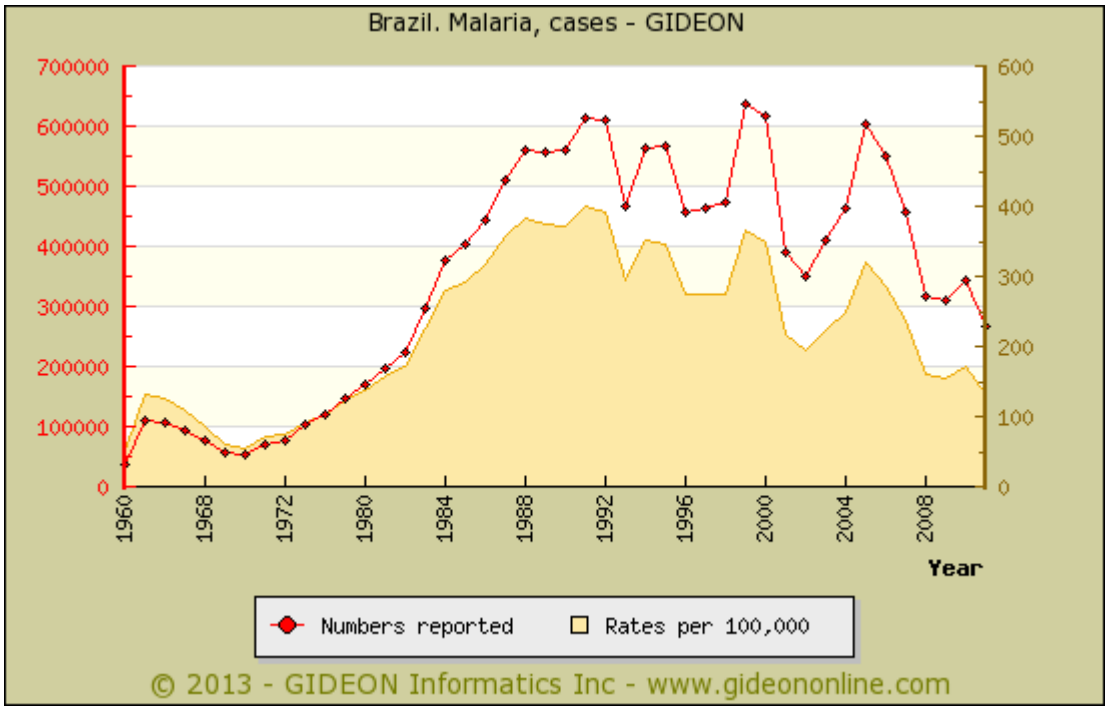

Graph: Brazil. Malaria, cases

Notes:
1. 9,597,144 cases were reported during 1980 to 2000 - 28.6% in Rondonia, 29.2% in Para, 11.2% in Mato Grosso, 10.1% in Amazonas and 7.2% in Maranhao
2. 1989 - An outbreak of 3,000 cases were reported in Foz do Iguacu
Individual years:
1990 - 300 cases were reported in Foz do Iguacu.
1991 - 98% from the Amazon basin.
1994 - Brazil accounted for 50.6% of all malaria and 60% of all *P. falciparum* infections reported in the Americas. Highest rates in Mato Grosso, Rondonia, Para, Roraima and Amazonas.
1996 - 33% from Para, 16% from Amazonas.
1997 - 37.4% from Para.
2008 - Brazil accounted for 56% of all cases reported in the Americas and the Caribbean. [117]

Disease rates per 100,000 in the Amazon region: 1,529.6 in 2001; 1,319.4 in 2002; 1,898.5 in 2003; 2,119.2 in 2004; 2,661.2 in 2005; 2,364.9 in 2006. [118]

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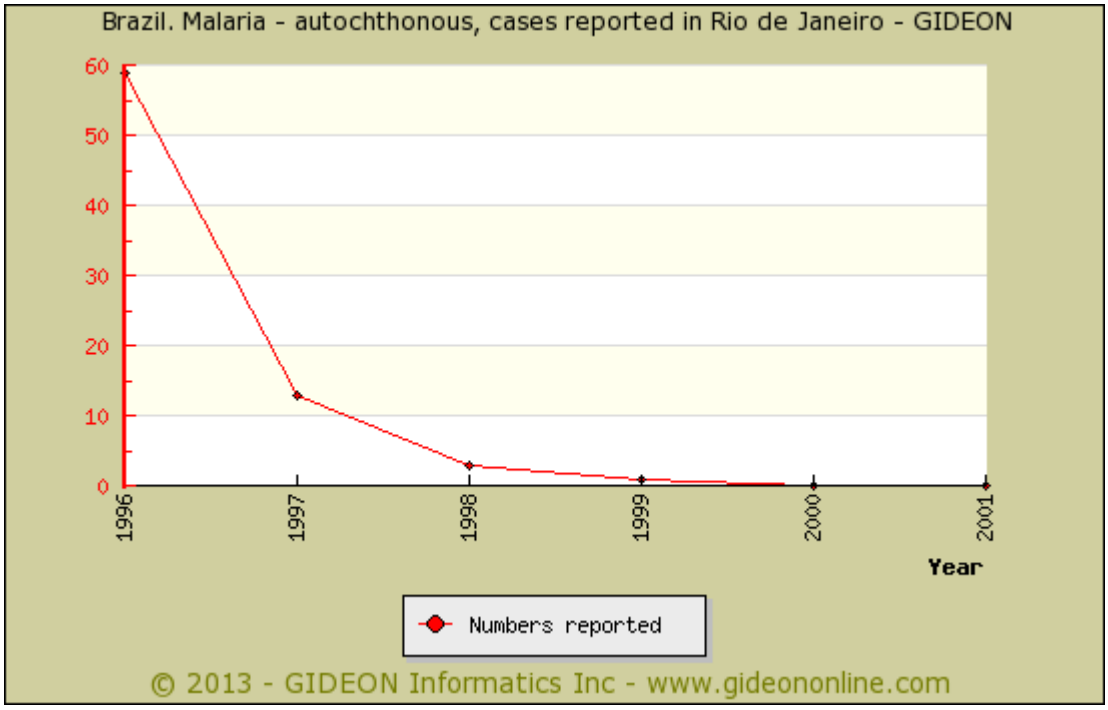
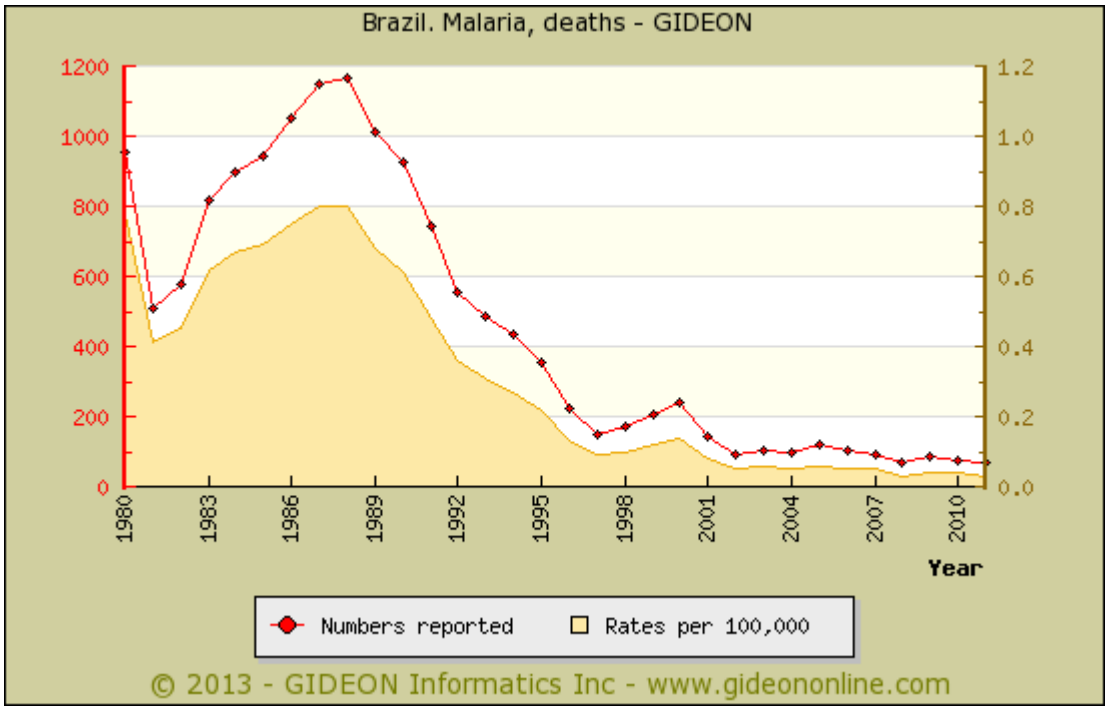


Graph: Brazil. Malaria - autochthonous, cases reported in Rio de Janeiro

Notes:
1. Four autochthonous cases were reported in Paraty (Rio de Janeiro) following introduction of *P. vivax* by an Argentinean tourist in 2002. [119]

78 Brazilian soldiers (out of 439) acquired malaria while deployed in Angola during 1995 to 1996 - three of these died of cerebral malaria. [120]



Graph: Brazil. Malaria, deaths

Notes:
1. 3,700 fatal cases were reported during 1983 to 1986
2. The true number of fatal cases has been estimated at 10,000 per year.

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Vectors:

The principal vectors are currently *Anopheles darlingi* [121] [122] and *An. nuneztovari*

- *An. mattogrossensis*, *An. braziliensis*, *An. mediopunctatus* and *An. peryassui* are also implicated in transmission in Brazil. [123]

- *Anopheles gambiae* was introduced into northeastern Brazil during 1930 to 1941, and caused extensive epidemics in 1930 to 1931, and 1938 to 1939 (over 100,000 cases per month at the peak of the outbreak).

- *An. albitarsis* [124] , *An. nuneztovari*, *An. braziliensis*, *An. triannulatus*, *An. oswaldoi* and *An. rangeli* are implicated in transmission in Amapa. [125]

- *An. aquasalis* is found along the entire Atlantic coast, and *An. oswaldoi* is found in Acre. [126]

- *An. aquasalis* predominates in Maranhao, followed by *An. galvaoi*, *An. albitarsis*, *An. evansae*, *An. nuneztovari* and *An. triannulatus davisi*. [127]

- *An. costai* has been implicated in transmission in Espirito Santo State. [128]

- *An. darlingi* and *An. aquisalis* are the principal vectors in Belem (Para State). [129]

- *An. deaneorum* is suspected to be an important vector in the western Amazon. [130]

- *An. marajoara* is the principal vector in Macapa (Amapa State). [131]

- *An. darlingi*, *An. albitarsis* and *An. rondoni* are the principal vectors in Para State. [132]

- *An. (Nyssorhynchus) darlingi*, *An. (Nyssorhynchus) albitarsis*, *An. (Nyssorhynchus) triannulatus* and *An. (Nyssorhynchus) parvus* have been implicated in transmission in Monte Alegre de Minas, State of Minas Gerais. [133]

- *An. darlingi* is the principal vector in Porto Velho, Rondonia State. *An. triannulatus*, *An. nuneztovari*, *An. gilesi* and *An. costai* are also active in the region. [134]

- *An. cruzi* infected by *Plasmodium malariae* have been identified in Itahhaem, Sao Paulo. [135]

Notable outbreaks:

1990 (publication year) - An outbreak (12 cases) of *Plasmodium vivax* malaria was reported among injecting drug users (IDU) in Sao Paulo. [136] [137]

1991 (publication year) - An outbreak of *Plasmodium vivax* malaria was reported in Mantena (Minas Gerais), a non-endemic area. [138]

1991 to 1992 - An outbreak (163 cases) was reported on a farm settlement in Rondonia. [139]

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Malignant otitis externa

Agent	BACTERIUM. Pseudomonas aeruginosa: aerobic gram-negative bacillus (virtually all cases)
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture of otic exudate and biopsy material. Careful roentgenographic and neurological examinations.
Typical Adult Therapy	Early debridement complemented by at least 2 parenteral antibiotics active against Pseudomonas aeruginosa
Typical Pediatric Therapy	As for adult
Clinical Hints	Otic pain, swelling and discharge; infection of bony and cartilaginous ear canal; over 80% of patients are diabetics over age 50; cranial nerve (usually VII) signs in 50%. case-fatality rate > 55%.
Synonyms	

Clinical

The case definition of Malignant Otitis Externa consists of pain, edema, exudate, granulations, microabscesses (when explored), positive bone scan or failure of local treatment often more than 1 week. [1]
• Additional criteria may include cranial nerve involvement, positive radiograph, debilitating condition and old age.

Severe pain and tenderness in the mastoid area are accompanied by drainage of pus from the external canal. [2]
• Involvement of the temporal bone, meninges, venous sinuses, cranial nerves (IX, X, XII) and brain may follow.

This disease is endemic or potentially endemic to all countries.

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Mammomonogamiasis

Agent	PARASITE - Nematoda. Phasmidea: Mammomonogamus (Syngamus) laryngeus
Reservoir	Mammal Bird Coyote Cat Cattle Orangutan
Vector	None
Vehicle	Uncooked Vegetables Water
Incubation Period	6d - 11d
Diagnostic Tests	Identification of ova in feces, or of adults from respiratory tract.
Typical Adult Therapy	Extraction or expulsion of parasite
Typical Pediatric Therapy	As for adult
Clinical Hints	Cough and hemoptysis associated with a laryngeal 'foreign body'; may persist for months.
Synonyms	Gapeworm, Mammomonogamus, Syngamus laryngeus. ICD9: 128.8 ICD10: B83.3

Clinical

Symptoms suggest a foreign body in the upper respiratory tract, with cough, wheezing and occasionally hemoptysis. [1]
• The condition is usually diagnosed following expulsion of characteristic red, Y-shaped adult worms (male 4 to 5 mm, and female 15 to 20 mm in length). [2]

This disease is endemic or potentially endemic to 17 countries.

Mammomonogamiasis in Brazil

Human infection has been reported in Sao Paulo State. [3]

A Portuguese tourist acquired infection in either Rio de Janeiro or Ilheus in 2004.

Mammomonogamus laryngeus has been identified in cattle in Sao Paulo State [4] ; and buffalo (*Bubalus bubalis*) in Para State [5] and Rio Grande do Sul. [6]

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Mansonelliasis - M. ozzardi

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Mansonella ozzardi
Reservoir	Human
Vector	Fly (black fly = Simulium) or midge (Culicoides)
Vehicle	None
Incubation Period	5m - 18m (range 1m - 2y)
Diagnostic Tests	Identification of microfilariae in skin snips or blood. Nucleic acid amplification.
Typical Adult Therapy	Ivermectin 150 ug/kg PO as single dose
Typical Pediatric Therapy	As for adult
Clinical Hints	Arthralgia, pruritus, urticaria, rash, bronchospasm, headache, lymphadenopathy and eosinophilia.
Synonyms	Filaria ozzardi, Mansonella ozzardi, Microfilaria bolivarensis, Ozzardiasis, Tetrapetalonema ozzardi. ICD9: 125.5 ICD10: B74.4

Clinical

Clinical features are mild, and limited to any combination of pruritus, bronchospasm, rash, headache, arthralgias, fever, eosinophilia and lymphadenopathy. Nummular keratitis, associated with the presence of microfilaria in the cornea, is common. [1-3]

This disease is endemic or potentially endemic to 23 countries.

Mansonelliasis - M. ozzardi in Brazil

Mansonella ozzardi infection is endemic to rural areas of Amazonas.

Prevalence surveys:

3% in the Federal Territory of Roraima (1985 publication) [4]
4.4% among mesticos on the river Purus (Amazonas, 1975 publication) [5]
24.86% of persons on the Pauini River and 24.19% on the Purus river (2009 publication) [6]
0% of persons living along the Madeira, Mamore, Guapore, Machado and Preto Rivers (Rondonia, 2011 publication) [7]
13.3% of persons in Coari, middle Solimoes River region (2010 publication) [8]
18.9% of persons in Coari rural area on Solimoes River right margin (2008 publication) [9]
26.4% of persons in Coari City (2012 publication) [10]
30.33% of persons living along the Ituxi river, Labrea (Amazonas State, 2008 publication) [11]
20.7% of persons and 0.6% of vector flies (*Cerqueirellum amazonicum*) in Labrea, Amazonas State (2011 publication) [12]
22.2% of women, 32% of men, 49% of farmers, and 0.98% of blackflies in communities on the Purus River, Boca do Acre municipality, Amazonas State (2009 publication) [13]

The principal vector is *Simulium (Cerqueirellum) amazonicum* [14] ; however, *Culicoides insinuatus* and *Cerqueirellum pydanielli* [15] may also be active.
- *Cerqueirellum argentiscutum* has been identified as a vector in Amazonas. [16]

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Mansonelliasis - M. perstans

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Mansonella (Esslingeria) perstans
Reservoir	Human
Vector	Midge (Culicoides spp.)
Vehicle	None
Incubation Period	5m - 18m (range 1m - 2y)
Diagnostic Tests	Identification of microfilariae in blood. Nucleic acid amplification.
Typical Adult Therapy	Albendazole 400 mg PO BID X 10 d OR Mebendazole 100 mg PO BID X 30 d. Recent data suggest that addition of doxycycline may be of benefit.
Typical Pediatric Therapy	Age >2 years: As for adult. OR Albendazole 10 mg/kg/day PO X 10d
Clinical Hints	Recurrent pruritic subcutaneous lesions, arthralgia and eosinophilia; headache, fever or abdominal pain may also be present.
Synonyms	Acanthocheilonema perstans, Bung eye disease, Dipetalonema berghei, Dipetalonema perstans, Dipetalonema semiclarum, Esslingeria perstans, Filaria perstans, Mansonella perstans, Mansonella rhodhaini, Mansonella semiclarum, Meningonema peruzzii, Tetrapetalonema berghei, Tetrapetalonema perstans. ICD9: 125.4 ICD10: B74.4

Clinical

Patients develop recurrent pruritic subcutaneous swellings, fever, headache, joint pain, abdominal or chest pain and eosinophilia.
- Hepatosplenomegaly and intraocular lesions are occasionally seen.
- Asymptomatic microfilaremia from transfusion of infected blood has been reported.
- "Bung eye,' characterized by the formation of yellowish nodules on the bulbar conjunctivae with proptosis and lid edema, has been reported in Uganda and neighboring countries. [1]
- Microfilariae of *Mansonella perstans* have been identified in vaginal secretions. [2] [3]

Human cerebral infection by *Meningonema peruzzii* has been reported. [4]

This disease is endemic or potentially endemic to 49 countries.

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Mayaro

Agent	VIRUS - RNA. Togaviridae, Alphavirus: Mayaro virus
Reservoir	? Non-human primate ? Bird
Vector	Mosquito (Haemagogus janthinomys)
Vehicle	None
Incubation Period	3d - 12d
Diagnostic Tests	Viral culture (blood). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Consider in a forest worker with headache, myalgia, arthralgia, lymphadenopathy and a maculopapular rash; although acute illness resolves within 5 days, joint pains may persist for months.
Synonyms	Una. ICD9: 066.3 ICD10: A92.8

Clinical

Mayaro is characterized by abrupt onset of fever, chills, myalgia and headache. [1]
• Gastrointestinal symptoms may also be present.
• Lymphadenopathy is found in 50% of patients.
• Arthralgias of the small joints of hands and feet are present.
• A maculopapular rash of the trunk and extremities appears on the second to fifth days of illness in two-thirds of patients.

There are no sequelae; however, joint pain may persist for several months.

This disease is endemic or potentially endemic to 11 countries.

Mayaro in Brazil

Mayaro virus was first identified as an agent of human disease in Brazil, in 1955. [2]

2010 - A French traveler acquired Mayaro virus infection in Brazil. [3] [4]

Prevalence surveys:
5.5% of patients with high fever, in Manaus (2011 publication) [5]

Seroprevalence surveys:
5.4% of inhabitants of Rio Branco, Acre (1999). [6]

Marmosets (*Calithrix argentata*) are the main amplifying host in this country.

Seropositivity was identified among sentinel primates in Mato Grosso do Sul during 2010. [7]

Vectors:
- *Haemagogus janthinomys* is the principal mosquito vector in Brazil.
- *Aedes albopictus* is considered a potential vector. [8]

Notable outbreaks:
1977 to 1978 - Outbreaks (800 cases = 20% of the local population, 0 fatal) of Mayaro and yellow fever were reported in Belterra (Para). [9-11]
2007 to 2008 - Outbreaks of Mayaro were reported in Belem, Para State (36 cases) [12] [13] and Manaus. [14]

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Measles

Agent	VIRUS - RNA. Paramyxoviridae, Paramyxovirinae, Morbillivirus: Measles virus
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	8d - 14d
Diagnostic Tests	Viral culture (difficult and rarely indicated). Serology. Nucleic acid amplification.
Typical Adult Therapy	Respiratory isolation; supportive. Ribavirin 20 to 35 mg/kg/day X 7 days has been used for severe adult infection
Typical Pediatric Therapy	As for adult
Vaccines	Measles Measles-Mumps-Rubella Measles-Rubella
Clinical Hints	Coryza, fever, headache, conjunctivitis, photophobia and a maculopapular rash after 3 to 5 days; Koplik's spots (bluish-grey lesions on buccal mucosa, opposite second molars) often precede rash; encephalitis or viral pneumonia occasionally encountered.
Synonyms	Masern, Massling, Mazelen, Meslinger, Morbilli, Morbillo, Rubeola, Rugeole, Sarampion, Sarampo. ICD9: 055 ICD10: B05

Clinical

WHO Case definition for surveillance:
Any person with:
• fever, and
• maculopapular (i.e. non-vesicular) rash, and
• cough, coryza (i.e. runny nose) or conjunctivitis (i.e. red eyes).
or
Any person in whom a clinician suspects measles infection.
Laboratory criteria for diagnosis
• At least a fourfold increase in antibody titer or
• Isolation of measles virus or
• Presence of measles-specific IgM antibodies
Case classification
• Clinically confirmed: A case that meets the clinical case definition.
• Probable: Not applicable.
• Laboratory-confirmed: only for outbreak confirmation and during elimination phase A case that meets the clinical case definition and that is laboratory-confirmed or linked epidemiologically to a laboratory-confirmed case.

Acute illness:

Symptoms begin to appear about 10 to 12 days after exposure to the virus, with fever followed by cough, rhinorrhea, and/or conjunctivitis. [1]
• The rash appears approximately 14 days after exposure and lasts 5 to 6 days.
• The rash begins at the hairline, spreading to the face and neck.
• Over the next three days, the rash gradually extends, eventually reaching the hands and feet. [2]

Complications:

Complications of measles include diarrhea, otitis media (10%), pneumonia (5%), encephalitis (0.1%) [3] [4], arthropathy (28%) [5], seizures, and death. [6]
• Twenty percent of patients experience one or more complications, most often children below five years of age and adults over 20.
• Measles in pregnancy may be associated with maternal pneumonia, abortion, low birth weight [7] [8] or congenital infection of the newborn. [9] [10]
• In developing countries, measles has been known to kill as many as one out of four people.

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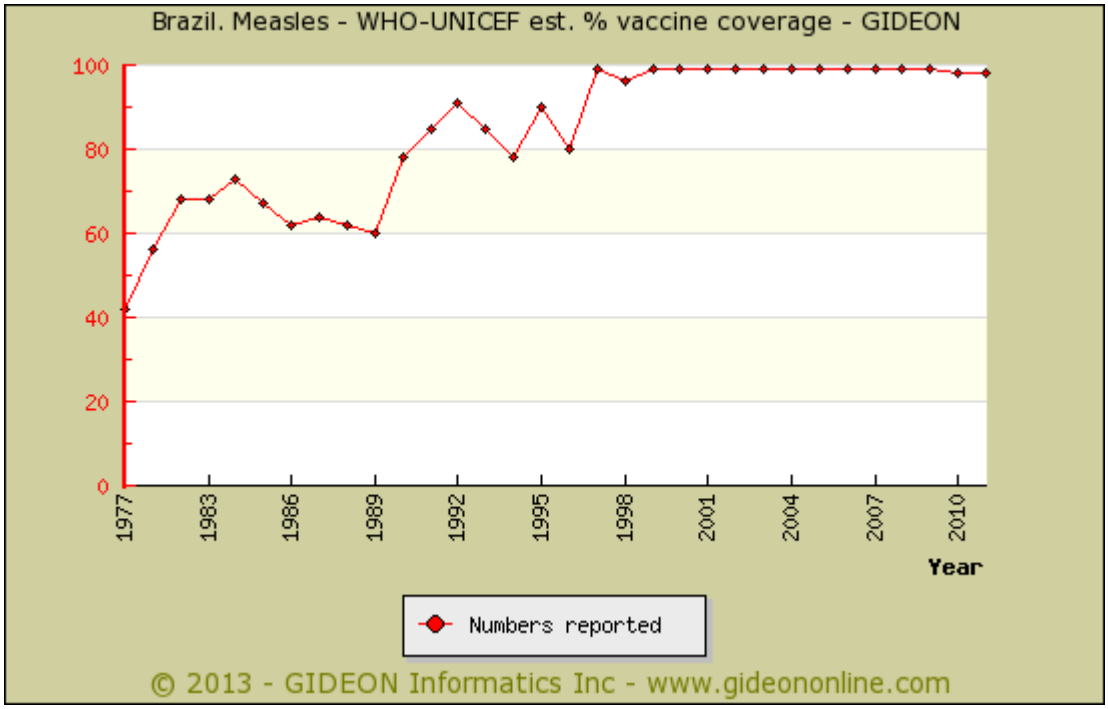
- Measles is the leading cause of blindness among African children, as a result of concomitant vitamin A deficiency.
- Measles pneumonia accounts for approximately 17% of bronchiolitis obliterans in children (Beijing, 2001 to 2007) [11]
- Rare instances of thyroiditis, pancreatitis and sialoadenitis have been reported. [12]

This disease is endemic or potentially endemic to all countries.

Measles in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Routine immunization against measles was introduced in 1973.
- Routine immunization (monovalent vaccine) was introduced in 1985 and administered at age 9 to 11 months
- Monovalent vaccine was replaced by MMR at age 12 months, as of 2000.



Graph: Brazil. Measles - WHO-UNICEF est. % vaccine coverage

Prevalence surveys:

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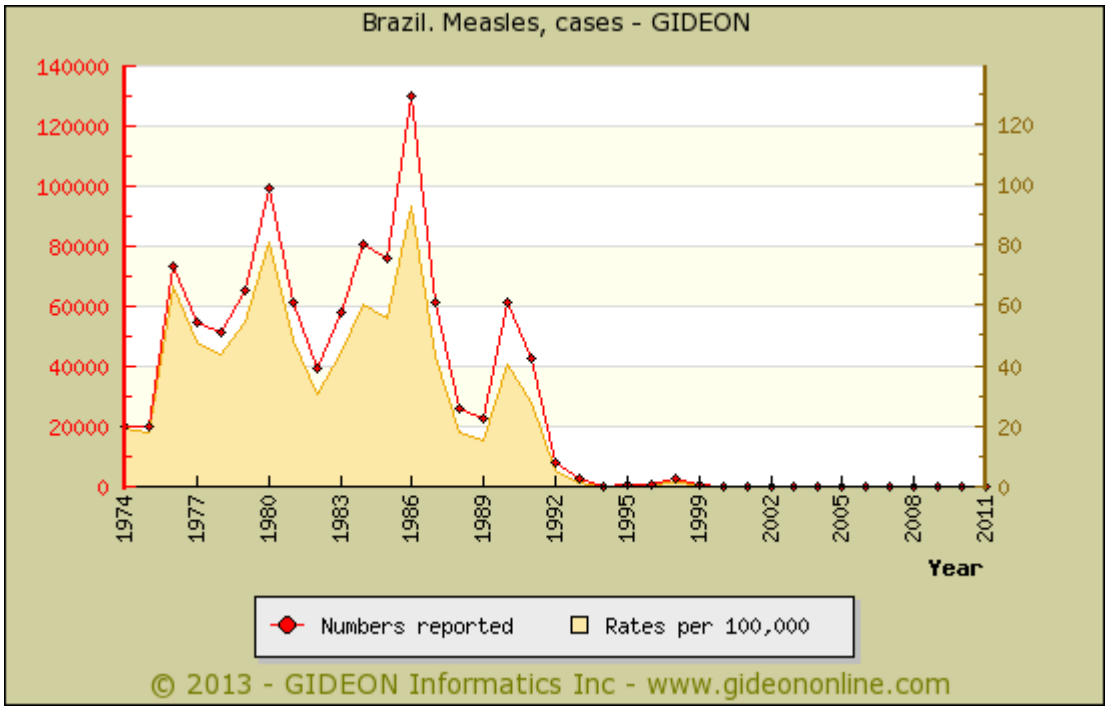
6.7% of patients with maculopapular rash (Niteroi, Rio de Janeiro State, 1994 to 1998) [13]

Seroprevalence surveys:

93.4% of blood donors in Rio de Janeiro (2000)

98% of hospital pediatricians in Sao Paulo (1999 publication) [14]



Graph: Brazil. Measles, cases

Notes:

1. Mandatory reporting of measles was introduced in 1968.
2. 834,153 cases were reported during 1980 to 2000.
3. All cases confirmed in 2001, 2002 and 2003 were imported. [15]
4. Sao Paulo reported 3,395 cases during 1988 to 1992. Only 22 cases were confirmed in 1996. Sao Paulo accounted for 77% of reported cases in 1997; 37% in 2000.
5. Nine cases of measles were reported during 2001 to 2005 - all related to imported cases (Japan, Germany and the Falkland Islands). [16]

Individual years:

1997 - 3.8% of cases reported from Bahia.

1997 - 53,335 clinical cases were reported in 1997

1999 - 10,007 clinical cases were reported (25% of all cases for the Americas).

2000 - 8,560 clinical cases were reported (36 confirmed) - 37% in Acre (including a single outbreak of 15 cases).

2001 - 8,680 clinical cases were reported, including only one confirmed case (imported). [17]

2005 - Case in Santa Catarina. [18]

16.9% of patients suspected to have measles and/or rubella were found to have Parvovirus B19 infection, and 16.9% dengue. (Pernambuco, 2001 to 2004) [19]

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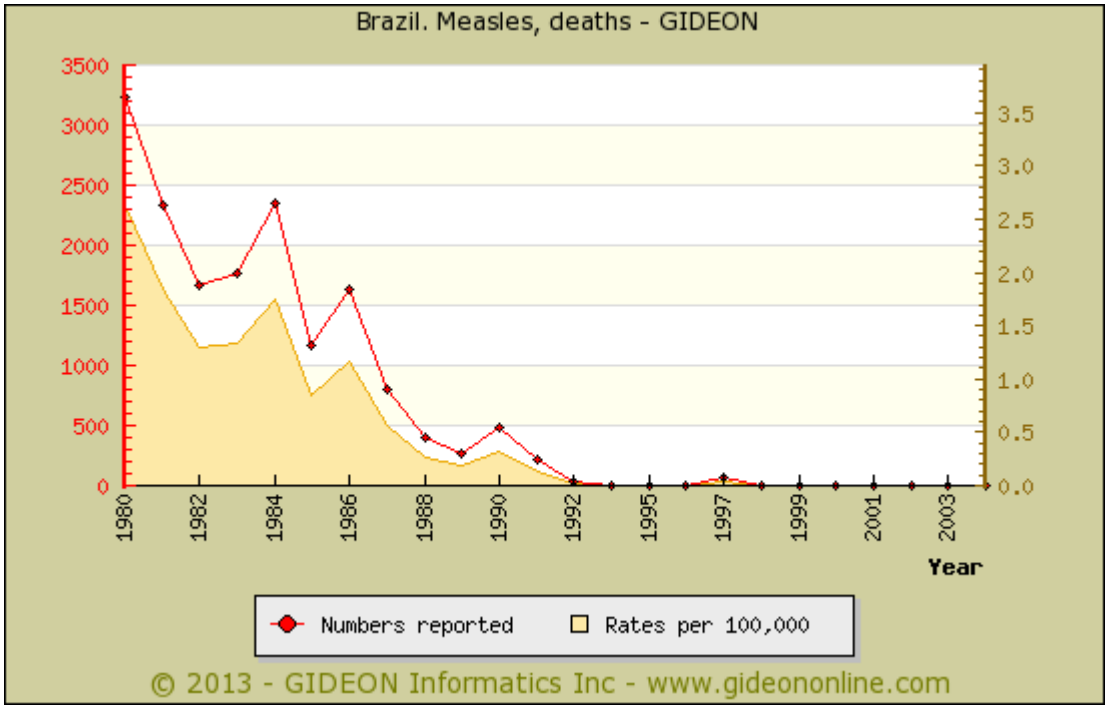

Graph: Brazil. Measles, deaths

Notes:
1. 6,948 deaths were ascribed to measles during 1982 to 1985.
2. Mean annual measles deaths: 2,271 during 1980 to 1984; 851 during 1985 to 1989; 148 during 1990 to 1994.
3. The measles mortality rate was 2.7 per 100,000 in 1980; 0.33 per 100,000 in 1990.

Notable outbreaks:

1983 - An outbreak of measles was reported in Brasilia. [20] [21]

1986 - An outbreak (83,000 cases, estimated) was reported among children below age 15 in Sao Paulo (30.7 per 100,000), with 2,800 hospitalizations.

1990 - An outbreak (2,888 cases, 12 fatal) was reported in Rio, and a concurrent major outbreak in Sao Paulo.

1991 to 1992 - An outbreak was reported in Niteroi, State of Rio de Janeiro. [22] [23]

1996 to 1997 - An outbreak (18,933 cases) was reported in Sao Paulo. [24-31]

1997 - An outbreak (47 cases confirmed) was reported in Niteroi, Rio de Janeiro. [32]

2006 to 2007 - An outbreak (55 cases) was reported in Bahia. [33]

2010 - An outbreak was reported in Belem. [34]

2011 - An outbreak (18 cases) in Colombia was related to an index patient who had arrived from Brazil. [35-37]

2011 - An outbreak (27 cases) in Sao Paulo was related to an index case which arrived from the United States. [38]

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Melioidosis

Agent	BACTERIUM. Burkholderia pseudomallei An aerobic gram-negative bacillus
Reservoir	Soil Water Sheep Goat Horse Pig Rodent Monkey Marsupial
Vector	None
Vehicle	Water: Contact, ingestion, aerosol Breast milk (rare)
Incubation Period	3d - 21d (range 2d - 1y)
Diagnostic Tests	Culture of blood, sputum, tissue. Serology. Nucleic acid amplification.
Typical Adult Therapy	Ceftazidime or Meropenem or Imipenem IV X at least 14 days May be combined with Sulfamethoxazole/trimethoprim PO Follow with Sulfamethoxazole/trimethoprim +/- Doxycycline X at least 3 months.
Typical Pediatric Therapy	Ceftazidime or Meropenem or Imipenem IV X at least 14 days May be combined with Sulfamethoxazole/trimethoprim PO Follow with Sulfamethoxazole/trimethoprim X at least 3 months.
Clinical Hints	May present as: lymphangitis with septicemia; or fever, cough and chest pain; or diarrhea; bone, central nervous system, liver and parotid infection are occasionally encountered; case-fatality rate 10% to over 50% (septicemic form).
Synonyms	Burkholderia pseudomallei, Burkholderia thailandensis, Melioidose, Nightcliff Gardeners' Disease, Whitmore disease. ICD9: 025 ICD10: A24.1,A24.2,A24.3,A24.4

Clinical

The clinical features of melioidosis are similar to those of tuberculosis: prolonged fever, weight loss, latency with reactivation, upper-lobe infiltrates, etc. [1-5]

• As in tuberculosis, long latent periods may precede appearance of the disease; in some reports 29 years [6] , or even 69 years. [7]

• Disease rates are highest among diabetics. [8-10] Other predisposing conditions include collagen-vascular disease, alcoholism, malnutrition, chronic renal or hepatic disease, corticosteroid therapy, splenectomy, pregnancy, chronic granulomatous disease, leukemia and lymphoma.

Acute melioidosis can be divided into five clinical forms:
• septicemia without abscess formation
• septicemia with disseminated foci
• localized infection
• transitory bacteremia
• "fever of unknown origin"

Most patients with overt infection present with pneumonia which may include pulmonary nodules, consolidation, necrotizing lesions, pleural effusion, pleural thickening and mediastinal abscesses. [11-13]
• Occasionally, the only lesion may be a pleural mass.
• Although confluent upper lobe infiltrates are common, the apices are generally spared in non-septicemic cases.
• Rapid progression and early cavitation are common.
• Pleural effusion is seen in 21% of patients with acute disease, and 13% of patients with chronic melioidosis
• Pericarditis occurs in six to ten percent of all patients.
• Patients with cystic fibrosis (ie, traveling to endemic countries) appear to be at high risk for pulmonary infection.
• The pattern of organ involvement in recurrent or relapsing melioidosis is similar to that of primary infection. [14]

45% of cases present as septicemia with infection of multiple organs.
• Pericarditis [15] [16] may complicate the pulmonary infection, and necessitate surgical drainage for tamponade.
• Visceral abscesses may involve the spleen [17] [18] , liver [19-21] , kidneys [22] , pancreas [23] , prostate [24] or other organs. [25]
• Osteomyelitis is common. [26-30]
• Generalized or local suppurative lymphadenitis is occasionally encountered. [31]
• Primary cutaneous diseases occurs in 12% of cases, and secondary cutaneous dissemination in 2% [32] [33]
• Complications of melioidosis include nasopharyngitis, brain abscess [34] , septic arthritis [35] [36] , dural sinus thrombosis [37] , brainstem dysfunction [38] , orbital infection [39] [40] , meningitis, transverse myelitis [41] , urinary tract infection [42] ,

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epididymo-orchitis, prostatitis [43] [44], suppurative parotitis [45], mycotic aneurysm [46], parapharyngeal abscess, corneal ulcer, necrotizing fasciitis [47-49], septic arthritis [50] [51], psoas and other muscular abscesses. [52-54]
• Persistent bacteremia beyond one week under therapy is associated with poor prognosis. [55]
• Melioidosis is the most common cause of mycotic aneurysm in some areas of Thailand. [56]

Renal failure occurs in up to one-third of hospitalized patients with melioidosis, and carries a poor prognosis.

In nonendemic regions, patients present with reactivated disease occurring months to years after initial exposure to the organism.
• Typical symptoms include fever, cough, weight loss and apical changes on chest x-ray • all suggestive of tuberculosis.
• The presence of a mediastinal mass may mistakenly suggest malignancy. [57]
• The clinical features of melioidosis may also mimic those of enteric fever. [58]
• It is not uncommon for the two diseases to coexist.

This disease is endemic or potentially endemic to 73 countries.

Melioidosis in Brazil

The first laboratory-confirmed case of melioidosis in Brazil was reported in 2003. [59]

A Dutch national acquired melioidosis in Brazil in 2003. [60]

Four cases (three fatal) were reported (in Ceara) in 2003; 1 (in Ceara) in 2004. [61-64]
- A case of melioidosis was reported in the northeastern region (2012 publication) [65]
- A case series (13 cases) was published from Ceara in 2012. [66]

Prevalence surveys:

4.3% of environmental samples in Ceara State, during the dry and rainy seasons (2008 publication). [67]

Seroprevalence surveys:

58.5% of persons in Tejucuoca and Banabuiu, Ceara (2006) [68]

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Meningitis - aseptic (viral)

Agent	VIRUS - RNA. Picornaviridae, enteroviruses
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Droplet
Incubation Period	Variable
Diagnostic Tests	Viral isolation (stool, CSF, throat). Serology.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Lymphocytic meningitis (normal CSF glucose); often follows sore throat; typically occurs during late summer and early autumn in temperate regions.
Synonyms	Aseptic meningitis, Encephalitis - viral, Meningite virale, Meningitis, viral, Meningo-encefalite virale, Viral encephalitis, Viral meningitis. ICD9: 047,048,049,320.2 ICD10: A87,G03.0

Clinical

WHO Case definition for surveillance:
Clinical case definition
A case with fever 38.5°C and one or more of the following:
• neck stiffness
• severe unexplained headache
• neck pain and 2 or more of the following: photophobia, nausea, vomiting, abdominal pain, pharyngitis with exudates
For children <2 years of age a case is defined as
• A case with fever 38.5°C and one or more of the following: irritability, bulging fontanelle
Laboratory criteria for confirmation
• The specific virus confirmed on cell culture.
Case classification
Suspected: A case that meets the clinical case definition and one or more of the following:
• normal CSF glucose and normal or mild increase in CSF protein (>50 mg/dl), moderate increase CSF cells (<500/mm3) and lymphocyte predominance (>50%)
• CSF Positive for viral genomic sequences using PCR (Polymerase Chain Reaction)
• Epidemiological link to a confirmed case
Confirmed: A suspected or probable case with laboratory confirmation.

As a group, the viral meningitides are characterized by fever, headache, meningismus and lymphocytic pleocytosis. [1] [2]
• Major complications and sequelae are unusual. [3] [4]
• The cerebrospinal fluid glucose level is normal, and a transitory neutrophilic pleocytosis is occasionally encountered.
• CSF pleocytosis is often absent among children with enteroviral meningitis. [5] [6]

This disease is endemic or potentially endemic to all countries.

Meningitis - aseptic (viral) in Brazil

Incidence and Prevalence:
- During 1998 to 2003, Enterovirus 30 accounted for 85.2% of enteroviral meningitis, Coxsackievirus B5 3.7%, Echovirus 13 3.7%, Echovirus 18 3%, Echovirus 6 1.2%, Echovirus 25 1.2%, Echovirus 1 0.6%, and Echovirus 4 0.6%. [7]
- Enteroviruses were found in 11.34% of CSF specimens from cases of suspected viral CNS infection (Sao Paulo, 2006 publication) [8]
- Enteroviruses were found in 19.8% of aseptic meningitis in Para State (2005 to 2006) [9]
- During 2002 to 2003, Enteroviruses accounted for 10.7% of CSF samples from meningitis cases (10.3% Echovirus 30, 0.4% Coxsackie B virus) (Belem, Para state) [10]

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- 29 cases of Enterovirus 30 meningitis were identified in Belem during 2002 to 2003 [11] ; 20 in Para during 2005 to 2006. [12]

Notable outbreaks:

1978 - An outbreak of viral meningitis was reported in Rio de Janeiro. [13]
1983 (publication year) - An outbreak of Echovirus 9 meningitis was reported in Rio de Janeiro. [14]
1997 - An outbreak (58 cases) of vaccine-related mumps meningitis was reported. [15] [16]
2003 - An outbreak (17 cases, 0 fatal) of aseptic meningitis due to Echovirus 13 was reported in Rio Grande do Sul. [17]
2005 - An outbreak (22 cases confirmed) of Echovirus 30 meningitis was reported in Rio de Janeiro State. [18] [19]

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Meningitis - bacterial

Agent	BACTERIUM. Neisseria meningitidis, Streptococcus pneumoniae, Haemophilus influenzae, et al
Reservoir	Human
Vector	None
Vehicle	Air Infected secretions
Incubation Period	Variable
Diagnostic Tests	CSF microscopy and culture. Blood culture. Note: Antigen detection is non-specific and rarely useful.
Typical Adult Therapy	Bactericidal agent(s) appropriate to known or suspected pathogen + dexamethasone
Typical Pediatric Therapy	As for adult
Vaccines	H. influenzae (HbOC-DTP or -DTaP) Haemophilus influenzae (HbOC) Haemophilus influenzae (PRP-D) Haemophilus influenzae (PRP-OMP) Haemophilus influenzae (PRP-T) Meningococcal Hepatitis B + Haemoph. influenzae
Clinical Hints	Headache, stiff neck, obtundation, high fever and leukocytosis; macular or petechial rash and preceding sore throat suggest meningococcal infection.
Synonyms	Bacterial meningitis, Enfermedad Meningococica, Haemophilus influenzae, Haemophilus influenzaes, HIB meningitis, HIBs, Infections a meningocoque, Meningite batterica, Meningite meningococcica, Meningococcal, Meningokokken Erkr., Meningokokkose. ICD9: 036.0,320 ICD10: A39,G00,G01,G02

Clinical

WHO Case definition for surveillance of Meningococcal infection:
Clinical case definition
• An illness with sudden onset of fever (>38.5°C rectal or >38.0°C axillary) and one or more of the following:
• Neck stiffness
• Altered consciousness
• Other meningeal sign or petechial or purpuric rash
• In patients <1 year, suspect meningitis when fever accompanied by bulging fontanelle.
Laboratory criteria for diagnosis
• Positive CSF antigen detection or
• Positive culture
Case classification
• Suspected: A case that meets the clinical case definition.
• Probable: A suspected case as defined above and turbid CSF (with or without positive Gram stain) or ongoing epidemic and epidemiological link to a confirmed case
• Confirmed: A suspected or probable case with laboratory confirmation.

WHO Case definition for surveillance of *Haemophilus influenzae* type b (Hib disease):
Clinical description
• Bacterial meningitis is characterized by fever of acute onset, headache and stiff neck.
• Meningitis is not a specific sign for Hib disease, and Hib disease cannot be diagnosed on clinical grounds.
Laboratory criteria for diagnosis
• Culture: isolation of Hib from a normally sterile clinical specimen, such as cerebrospinal fluid (CSF) or blood.
• Culture of Hib from non-sterile sites such as the throat, where bacteria can grow without causing disease, does not define Hib disease.
• Antigen detection: identification of Hib antigen in normally sterile fluids, by methods such as latex agglutination or counter-immunoelectrophoresis (CIE).
Case classification
• Potential: (bacterial meningitis case): a child with a clinical syndrome consistent with bacterial meningitis.
• Probable: Not applicable.
• Confirmed: A case that is laboratory-confirmed (growth or identification of Hib in CSF or blood).

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Note: Any person with Hib isolated from CSF or blood may be reported as a confirmed case, regardless of whether their clinical syndrome was meningitis.

As a group, the bacterial meningitides are characterized by signs of sepsis, fever, headache, meningismus and neutrophilic pleocytosis. [1] [2]

- 33% to 69% of patients with meningococcal infection have hyperglycemia on admission [3] [4]
- 7.5% of patients with meningococcal infection present with arthritis. [5]
- Major complications and sequelae are common.
- Delayed cerebral thrombosis is encountered in 1.1% of cases. [6]

This disease is endemic or potentially endemic to all countries.

Meningitis - bacterial in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Routine administration of meningococcal C conjugate vaccines was introduced in 2010. [7]

128,244 cases of meningitis (all causes) were reported during 1987 to 1991.

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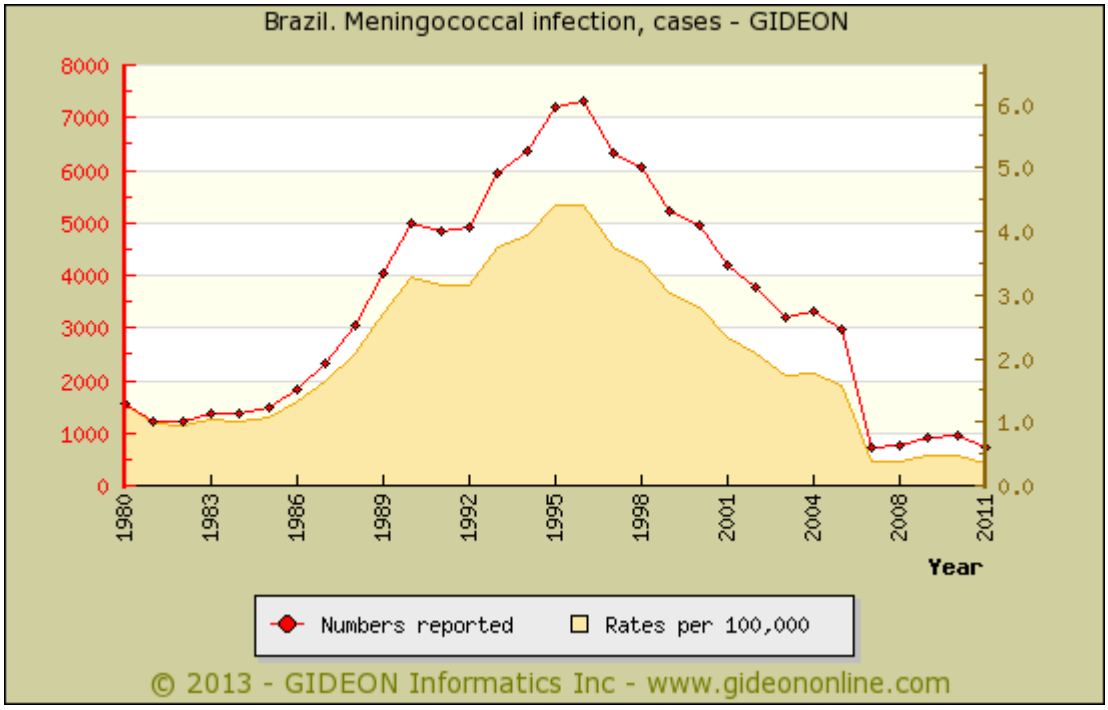
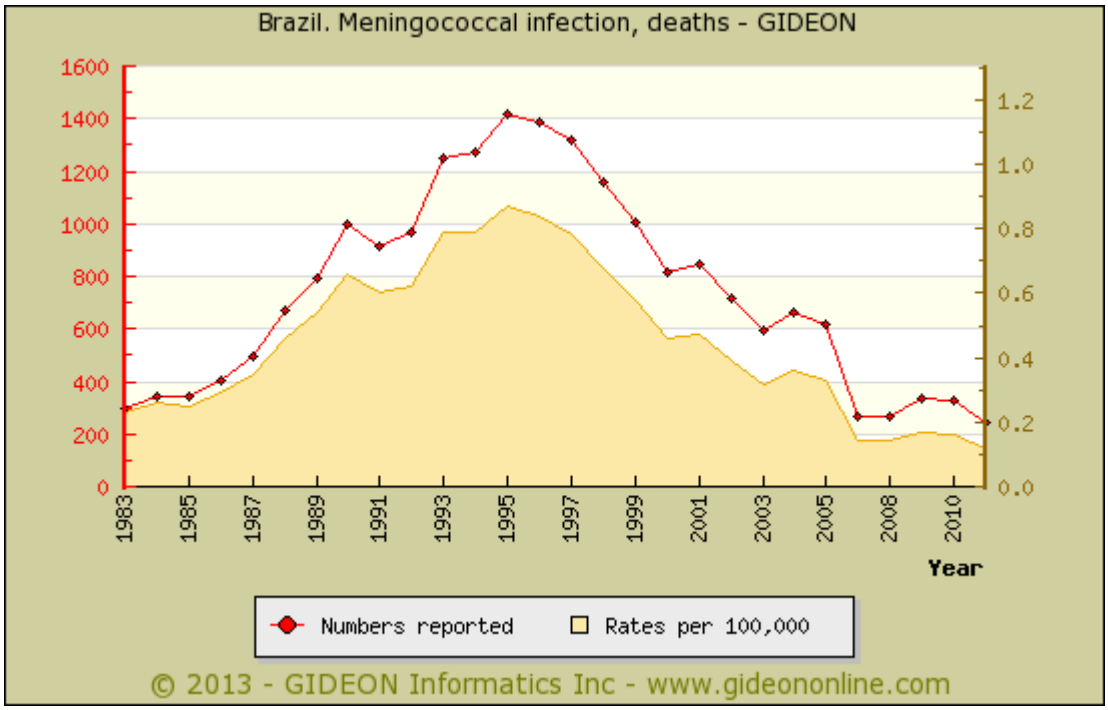


Graph: Brazil. Meningococcal infection, cases

Notes:
1. 80,683 cases of meningococcal infection were reported during 1980 to 2000.



Graph: Brazil. Meningococcal infection, deaths

Recent outbreaks of meningococcal disease have been reported, with mortality rates of 0.3 per 100,000 in 1986.
- Epidemics of type C disease were reported during 1971 to 1972; and of type A in 1974.
- Serotype B has predominated over types A and C since 1979 [8] ; however during an epidemic in Rio de Janeiro during 1995, 57% of isolates were type C and 42% type B. [9]
- Serotype B accounted for 67% of invasive *N. meningitidis* during 1990 to 2001, and serotype C 30%. [10]

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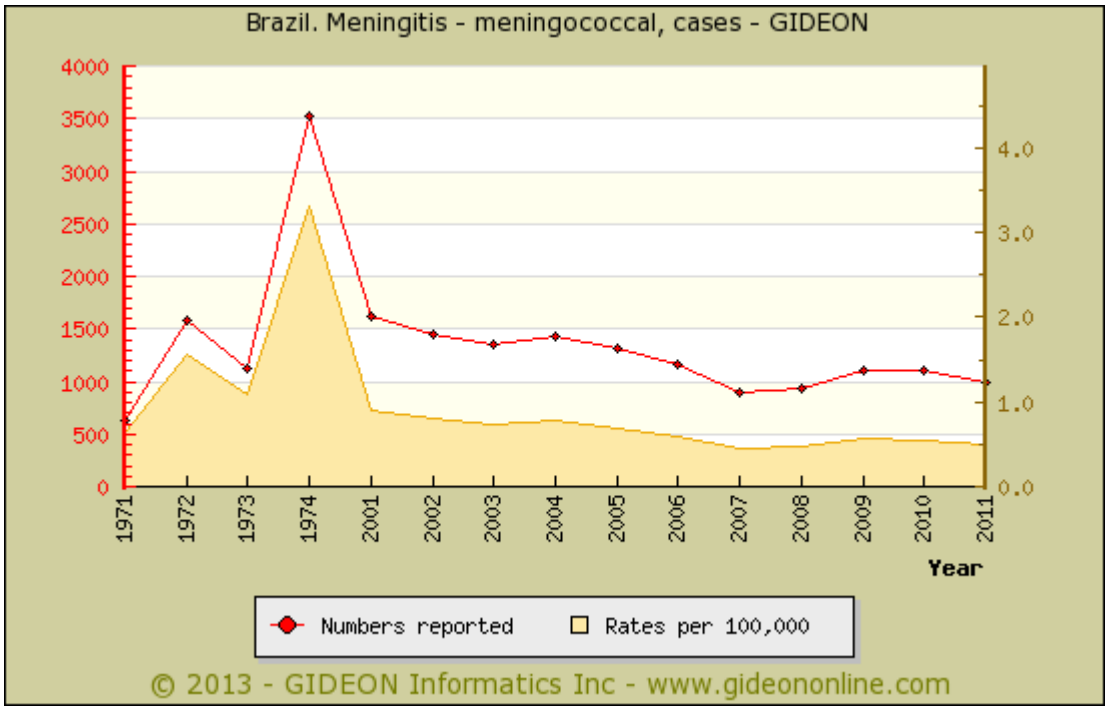
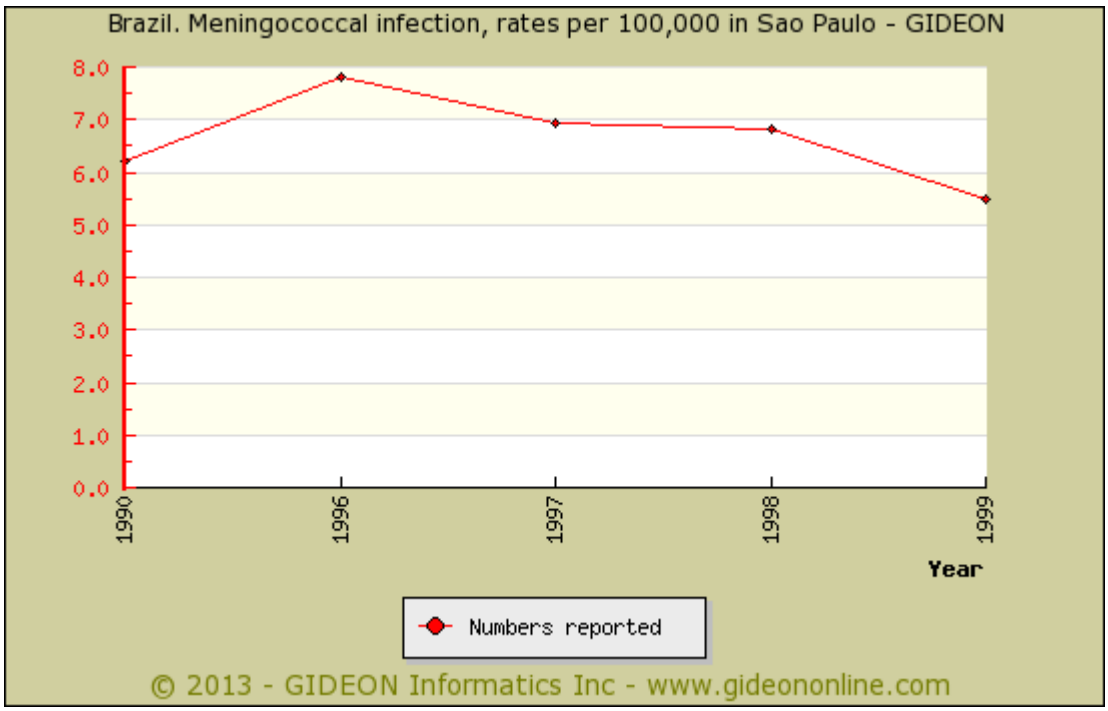
- The relative frequency of type C has been increasing in the South and Southeast since 1987.



Graph: Brazil. Meningitis - meningococcal, cases

Notes:
1. 408 cases (32 fatal) of meningococcal meningitis were reported in Salvador during February 1996 to January 2001 - 82% serogroup B. [11]
Individual years:
1971 - *N. meningitidis* type A predominated.



Graph: Brazil. Meningococcal infection, rates per 100,000 in Sao Paulo

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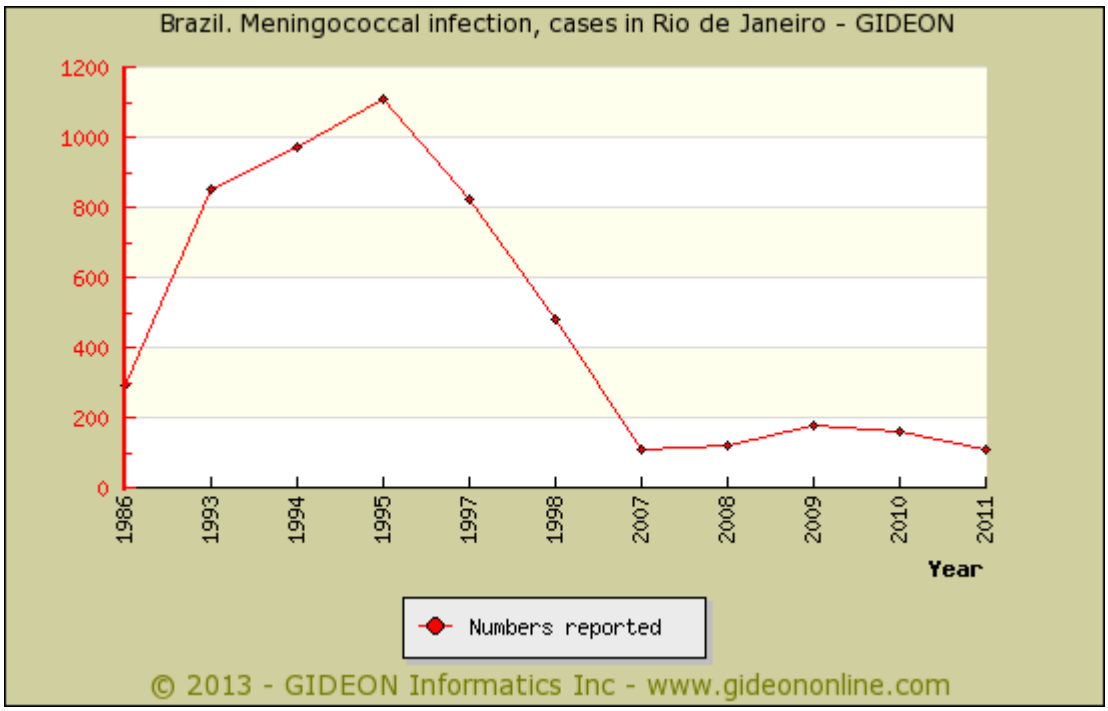
Notes:
1. Sao Paulo accounted for 31% of all meningococcal infections reported during 1980 to 2000.
2. A large outbreak of type B disease has been ongoing in Sao Paulo since 1988. [12]
3. 834 isolates of group B meningococci were confirmed in the city during 1990 to 1996. [13]
4. 1,167 cases of meningococcal disease were reported in Sao Paulo in 1997; 1,152 in 1998.
   Individual years:
   1990 - 50% *N. meningitidis* type B.
   1997 - 40.8% *N. meningitidis* type B.
   1998 - 53.2% *N. meningitidis* type B.



Graph: Brazil. Meningococcal infection, cases in Rio de Janeiro

Notes:
   Individual years:
   1995 - 40% of patients below age 5, mortality rate 16%. [14]
   1997 - 6.08 per 100,000. 16.9% of isolates group B.
   1998 - 3.52 per 100,000; 37/2% of isolates group B.

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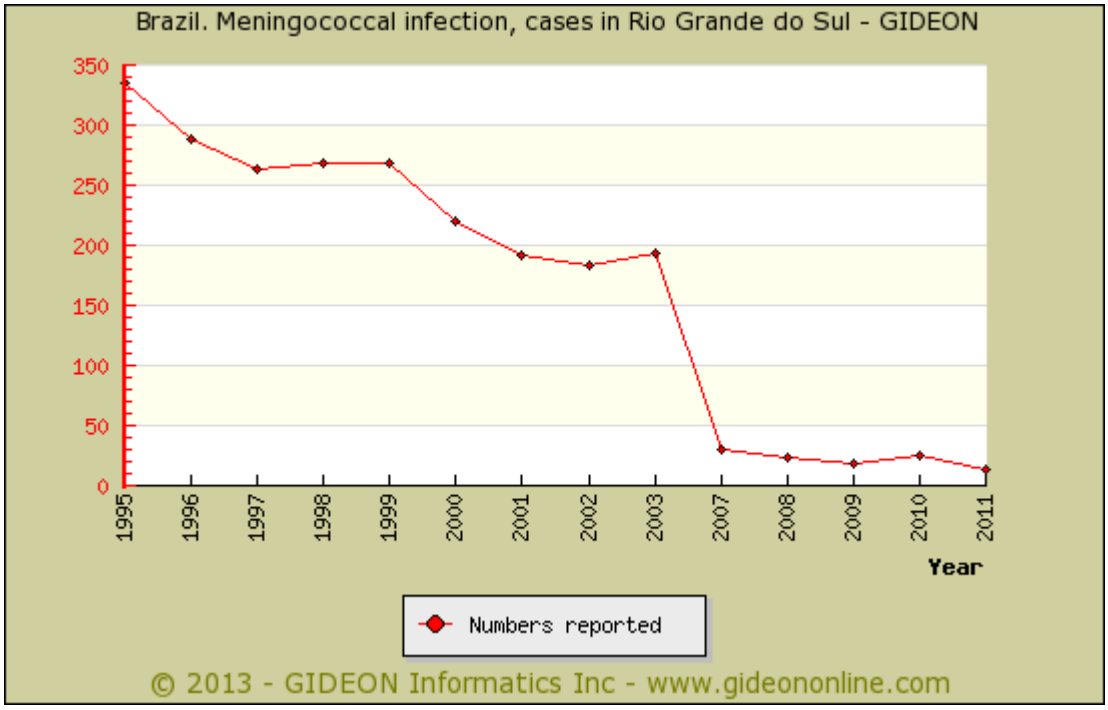
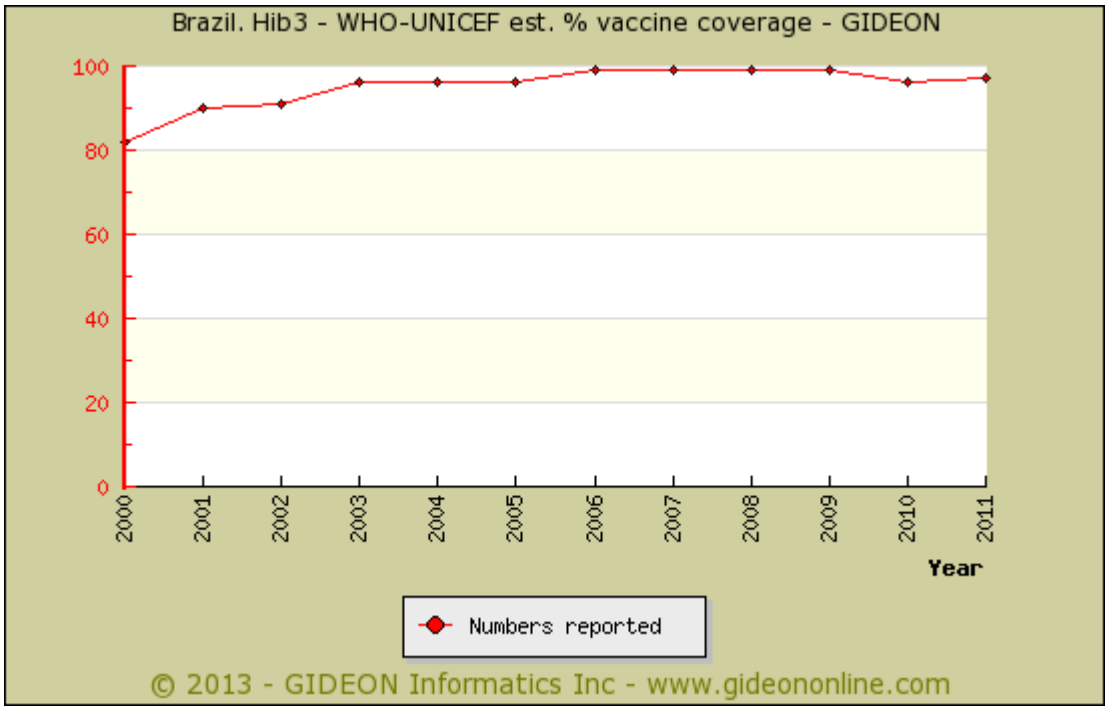


Graph: Brazil. Meningococcal infection, cases in Rio Grande do Sul

Notes:
1. 2,215 cases were reported during 1995 to 2003 - 79% type B, 14.1% type C, 6.2% type W135. [15]



Graph: Brazil. Hib3 - WHO-UNICEF est. % vaccine coverage

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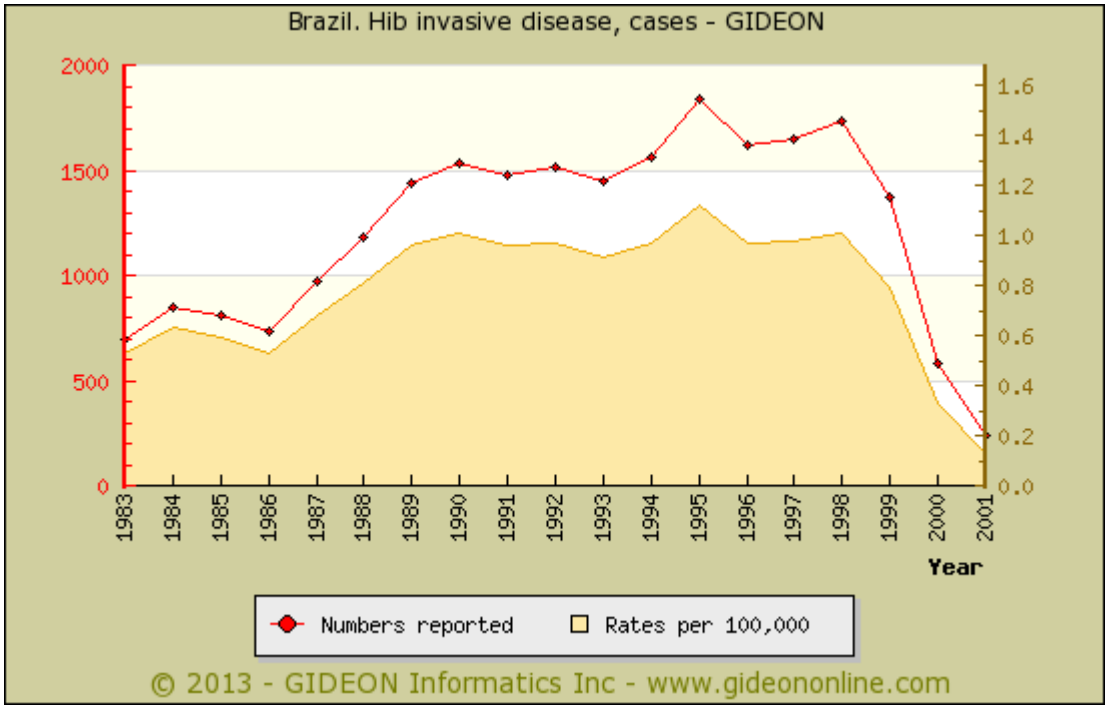
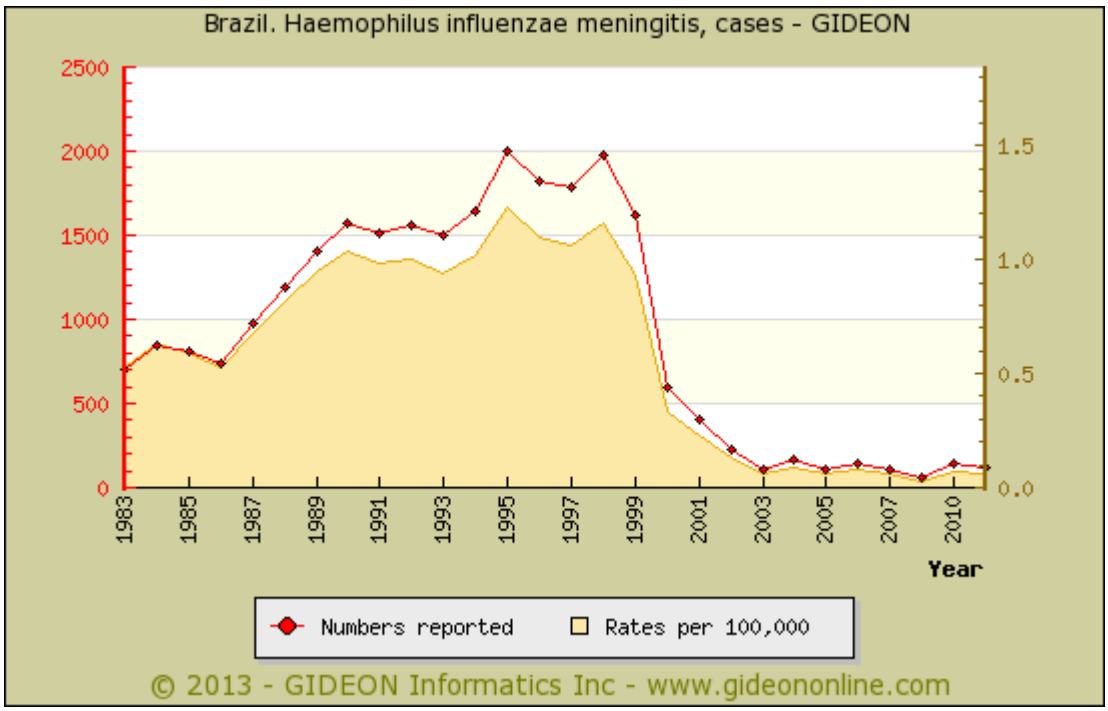


Graph: Brazil. Hib invasive disease, cases

Notes:
1. 22,961 cases were reported during 1983 to 2000.
2. 1987 - Case/fatality rate 22%.



Graph: Brazil. *Haemophilus influenzae* meningitis, cases

Notes:
1. 6,342 cases of *Haemophilus influenzae* meningitis were reported during 1987 to 1991, accounting for 5% of all meningitis reported during this period.
2. 1991 - 18.4 cases of *Haemophilus influenzae* meningitis per 100,000 children below age 1.
3. Following introduction of routine vaccination, Hib meningitis rates in Metropolitan Salvador decreased from 60.9 per 100,000 (1996) to 3.1 per 100,000 (2004). [16]

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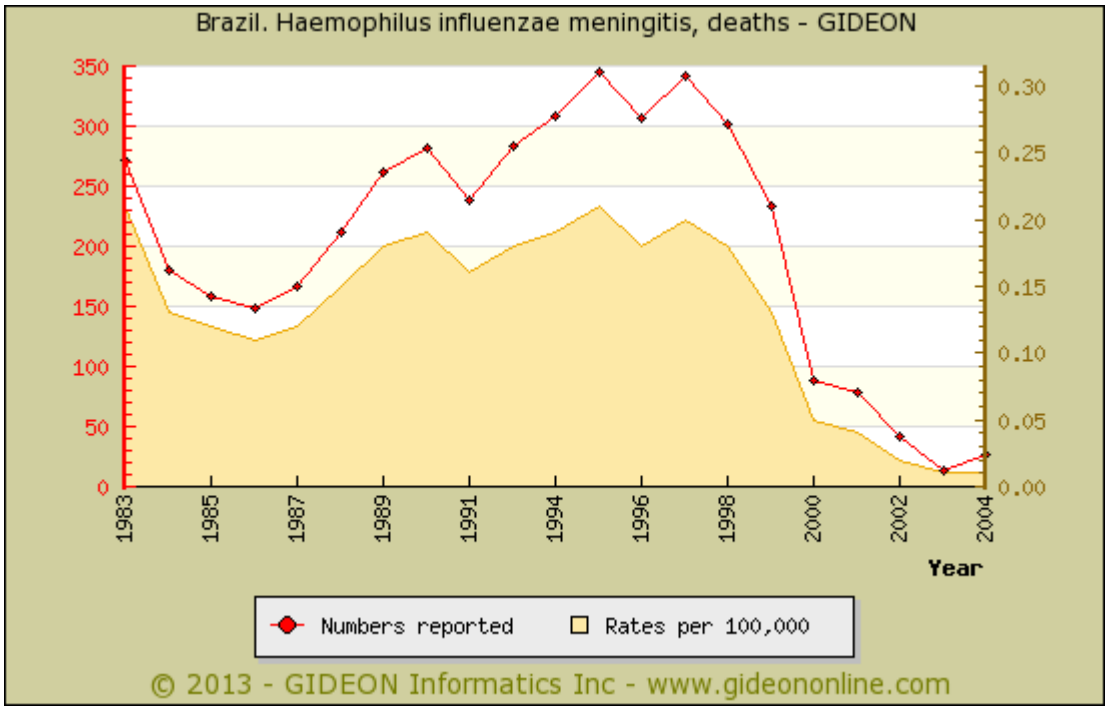

Graph: Brazil. *Haemophilus influenzae* meningitis, deaths

Notable outbreaks:

1971 to 1975 - An outbreak of group C meningococcal disease was reported in Sao Paulo. [17] [18]

1980 (publication year) - An outbreak of meningococcal infection was reported in Parana. [19-22]

1988 to 1990 - An outbreak of group B meningococcal disease was reported in Sao Paulo. [23-25]

1990 to 1991 - An outbreak of group C meningococcal disease was reported in Curitiba, Parana State. [26]

2001 - An outbreak (8 cases) of group C meningococcal disease was reported in northern Santa Catarina State. [27]

2008 - An outbreak (8 cases, 5 fatal) of group C meningococcal disease was reported among workers at a food-processing plant. [28]

2009 - An outbreak (1,445 cases, 138 fatal) of group C meningococcal meningitis was reported in Bahia. [29-31]

2010 - An outbreak (18 cases) of group C meningococcal disease was associated with an oil refinery in Sao Paulo. [32]

2010 to 2011 - An outbreak (1,670 cases, 109 fatal) of meningococcal meningitis was reported in Bahia. [33] [34]

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Microsporidiosis

Agent	PARASITE - Protozoa. Microspora: Enterocytozoon, Encephalitozoon (Septata), Vittaforma (Nosema), Pleistophora, Trachipleistophora, et al.
Reservoir	Rabbit Rodent Carnivore Non-human primate Fish Dog Bird
Vector	None
Vehicle	? Fecal-oral
Incubation Period	Unknown
Diagnostic Tests	Microscopy of duodenal aspirates. Inform laboratory if this organism is suspected. Nucleic acid amplification.
Typical Adult Therapy	Albendazole 400 mg PO BID X 3 weeks. Add Fumagillin for ocular S. intestinalis may respond to Albendazole and Fumagillin Nitazoxanide has been used for E. bieneusi.
Typical Pediatric Therapy	Albendazole 200 mg PO BID X 3 weeks. Add Fumagillin for ocular S. intestinalis may respond to Albendazole and Fumagillin Nitazoxanide has been used for E. bieneusi.
Clinical Hints	In AIDS patients, infection is characterized by chronic diarrhea, wasting and bilateral keratoconjunctivitis; hepatitis and myositis may be present.
Synonyms	Brachiola, Encephalitozoon, Enterocytozoon, Microsporidium, Nosema, Pleistophora, Trachipleistophora, Tubulinosema, Vittaforma. ICD9: 136.8 ICD10: A07.8

Clinical

Intestinal disease in immunocompetent patients is characterized by self-limited diarrhea, traveler's diarrhea or asymptomatic carriage. [1]

• Immunocompromised patients present with diarrhea, cholangitis, cholecystitis, sinusitis or pneumonia. [2] [3]

Ocular microsporidiosis is associated with keratoconjunctivitis.

Other syndromes include sinusitis, nephritis, myositis and prostatitis. [4]

This disease is endemic or potentially endemic to all countries.

Microsporidiosis in Brazil

Prevalence surveys:

27.5 to 46% of HIV-positive patients with diarrhea (*Enterocytozoon bieneusi*) (2005 publication) [5]

1.3% of immunosuppressed patients in Goias, Goiana (2006 publication) [6]

36% of patients undergoing anti-tumor necrosis factor/disease-modifying anti-rheumatic drug treatment. for rheumatic diseases, vs. 4% of controls (2011 publication) [7]

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Moniliformis and Macracanthorhynchus

Agent	PARASITE - Archiacanthocephala. Moniliformida: Moniliformis moniliformis, Oligocanthorhynchida: Maracanthorhynchus hirudinaceus.
Reservoir	Pig (Maracanthorhynchus), rat and fox (Moniliformis),
Vector	None
Vehicle	Insect (ingestion)
Incubation Period	Unknown - presumed 15 to 40 days
Diagnostic Tests	Identification of worm in stool.
Typical Adult Therapy	Infection is usually self-limited. Pyrantel pamoate has been used against Moniliformis moniliformis - 11 mg/kg PO - repeat once in 2 weeks
Typical Pediatric Therapy	Infection is usually self-limited. Pyrantel pamoate has been used against Moniliformis moniliformis - 11 mg/kg PO - repeat once in 2 weeks
Clinical Hints	Most infections are characterized by asymptomatic passage of a worm; however, vague complaints such as 'periumbilical discomfort' and 'giddiness' have been described.
Synonyms	Acanthocephalan worms, Macracanthorhynchus, Moniliform acanthocephalan, Moniliformis moniliformis. ICD9: 128.9 ICD10: B83.8

Clinical

Most infections are characterized by asymptomatic passage of a worm; however, vague complaints such as 'periumbilical discomfort' and 'giddiness' have been described. [1]

- In one instance, a man developed marked abdominal pain following experimental self-infection.
- In another case, intestinal perforation was associated with *Macracanthorhynchus hirudinaceus* infestation. [2]

This disease is endemic or potentially endemic to all countries.

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Mumps

Agent	VIRUS - RNA. Paramyxoviridae, Paramyxovirinae, Rubulavirus: Mumps virus
Reservoir	Human
Vector	None
Vehicle	Aerosol
Incubation Period	14d - 24d (range 12d - 24d)
Diagnostic Tests	Viral culture (saliva, urine, CSF) indicated only in complicated cases. Serology. Nucleic acid amplification.
Typical Adult Therapy	Respiratory isolation; supportive
Typical Pediatric Therapy	As for adult
Vaccines	Measles-Mumps-Rubella Mumps Rubella - Mumps
Clinical Hints	Fever, parotitis, orchitis (20% of post-pubertal males), meningitis (clinically apparent in 1% to 10%), oophoritis, or encephalitis (0.1%); most cases resolve within 1 to 2 weeks.
Synonyms	Bof, Epidemic parotitis, Fiebre urliana, Infectious parotitis, Kusma, Oreillons, Paperas, Parotidite epidemica, Parotiditis, Parotite epidemica, Passjuka. ICD9: 072 ICD10: B26

Clinical

One third of Mumps virus infections are asymptomatic.

Acute illness:
The prodrome of mumps consists of low-grade fever, anorexia, malaise, and headache.
• Usually within one cay, the patient complains "earache" and tenderness is noted over the parotid gland. [1]
• The gland is soon visibly enlarged and progresses to maximum size over the next 2 to 3 days, often with lifting of the ear lobe upward and outward.
• The orifice of Stensen's duct is edematous and erythematous, and trismus and pain on chewing may be present.
• It is important to remember that the enlarged gland obscures the angle of the mandible, while cervical adenopathy does not.
• Parotid involvement if unilateral in 25% of cases.
• As the disease progresses, fever may reach 40C.
• Subsequently pain, fever, and tenderness resolve, and the parotid gland returns to normal size within a week.
• Involvement of the other salivary glands occurs in 10% of cases, but are rare in the absence of parotid involvement.
• Presternal edema develops in 6% of patients, most often in those who have submandibular adenitis.

8% to 15% of patients will continue shedding Mumps virus 5 days after the onset of symptoms. [2]

Neurological manifestations:
Central nervous system involvement is the most common extrasalivary gland manifestation of this disease.
• Cerebrospinal fluid pleocytosis has been documented in 51% patients with mumps, without other evidence of meningitis.
• Clinical meningitis occurs in 1 to 10% of persons with mumps parotitis; while parotitis is documented in less than 50% of patients with mumps.
• Meningitis may occur before, during or after salivary gland involvement.
• The features of mumps meningitis are similar to those of other viruses, and the clinical course is benign; however, polymorphonuclear CSF pleocytosis and reduced glucose levels are not unusual.

Encephalitis occurs in less than 0.1% of cases, and may be accompanied by altered consciousness, seizures, paresis, aphasia, involuntary movements; and sequelae such as psychomotor retardation, deafness (1 per 1,000 to 20,000 cases [3]) [4] and convulsive disorders.
• Other neurological complications of mumps include cerebellar ataxia [5] , facial nerve palsy, transverse myelitis, Guillain-Barre syndrome, and aqueductal stenosis.

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Epididymo-orchitis:

Epididymo-orchitis is the most common extra-salivary gland manifestation in adults, developing in 20 to 30% of infected postpubertal males.
• This complication is bilateral in 15% of cases, and appears during the first week of mumps in 70% of cases.
• Rarely, this is the only manifestation of mumps.
• Onset is abrupt, with elevation of fever, chills, headache, vomiting, and testicular pain.
• The testis is warm, swollen (to as much as four times normal size), and tender, with erythema of the scrotum.
• Epididymitis is present in 85%, and usually precedes the orchitis.
• Tenderness may persist for more than 2 weeks in 20% of cases; and some degree of atrophy is noted in 50% of the patients, even after 2 years.
• Impotence is not encountered, and sterility is rare.

Additional manifestations of mumps:

Other features of mumps include oophoritis, fetal wastage [6] , migratory polyarthritis, monoarticular arthritis and arthralgia, electrocardiographic changes (with or without overt myocarditis), nephritis, thyroiditis, mastitis, prostatitis, hepatitis, cholecystitis and thrombocytopenia.

This disease is endemic or potentially endemic to all countries.

Mumps in Brazil

Vaccine Schedule:

BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Routine immunization using MMR at age 12 months was introduced in 2000.

Mumps, cases: None reported between 2001 and 2011

Notable outbreaks:

1997 - An outbreak (58 cases) of vaccine-related mumps meningitis was reported. [7] [8]

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Myalgic encephalomyelitis

Agent	UNKNOWN
Reservoir	Unknown
Vector	None
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Clinical diagnosis; ie, discount other diseases.
Typical Adult Therapy	Supportive; ? immune modulators (experimental)
Typical Pediatric Therapy	As for adult
Clinical Hints	Unexplained depression, fatigue, cognitive disorders, sleep disturbance, recurrent bouts of pharyngitis and adenopathy, rheumatological symptoms and fever lasting more than six months.
Synonyms	Chronic fatigue syndrome. ICD9: 780.71 ICD10: G93.3

Clinical

The CDC (The United States Centers for Disease Control) consensus definition of Chronic Fatigue Syndrome (currently Myalgic encephalomyelitis [1]) requires the presence of two major criteria, in addition to at least six symptom criteria and at least two physical criteria (or the presence of eight symptom criteria, without need for physical criteria) as follows: [2-6]

Major criteria:
A. New onset of persistent or relapsing, debilitating fatigue or fatigability without a history of similar illness. Fatigue does not resolve with bed rest, and reduces daily activity by at least 50% for at least 6 months.
B. Exclusion of other disorders through history, physical examination and laboratory studies.

Minor criteria:
A. Symptoms.
1. Mild fever or chills
2. Sore throat
3. Painful cervical or axillary adenopathy
4. Myalgia
5. Muscle weakness
6. Migratory arthralgia
7. Prolonged fatigue not meeting major criteria
8. Generalized headaches
9. Neuropsychological complaints (photophobia), scotomata, forgetfulness, irritability, confusion, problems in thinking or concentration [7-9] , depression)
10. Sleep disturbances
11. Description of the initial symptom complex as developing over a period of hours to days.

B. Physical criteria.
1. Low grade fever
2. Nonexudative pharyngitis
3. Cervical or axillary lymphadenopathy (nodes may be tender, and are usually no larger than 2 cm).

Affected children present with low levels of school attendance, fatigue, anxiety, functional disability and pain. [10]
• Three phenotypes of Chronic Fatigue Syndrome are described in children: musculoskeletal, migraine and "sore throat." [11]

Patients with disease onset above age 50 years present with relatively high rates of fatigue, depression and autonomic dysfunction. [12]

Additional findings described in Chronic fatigue syndrome have included generalized hyperalgesia [13] [14] , impaired cardiac function [15] , migraine headache [16] and postural orthostatic tachycardia. [17] [18]

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In one series, 33% of patients referred to an Infectious Diseases clinic for suspected Lyme disease were found to have Chronic Fatigue Syndrome, and only 23% Lyme disease. [19]

This disease is endemic or potentially endemic to all countries.

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Mycetoma

Agent	BACTERIUM OR FUNGUS. Nocardia spp, Madurella mycetomatis, Actinomadura pellitieri, Streptomyces somaliensis, et al
Reservoir	Soil Vegetation
Vector	None
Vehicle	Contact Wound Soil
Incubation Period	2w - 2y
Diagnostic Tests	Bacterial and fungal culture of material from lesion.
Typical Adult Therapy	Antimicrobial or antifungal agent as determined by culture. Excision as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Painless, chronic, draining, fistulous subcutaneous nodule - usually involving lower extremity; osteolytic lesions may be noted on x-ray; usually no fever; most patients are males age 20 to 40 (ie, occupational exposure).
Synonyms	Leptosphaeria tompkinsii, Madura foot, Madura-Fuss, Madurella, Mycetom, Pleurostomophora, White grain eumycetoma. ICD9: 039.4,117.4 ICD10: B47

Clinical

Mycetoma is typically characterized by a painless nodule or thickening, which involve the feet in 80% of cases. [1]
• The lesions slowly enlarge and form sinus tracts which drain bloody, serous or purulent fluid containing granules of various colors. [2]
• Systemic findings are absent.
• Lesional hyperhydrosis is common, and tendons and nerves are usually spared until late stages of the infection. [3]
• Regional lymphadenopathy is encountered in 1% to 3% of cases.
• Lupus vulgaris may mimic mycetoma. [4]

Hematogenous spread of infection is extremely rare. [5]
• Mycetoma may spread to involve contiguous bone or regional lymph nodes.
• In Actinomycotic infections, the course is more rapid and aggressive, with prominent inflammation and early destruction of bone.

Dark granules characterize Madurella infection, while pale colored granules are seen in Acremonium infection.
• *Actinomadura madurae*, *Nocardia brasiliensis*, and *Streptomyces somaliensis* produce smaller white, yellow, or brownish granules.

Rare instances of mycetoma of the scalp due to *Microsporum canis* have been reported. [6]
• Perianal actinomycetoma may mimic other chronic diseases of the anal region.
• Ocular mycetoma has been reported as a complication of a trauma [7] or sub-tenon injection [8]
• Rare cases of palatal [9], lingual [10], paranasal and cavernous sinus infection have been reported. [11] [12]
• The clinical features of mycetoma may mimic those of soft tissue tumors. [13]

Diagnosis is based on radiological and ultrasonic imaging, histology, culture and serology.
• A characteristic "dot in circle" sign may be seen on magnetic resonance imaging (MRI) studies. [14]
• Although Actinomycotic lesions may be amenable to antibiotic therapy, eumycetoma requires aggressive surgical excision.

This disease is endemic or potentially endemic to all countries.

Mycetoma in Brazil

41 cases of mycetoma were identified by active case-finding for Sao Paulo during 1978 to 1989.
- *Nocardia braziliensis* accounted for 13 of the cases. [15]

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Cases of mycetoma due to *Madurella grisea* [16] have been reported in the southern region [17] and northern region. [18]
- Cases of mycetoma are also reported from the Amazon region. [19]

A case of mycetoma caused by *Nocardia caviae* was reported in Minas Gerais (2010 publication) [20]

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Mycobacteriosis - M. marinum

Agent	BACTERIUM. Actinomycetes, Mycobacterium marinum An aerobic acid-fast bacillus
Reservoir	Fresh and salt water (eg, swimming pools, aquaria) Fish (ornamental, salmon, sturgeon, bass)
Vector	None
Vehicle	Water per areas of minor skin trauma
Incubation Period	5d - 270d (median 21d)
Diagnostic Tests	Mycobacterial culture from lesion. Alert laboratory when this organism is suspected.
Typical Adult Therapy	Rifampicin 600 mg/day + Ethambutol 20 mg/kg/day X 6w. Alternative: Minocycline
Typical Pediatric Therapy	Sulfamethoxazole/trimethoprim 5 mg-25 mg/kg BID X 6w. Alternative Minocycline (Age >= 8)
Clinical Hints	Violaceous papule, ulcer, plaque, psoriaform lesion; onset weeks after exposure (swimming pool, aquarium); commonly involves the elbow, knee, hand or foot.
Synonyms	Aquarium granuloma, Fish fanciers' finger syndrome, Fish tank granuloma, Mariner's TB, Mycobacterium balnei, Mycobacterium marinum, Mycobacterium scrofulaceum, Spam, Swimming pool granuloma. ICD9: 031.1 ICD10: A31.1

Clinical

The incubation period varies from 5 to 170 days (median 21 days); with 35% of cases exceeding 30 days.

- Characteristic painful, slowly-growing blue papules usually involve the extremities, and may ulcerate. [1]
- The upper extremities are involved in 75% to 95%, and spread to deeper structures (tendons, bones, joints) occurs in 29%. [2-5]
- Dissemination is rare, but has been described in AIDS patients. [6]
- Multiple sporotrichoid subcutaneous nodules have been reported. [7] [8]
- Extensive verrucous dermal plaques have been reported among Pacific Islanders infected by *Mycobacterium marinum*. [9] [10]
- Tenosynovitis ("fish-tank finger") is occasionally encountered. [11-14]
- A rare case of nasal infection presenting as epistaxis has been reported. [15]
- Scarring may occur, but is less pronounced than that which follows *M. ulcerans* infection.

This disease is endemic or potentially endemic to all countries.

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Mycobacteriosis - M. scrofulaceum

Agent	BACTERIUM. Actinomycetes, Mycobacterium scrofulaceum An aerobic acid-fast bacillus
Reservoir	Water (lakes, rivers) Soil Raw milk Plant material
Vector	None
Vehicle	Water Soil ? Through areas of minor trauma
Incubation Period	Unknown
Diagnostic Tests	Culture of tissue or aspirates.
Typical Adult Therapy	Excision. Drugs (Isoniazid - Rifampin - streptomycin - Cycloserine) are rarely indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Painless lymphadenopathy, most commonly unilateral and submandibular (true tuberculosis involves the lower neck and produces a strongly positive tuberculin reaction and/or suggestive chest X ray). The disease is most common during early childhood.
Synonyms	

Clinical

Mycobacterium scrofulaceum is a common cause of lymphadenitis, most commonly among children ages 1 to 3 years.
• Most infections involve the submandibular region, however involvement of other lymph node groups or body organs may occur. [1]
• Rare instances of dissemination are reported. [2] [3]

This disease is endemic or potentially endemic to all countries.

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Mycobacteriosis - M. ulcerans

Agent	BACTERIUM. Actinomycetes, Mycobacterium ulcerans An aerobic acid-fast bacillus
Reservoir	Vegetation
Vector	Mosquitoes (probable)
Vehicle	Contact
Incubation Period	3w - 3m
Diagnostic Tests	Mycobacterial culture from lesion. Alert laboratory that this organism is suspected.
Typical Adult Therapy	Rifampin + amikacin. OR Ethambutol + Sulfamethoxazole/trimethoprim X 6w. OR Rifampin + a fluoroquinolone. Excision as indicated
Typical Pediatric Therapy	Rifampin 20 mg/kg/day + amikacin 7.5 mg/kg q12h X 6w. Excision as indicated
Clinical Hints	Slowly growing, painless ulcerative nodule with undermined edges - lymphadenopathy usually not present; in most cases, a single leg lesion involving the extensor surface (face and trunk may be involved in children).
Synonyms	Bairnsdale ulcer, Buruli ulcer, Kakerifu ulcer, Kasongo ulcer, Kisikro, Kumusi ulcer, Mexican ulcer, Mycobacterium buruli, Mycobacterium ulcerans, Searl's ulcer, Tora ulcer, Ulcerans disease. ICD9: 031.1 ICD10: A31.1

Clinical

Buruli ulcer is a chronic, indolent, necrotizing disease of the skin. [1]
• Lesions usually involve the limbs (the face [2] and trunk in children); 60% involve the lower limbs, 30% upper limbs and 10% other body regions. [3-5]
• The process begins as firm, nontender subcutaneous nodules 1 to 2 cm in diameter at sites of penetrating skin trauma.
• Within the next 1 to 2 months, these areas become fluctuant, followed by the formation of a painless, undermined ulceration. [6]

Ulcerations may involve up to 15% of the patient's skin surface, and may destroy nerves and blood vessels, or even invade bone.
• Some cases may present as dermal plaques or cellulitis, without ulcer.
• Most lesions eventually heal spontaneously, but frequently result in chronic lymphedema and disfiguring scarring. [7] [8]
• Skin lesions may re-emerge as long as one year following therapy, possibly as a result of immune reconstitution. [9]
• Severe infection may induce contracture and atrophy of contiguous skeletal muscles. [10]
• Systemic symptoms are rare, and healing may take 4 to 6 months, with extensive scar formation and limb deformity.
• Rare instances of systemic spread [11] and multifocal osteomyelitis have been reported. [12]
• Hemoglobinopathy appears to be a risk factor for *Mycobacterium ulcerans* osteomyelitis [13]

This disease is endemic or potentially endemic to 37 countries. Although Mycobacteriosis - M. ulcerans is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Mycobacteriosis - M. ulcerans in Brazil

A single case report of *Mycobacterium ulcerans* infection in Brazil was published in 2007. [14] [15]

A British traveler acquired Buruli ulcer - presumably in the Brazilian Pantanal. [16]

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Mycobacteriosis - miscellaneous nontuberculous

Agent	BACTERIUM. Actinomycetes, Mycobacterium spp. An aerobic acid-fast bacillus
Reservoir	Water Soil Fish Mammal Bird
Vector	None
Vehicle	Air Water Contact Ingestion Trauma
Incubation Period	Variable
Diagnostic Tests	Microscopy & culture of tissue, secretions, blood. Nucleic acid amplification. Inform laboratory if suspected
Typical Adult Therapy	Drug, route and duration appropriate to clinical setting and species [in Therapy module, scroll through upper left box]
Typical Pediatric Therapy	As for adult
Clinical Hints	Pneumonia, or chronic granulomatous infection of various tissues; systemic disease may complicate immune suppression; *M. avium-intracellulare* characterized by aggressive course and resistance to most antimycobacterial drugs.
Synonyms	Mycobacterium abscessus, Mycobacterium avium, Mycobacterium avium-intracellulare, Mycobacterium franklinii, Mycobacterium immunogenum, Mycobacterium jacussii, Mycobacterium xenopi, Segniliparus. ICD9: 031.9,031.2 ICD10: A31.0,A31.1,A31.8

Clinical

The clinical features of systemic mycobacterial infection are protean, and can involve disease of virtually any organ or tissue. [1-5]

• Specific syndromes reflect the immune status of the patient and the specific fungal species involved (see Worldwide note)

Mycobacterium avium-intracellulare infection is clinically similar to tuberculosis, producing localized pulmonary disease [6] or disseminated lesions of virtually any organ. [7] [8]
- Bacteremia is common, and can be detected using specialized blood culture systems.

Mycobacterium kansasii infection is characterized by productive cough, dyspnea, and chest pain.
• 16% of patients are asymptomatic.
• A right sided, apical or subapical, thin walled cavitary infiltrate is characteristic. [9]

Mycobacterium malmoense infection is usually characterized by pulmonary disease suggestive of tuberculosis, or pediatric cervical lymphadenopathy. [10]

Note: Over 110 species of *Mycobacterium* have been associated with human infection.
• See Microbiology • Mycobacteria module

This disease is endemic or potentially endemic to all countries.

Mycobacteriosis - miscellaneous nontuberculous in Brazil

Prevalence surveys:

10% of submandibular and 7% of sublingual lymphadenopathy in advanced AIDS patients (2009 publication) [11]

Notable outbreaks:

2000 - An outbreak (10 cases) of *Mycobacterium chelonae* keratitis was associated with laser in situ keratomileusis (LASIK). [12]

2002 to 2004 - An outbreak (14 cases) of mycobacterial wound infection associated with silicone breast implant surgery was reported in Campinas, Sao Paulo. [13] [14]

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2003 - An outbreak (5 cases) of *Mycobacterium immunogenum* keratitis in Sao Paulo was associated with myopia surgery. [15]

2004 to 2005 - An outbreak (311 cases) of *Mycobacterium abscessus* infections in Belem was associated with invasive procedures. [16] [17] *Mycobacterium massiliense* and *M. bolletii* were also identified in some cases. [18-20]

2006 to 2007 - An outbreak (1,051 clinical cases) of *Mycobacterium massiliense* infections involving 63 hospitals in Rio de Janeiro state was related to contaminated instruments used in video-assisted surgery. [21] [22]

2008 - An outbreak (13 cases) of *Mycobacterium fortuitum* infections in Sao Paulo was associated with breast implant surgery. [23]

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Mycoplasma (miscellaneous) infections

Agent	BACTERIUM. Mycoplasmatales Mycoplasma genitalium, Mycoplasma hominis, Mycoplasma fermentans, Mycoplasma penetrans, Ureaplasma urealyticum
Reservoir	Human
Vector	None
Vehicle	Secretion, Sexual transmission
Incubation Period	Unknown
Diagnostic Tests	Culture (urine, pharynx). Serology. Nucleic acid amplification.
Typical Adult Therapy	Azithromycin 1 g orally as single dose OR Doxycycline 100 mg PO BID X 7 days OR Levofloxacin 500 mg daily X 7 days OR Ofloxacin 300 mg BID X 7 days
Typical Pediatric Therapy	Erythromycin 10 mg/kg PO QID X 2w
Clinical Hints	Urethritis, vaginitis, neonatal pneumonia; rarely stillbirth, prematurity or infertility
Synonyms	Acholeplasma laidlawii, Epirythrozoon, Hemotrophic Mycoplasma, Mycoplasma amphoriforme, Mycoplasma buccale, Mycoplasma faucium, Mycoplasma felis, Mycoplasma fermentans, Mycoplasma genitalium, Mycoplasma hominis, Mycoplasma lipophilum, Mycoplasma orale, Mycoplasma penetrans, Mycoplasma pirum, Mycoplasma primatum, Mycoplasma salivarium, Mycoplasma spermatophilum, T Mycoplasmas, T strains, Ureaplasma parvum, Ureaplasma urealyticum. ICD9: 041.81 ICD10: A49.3

Clinical

Asymptomatic pharyngeal and vaginal carriage of *Mycoplasma* species and *Ureaplasma* is common.
• As many as 70% of sexually-active persons are colonized.

The signs and symptoms of infection are similar to those of *Chlamydia* infection. [1]
• Urogenital infection may present as vaginitis, cervicitis, non-gonococcal urethritis, epididymitis [2] , prostatitis [3] or urethral discharge.
• Less common findings may include pelvic inflammatory disease [4-6] , post-partum fever [7] [8] , chorioamnionitis, infertility [9] , prematurity [10] and stillbirth. [11-14]
• Bronchitis, arthritis [15] [16] , neonatal meningitis and encephalitis [17-19] , osteitis [20] , endocarditis [21] [22] , brain abscess [23] , soft tissue infections [24] , genital under disease [25] , bacteremia [26] , respiratory distress in the newborn [27] and pneumonia have been reported. [28-31]

Infection by hemotrophic *Mycoplasma* species (formerly *Epirythrozoon*) is characterized by fever, anemia and hemolytic jaundice • notably among pregnant women and newborns. [32]

This disease is endemic or potentially endemic to all countries.

Mycoplasma (miscellaneous) infections in Brazil

Prevalence surveys:

28.3% of men undergoing routine semen analysis were found to be infected by *Ureaplasma urealyticum*, and 24.8% *Mycoplasma hominis* (2003 publication) [33]

20% of asymptomatic women were found to be colonized by *Ureaplasma urealyticum* (*Mycoplasma hominis* 6.7%); 36% (4% *M. hominis*) of women with cervicitis and 56.8% (5.4% *M. hominis*) (Sao Paulo, 1985 to 1986) [34]

Mycoplasma genitalium was found in cervical specimens from patients attending public health services in Belo Horizonte and Contagem, Minas Gerais, *M. hominis* 21.9% and *Ureaplasma* spp. 38.4% (2011 publication) [35]

Mycoplasma hominis was found in 2.1% of HIV-positive women in Salvador, Bahia, *U. urealyticum* in 2.1% (2012 publication) [36]

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Mycoplasmataceae were found in 0% of Fallopian tubes of infertile women, vs. 10% of fertile women (2012 publication) [37]

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Mycoplasma pneumoniae infection

Agent	BACTERIUM. Mollicutes. Mycoplasma pneumoniae
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	6d - 23d
Diagnostic Tests	Culture (sputum, throat). Serology. Nucleic acid amplification.
Typical Adult Therapy	Erythromycin 500 mg PO BID X 2w. OR Azithromycin 1 g, followed by 500 mg PO daily X 5 days. OR Doxycycline 100 mg PO BID
Typical Pediatric Therapy	Erythromycin 10 mg/kg PO QID X 2w
Clinical Hints	Coryza, "hacking" cough; subsegmental infiltrate; bullous otitis media is often present; most patients below age 30; cold agglutinins are neither sensitive nor specific for infection, and appear only during second week.
Synonyms	Mycoplasma pneumoniae, Primary atypical pneumonia. ICD9: 041.81,483.0 ICD10: B96.0

Clinical

Acute infection:
Onset is insidious and gradual, and characterized by fever, malaise, a dry cough, headache, 'scratchy' throat and chest wall (ie, muscular) pain. [1]
• Pleuritic pain, productive cough and rigors are unusual and should suggest infection by other bacterial species.
• A lymphocytic pleural effusion may be present. [2]
• The pharynx and tympanic membranes are often erythematous, without adenopathy; and the lungs are usually normal to auscultation.
• A macular, urticarial or vesicular rash is occasionally present; and erythema multiforme / mucositis [3] (including Stevens-Johnson syndrome) is reported in some cases. [4-11]

Atypical manifestations: [12]
Atypical and severe disease is encountered among older adults.
• Rare instances of acute hepatitis [13] [14], glomerulonephritis [15] [16], rhabdomyolysis [17-19], septic shock [20], endocarditis [21], ARDS [22], multi-organ failure [23], acute respiratory distress syndrome [24], pericarditis and empyema have been reported. [25]
• Neurological findings may include encephalitis [26-36], brainstem / striatal encephalopathy [37], post-encephalitic seizures [38], aseptic meningitis [39-41], acute transverse myelitis [42] [43], stroke [44] [45], or polyradiculopathy. [46-48]
• Obsessive-compulsive disorder has been ascribed to *Mycoplasma pneumoniae* infection [49]
• Extrapulmonary manifestations also include hematologic (including autoimmune hemolytic anemia [50] [51]), pancytopenia [52], acute thrombocytosis [53]; renal; gastrointestinal; genitourinary [54]; hepatic [55] [56]; osteoarticular [57]; cutaneous (rash, angioedema with eosinophilia [58]), papular purpuric gloves and socks syndrome (PPGSS) [59], leukocytoclastic vasculitis [60], urticarial vasculitis suggestive of adult Still's disease [61], toxic epidermal necrolysis [62], mucositis [63]; possible splenic infarction [64]; and ocular involvement (including vasculitis [65] and optic neuritis [66] / papillitis). [67]).
• *Mycoplasma pneumoniae* infection is implicated in the etiology of Guillain-Barre syndrome [68-70], recurrent tonsillitis [71] and asthma. [72-78]
• *Mycoplasma pneumoniae* infection is independently associated with risk of subsequent development of ischemic stroke [79] and may play a role in the development of atherosclerosis. [80]

This disease is endemic or potentially endemic to all countries.

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Mycoplasma pneumoniae infection in Brazil

Prevalence surveys:

10% of children below age 5 years with acute respiratory infection, presenting to an emergency service in northeastern Brazil. (2011 publication) [81]

3.1% of patients with respiratory infection (1991 publication) [82]

12,75% of community-acquired pneumonia and parapneumonic pleural effusion in children and adolescents (2000 to 2008) [83]

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Myiasis

Agent	PARASITE - Insecta (Diptera) larvae
Reservoir	Mammal
Vector	Biting arthropod
Vehicle	Fly eggs deposited by biting arthropod
Incubation Period	1w - 3m
Diagnostic Tests	Identification of extracted maggot.
Typical Adult Therapy	Removal of maggot
Typical Pediatric Therapy	As for adult
Clinical Hints	Pruritic or painful draining nodule; fever and eosinophilia may be present; instances of brain, eye, middle ear and other deep infestations are described.
Synonyms	Calliphora, Chrysomya, Chrysomyia, Cochliomyia, Cordylobia, Cuterebrosis, Dermatobia, Eristalis, Furuncular myiasis, Gasterophilus, Hypoderma, Lucilia, Lund's fly, Maggot infestation, Megaselia, Musca, Muscina, Oedemagena, Oestrus larvae, Ophthalmomyiasis, Psychoda, Rectal myiasis, Sarcophaga, Screw worm, Telmatoscopus, Urinary myiasis, Vaginal myiasis, Wohlfarthia. ICD9: 134.0 ICD10: B87

Clinical

Myiasis may be primary (active invasion) or secondary (colonization of wound). [1]
• Primary furuncular myiasis is usually characterized by one or more erythematous, painful "pustules" having a central perforation. [2]
• Eosinophilia may be present. [3]
• Other clinical forms include ophthalmomyiasis (migrating larvae in the conjunctival sac), pharyngeal, nasal [4] , urinary, vaginal, tracheopulmonary and rectal infestation.
• Larvae may rarely invade the paranasal sinuses and even cause eosinophilic meningitis. [5]
• Penile myiasis may mimic a sexually transmitted disease [6]

This disease is endemic or potentially endemic to all countries.

Myiasis in Brazil

Local pathogens include *Dermatobia hominis* [7] and *Cochliomyia macellaria* [8] , and *Cochliomyia hominivorax*. [9-12]

Myiasis due to the following has been reported in Goias State (in order of frequency): *Cochliomyia hominivorax* [13] , *Sarcodexia lambens*, *Dermatobia hominis*, *Chrysomya albiceps*, *Chrysomya megacephala*, *Lucilia cuprina*, and *Eristalis tenax*. [14]

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Necrotizing skin/soft tissue infx.

Agent	BACTERIUM. Streptococcus pyogenes, Clostridium perfringens, mixed anaerobic and/or gram-negative bacilli
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Clinical features. Smear and culture (including anaerobic culture) of exudate.
Typical Adult Therapy	Debridement and parenteral antibiotics directed by smear and culture results. Hyperbaric oxygen in more severe infections
Typical Pediatric Therapy	As for adult
Clinical Hints	At least 7 syndromes in this category: most characterized by local pain and swelling, skin discoloration or edema, gas formation, foul odor and variable degrees of systemic toxicity.
Synonyms	Anaerobic cellulitis, Chancrum oris, Clostridial cellulitis, Clostridium novyi, Fasciitis, Fournier's gangrene, Gangrenous cellulitis, Gangrenous stomatitis, Invasive group A strep. Infections, Meleney's synergistic gangrene, Necrotizing fasciitis, Noma, Streptococcal fasciitis, Synergistic necrotizing cellulitis. ICD9: 686.8,528.1 ICD10: M72.6,A69.0

Clinical

Infections often begin in areas of minor trauma or loss of dermal integrity (as in varicella), and may spread within hours to involve large areas and endanger life. [1-5]

Clinical forms of necrotizing skin and soft tissue infection (in alphabetical order):

Clostridial cellulitis usually follows local trauma or surgery, and has a gradual onset following an incubation period of 3 or more days.
• There is minimal pain and discoloration, with moderate swelling.
• A thin, occasionally foul and dark colored exudate is noted and copious gas is present.
• Systemic signs are minimal.

Clostridial myonecrosis is discussed elsewhere in this module • but is distinguishable from the above syndromes by its severity, prominent systemic toxicity and the presence of overt muscle involvement.

Fournier's gangrene is a form of necrotizing fasciitis which involves the scrotum and penis. [6-8]
• Most patients are over the age of 50 • diabetic, alcoholic or suffering from rectal cancer.
• The lesion is markedly destructive and mutilating, and typically due to a mixed flora of anaerobic and facultative or aerobic gram negative bacilli.
• Fournier's gangrene may occasionally complicate varicella [9]
• The case fatality rate for Fournier's gangrene is over 20% [10]

Gangrenous stomatitis (chancrum oris, Noma) is a mutilating condition of the skin and soft tissues of the face which affects primarily immune-suppressed [11-13] and malnourished children. [14-19]
• Most patients are under the age of 6 years.
• The disease usually begins as a painful red or purple intraoral lesion, which rapidly spreads to destroy surrounding bone and soft tissues of the mouth and face.
• The case-fatality rate is 70% to 90%.

Infected vascular gangrene is a complication of peripheral vascular insufficiency and has a gradual onset beginning 5 or more days after the initiating event.
• Onset is gradual, and pain may vary from absent to prominent.
• The area is discolored and painful, and associated with foul malodorous gas and involvement of underlying muscle.
• Systemic signs are minimal.

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Meleney's gangrene (progressive bacterial synergistic gangrene) usually involves sites of fistulae, retention sutures or draining empyema. [20] [21]
• The infection begins 1 to 2 weeks following surgery, and is characterized by erythema and moderate swelling, with minimal crepitus.

Necrotizing fasciitis is typically associated with diabetes mellitus or recent abdominal surgery. [22] [23]
• Following an incubation period of 1 to 4 days, the patient becomes increasingly ill, with moderate local pain and gas formation, and a foul seropurulent discharge.
• Pain may be severe, and areas of erythema and necrosis are evident.
• Relatively high mortality rates are associated with necrotizing fasciitis caused by *Aeromonas* or *Vibrio* species. [24]

Non-clostridial anaerobic cellulitis is usually associated with diabetes mellitus or a preexisting local infection.
• Onset may be gradual or rapid, with moderate swelling, dark pus, minimal discoloration and copious foul-smelling gas.
• Pain is minimal, and the patient is moderately ill.

Synergistic necrotizing cellulitis is associated with diabetes, renal disease, obesity or preexisting perirectal infection.
• The incubation period varies from 3 to 14 days, and onset is acute.
• Swelling may be marked, and associated with intense local pain, foul 'dishwater' pus and small amounts of gas.
• Moderate muscle involvement and marked systemic disease are present.

This disease is endemic or potentially endemic to all countries.

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Neutropenic typhlitis

Agent	BACTERIUM. Clostridium septicum (occasionally Clostridium tertium, Clostridium sporogenes, Clostridium sordellii or Clostridium tertium)
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Unknown
Diagnostic Tests	Typical findings in the setting of neutropenia. Ultrasonography may be helpful.
Typical Adult Therapy	Broad spectrum antimicrobial coverage, which should include clostridia and Pseudomonas aeruginosa. Role of surgery is controversial
Typical Pediatric Therapy	Broad spectrum antimicrobial coverage, which should include clostridia and Pseudomonas aeruginosa. Role of surgery is controversial
Clinical Hints	Fever, abdominal pain, diarrhea (occasionally bloody) and right lower quadrant signs in a neutropenic (leukemic, etc) patient; may spread hematogenously to extremities; case-fatality rate 50% to 75%.
Synonyms	Neutropenic enterocolitis. ICD9: 540.0 ICD10: A04.8

Clinical

Neutropenic typhlitis is clinically similar to acute appendicitis, but limited to patients with severe neutropenia. [1-3]

This disease is endemic or potentially endemic to all countries.

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New World phleboviruses

Agent	VIRUS - RNA. Bunyaviridae, Orthobunyavirus: Alenquer, Arboledas, Bujaru, Cacao, Candiru, Chagres and Punta Toro viruses
Reservoir	Fly (sandfly = Lutzomyia trapidoi, Lu. ylephiletor) ? Rodent
Vector	Fly (sandfly = Lutzomyia)
Vehicle	None
Incubation Period	3d - 4d (range 2d - 9d)
Diagnostic Tests	Viral culture (blood, CSF, biopsy). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Myalgia, eye pain, arthralgia, vomiting, facial flush and leukopenia; fatality and sequelae are not reported.
Synonyms	Alanquer, Arboledas, Bujaru, Cacao, Candiru, Chagres, Punta Toro. ICD9: 078.89 ICD10: A93.8

Clinical

Infections by New World phleboviruses are characterized by myalgia, eye pain, arthralgia, vomiting, facial flush and leukopenia.
• Deaths and sequelae are not reported.

This disease is endemic or potentially endemic to 3 countries.

New World phleboviruses in Brazil

The incidence of infection is unknown.

Sporadic cases or seroprevalence toward Alenquer [1] , Bujaru and Candiru viruses have been documented.

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Nocardiosis

Agent	BACTERIUM. Actinomycetes, Nocardia spp. An aerobic gram positive bacillus (acid-fast using special technique)
Reservoir	Soil
Vector	None
Vehicle	Air Dust Wound Contact
Incubation Period	? days to weeks
Diagnostic Tests	Culture and gram stain of exudates, sputa, tissue specimens. Advise laboratory when Nocardia suspected.
Typical Adult Therapy	Sulfamethoxazole/trimethoprim - dosage and duration of therapy appropriate to clinical severity
Typical Pediatric Therapy	As for adult
Clinical Hints	Pneumonia, lung abscess, brain abscess, or other chronic suppurative infection; often in the setting of immune suppression.
Synonyms	Nocardia, Nocardiose. ICD9: 039 ICD10: A43

Clinical

Nocardiosis may present as an acute or chronic suppurative infection with a tendency to remission and exacerbation. [1]
- Infections are most common among immunocompromised patients. [2] [3]
- The most common presentation is pneumonia.
- Brain abscesses account for 33% of cases.
- Infection of virtually any other organ may occur. [4-7]

Nocardiosis may mimic tuberculosis, particularly in the setting of HIV infection. [8]
- Nodular lymphadenitis, seen with *Nocardia brasiliensis* infection, may mimic nocardiosis. [9]

The ecology and phenotypic characteristics of *Nocardia* species [10] are discussed in the Microbiology module.

This disease is endemic or potentially endemic to all countries.

Nocardiosis in Brazil

Sporadic infections are reported [11] , including disseminated infection in transplantation recipients [12] and patients with AIDS. [13]

22 cases of *Nocardia* infection were diagnosed in Porto Alegre during 1977 to 1998. [14]

Prevalence surveys:

0.05% of solid organ transplant recipients (a single case, 2001 to 2006) [15]

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Oesophagostomiasis

Agent	PARASITE - Nematoda. Phasmidea: Oesophagostomum bifurcum (O. apiostomum, O. stephanostomum)
Reservoir	Non-human primate Soil
Vector	None
Vehicle	Feces Water Soil
Incubation Period	2w - 2m
Diagnostic Tests	Demonstration of parasite in tissue.
Typical Adult Therapy	Albendazole (400 mg as single dose), or Pyrantel pamoate may be effective. Excision as necessary
Typical Pediatric Therapy	Albendazole or Pyrantel pamoate may be effective. Excision as necessary
Clinical Hints	Right lower quadrant abdominal pain and tenderness, often with intraabdominal mass or peritoneal signs.
Synonyms	Dapaong tumor, Oesophagostomum apiostomum, Oesophagostomum bifurcum, Oesophagostomum stephanostomum, Ternidens. ICD9: 127.7 ICD10: B81.8

Clinical

Oesophagostomiasis is contracted through ingestion of soil-contaminated food or water, and is characterized by development of an inflammatory mass in the ileum or colon.
• Approximately 15% of patients present with multinodular disease, characterized by abdominal pain, fever, vomiting and mucous diarrhea. [1] [2]
• 85% of patients develop an intestinal mass adherent to the overlying abdominal wall (helminthoma = Dapaong tumor), often associated with pain and fever. [3] [4]

This disease is endemic or potentially endemic to 35 countries.

Oesophagostomiasis in Brazil

Sporadic cases of *Oesophagostomum bifurcum* infection have been reported. [5]

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Onchocerciasis

Agent	PARASITE - Nematoda. Phasmidea, Filariae: Onchocerca volvulus
Reservoir	Human
Vector	Fly (black fly = Simulium)
Vehicle	None
Incubation Period	12m - 18m
Diagnostic Tests	Identification of microfilariae in skin snips or on ophthalmoscopy. Nucleic acid amplification.
Typical Adult Therapy	Excision of nodules. Ivermectin 150ug/kg PO once. Repeat every 6 months Doxycycline 100 mg PO daily for 6 weeks prior to Ivermectin improves cure rate If eye involved, administer corticosteroid for several days prior to ivermectin.
Typical Pediatric Therapy	Excision of nodules. Ivermectin 150ug/kg PO once. Repeat every 6 months Age > 8 years: Doxycycline, as for adult
Clinical Hints	Macular, papular or dyschromic skin lesions; pruritus; lymphadenopathy; keratitis or uveitis; eosinophilia; firm nodules over bony prominences; adult worms may survive for 15 years in the human host.
Synonyms	Aswad, Craw-craw, Dipetalonema arbuta, Dipetalonema sprenti, Erysipelas de la Costa, Flussblindheit, Jur blindness, Lichenified onchodermatitis, Nakalanga syndrome, Onchocerca cervicalis, Onchocerca dewittei, Onchocerca guttarosa, Onchocerca jakutensis, Onchocerca lupi, Onchocerca retuculata, Onchocerca volvulus, Onchozerkose, River blindness, Robles' disease, Sowda. ICD9: 125.3 ICD10: B73

Clinical

WHO Case definition for surveillance:
• In an endemic area, a person with fibrous nodules in subcutaneous tissues.
Laboratory criteria for confirmation • one or more of the following
• Presence of microfilariae in skin snips taken from the iliac crest
• Presence of adult worms in excised nodules
• Presence of typical ocular manifestations, such as slit-lamp observations of microfilariae in the cornea, the anterior chamber, or the vitreous body
Case classification
Suspected: A case that meets the clinical case definition.
Probable: Not applicable.
Confirmed: A suspected case that is laboratory-confirmed.

W.H.O. recognizes five forms of skin disease for purposes of survey and control:
• acute papular onchodermatitis
• chronic papular onchodermatitis
• lichenified onchodermatitis
• atrophy
• depigmentation

The distribution of dermal nodules is related to body regions which are exposed to vector bites (ie, local clothing practices). [1]

The microfilariae of *Onchocerca* migrate throughout the body and give rise to visual impairment (punctate keratitis) [2] , rashes, intense pruritis and depigmentation of the skin [3] ; lymphadenitis; "hanging groin" and elephantiasis of the genitals. [4]

• Rare instance of eosinophilic meningitis have been reported. [5]
• Some studies have suggested a possible association between onchocerciasis "nodding syndrome" and epilepsy; however data are inconclusive. [6-21]

Onchocerciasis has been implicated in the etiology of Nakalanga syndrome (hyposexual dwarfism) in Sudan; and sowda (a form of endemic filarial limb dermatosis with adenopathy) on the Arabian Peninsula. [22]
• It has been suggested that sowda may be caused by a zoonotic species rather than *Onchocerca volvulus*.

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Dermal onchocerciasis may mimic dracunculiasis. [23]

There is extensive evidence that endosymbiont bacteria (*Wolbachia* spp.) are necessary for the development of filarial larvae, and fertility of adult parasites. [24-27]
• Doxycycline has proven effective in therapy, presumably through inhibition of *Wolbachia* spp. [28-30]

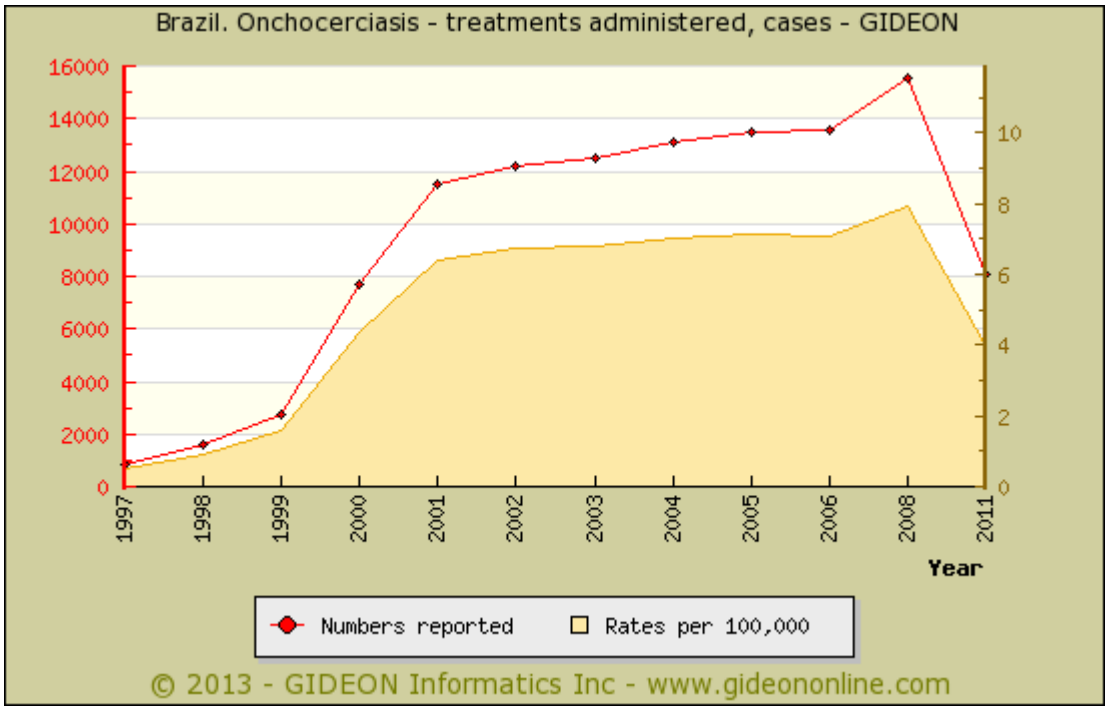
This disease is endemic or potentially endemic to 36 countries.

Onchocerciasis in Brazil

Time and Place:
Onchocerciasis is endemic to Amazonas and Roraima. [31]
- New foci were recently described in Minacu. [32]
- As of 2008, there was a single endemic focus - the Amazonas-Roraima region. [33-35]
- 7,200 were at risk in 147 endemic communities as of 1995; 8,004 in 17 endemic communities as of 2002; 12,521 as of 2011. [36] [37]
- Prevalence rates as high as 97.9% are found among the Yanomami Indians; however, blindness has not been described.

5,500 were infested and 100 blind in 1985.

Prevalence surveys:
31.2% in Amazonas in 1995; 2.2% in 2007 (microfilaria in anterior chamber) [38]
55% of individuals of the Yanomami tribe, Roraima State (2002) [39]
8.6% of blackflies in Balawau and 4% in Toototobi before institution of ivermectin treatment, 0.3% in Balawau and 0.1% in Toototobi after treatment (1998 to 1999) [40]



Graph: Brazil. Onchocerciasis - treatments administered, cases

Notes:
1. Mass treatment with ivermectin was initiated in 1995.

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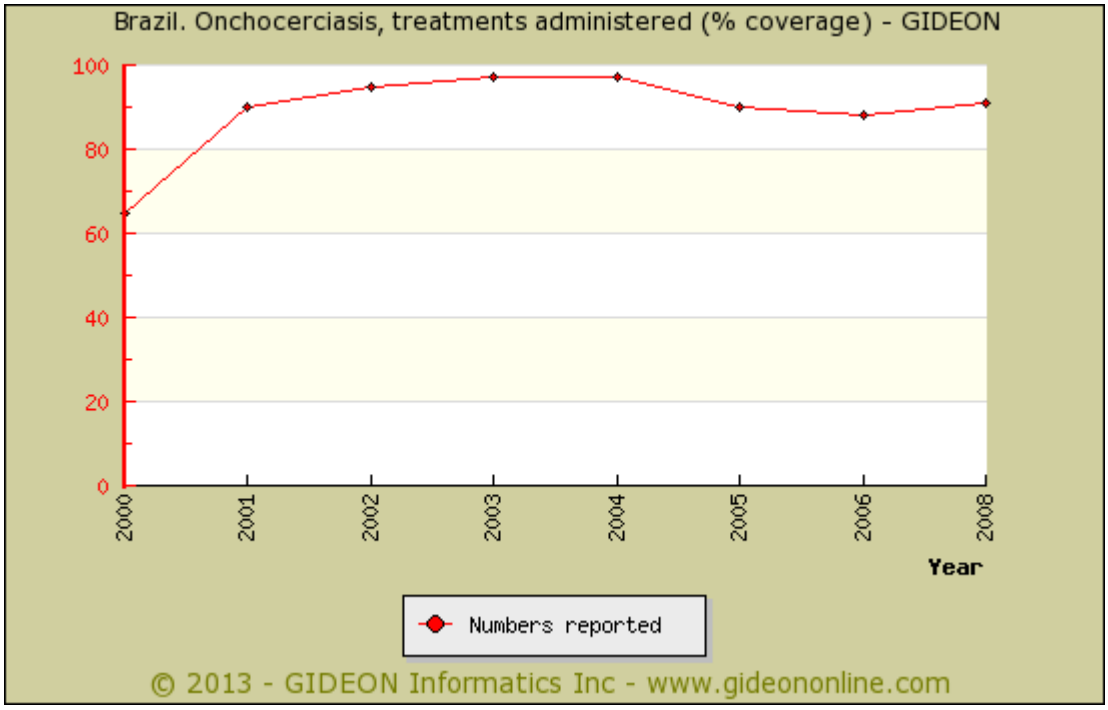

Graph: Brazil. Onchocerciasis, treatments administered (% coverage)

Notes:

1. Additional references: [41-43]

Vectors:

The principal vector is *Simulium exiguum*.

- *S. auripellitum*, *S. guianense*, *S. minusculum* and *S. nigrimanum* are considered potential vectors in central Brazil.

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Orbital and eye infections

Agent	BACTERIUM OR FUNGUS. Streptococcus pyogenes, oral anaerobes, Aspergillus spp., facultative gram-negative bacilli, et al
Reservoir	Endogenous Introduced flora (trauma, surgery)
Vector	None
Vehicle	Trauma Surgery Contiguous (sinusitis) Hematogenous
Incubation Period	Variable
Diagnostic Tests	Imaging techniques (CT or MRI). Culture of aspirates or surgical material.
Typical Adult Therapy	Local and systemic antimicrobial agents appropriate for species and severity
Typical Pediatric Therapy	As for adult
Clinical Hints	Proptosis, chemosis, extraocular palsy, or hypopyon associated with sinusitis, bacteremia, eye trauma or surgery. Involves the eye (endophthalmitis); periosteum (periorbital infection); orbit (orbital cellulitis); orbit + eye (panophthalmitis).
Synonyms	Bacterial keratitis, Ceratite, Cheratite, Endophthalmitis, Eye infection, Keratite, Keratitis, Orbital infection, Panopthalmitis, Queratitis. ICD9: 360.0 ICD10: H05.0

Clinical

Endophthalmitis involves the ocular cavity and adjacent structures. [1] [2]
• Infection may occur in the setting of endocarditis or other bacteremic infections, or follow surgery or penetrating trauma.
• The onset of fungal endophthalmitis is more gradual than infection due to bacteria.
• Several species of parasites (ie, *Toxoplasma*, *Toxocara*, *Onchocerca*, etc) and viruses (CMV, Herpes simplex, measles) may also infect a variety of orbital structures, and are discussed elsewhere in this module.

Panophthalmitis involves all ocular tissue layers, including the episclera. [3] [4]
• Pain on eye movement is prominent.

Orbital cellulitis is an acute infection of the orbital contents.
• Infection can easily spread to the cavernous sinuses.
• The most common sources for infection are the paranasal sinuses (most commonly ethmoid in children).
• Fever, lid edema, orbital pain, proptosis and limited motion of the globe are important symptoms.

Keratitis can be caused by viruses (Herpes simplex, zoster, smallpox), bacteria, fungi, protozoa (*Acanthamoeba*) or helminths (*Onchocerca volvulus*)
• Microbial keratitis complicating orthokeratology is mainly caused by *P. aeruginosa* or *Acanthamoeba* [5]

This disease is endemic or potentially endemic to all countries.

Orbital and eye infections in Brazil

Notable outbreaks:
2000 - An outbreak (10 cases) of *Mycobacterium chelonae* keratitis was associated with laser in situ keratomileusis (LASIK). [6]
2003 - An outbreak (5 cases) of *Mycobacterium immunogenum* keratitis in Sao Paulo was associated with myopia surgery. [7]

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Orf

Agent	VIRUS - DNA. Poxviridae, Parapoxvirus: Orf virus
Reservoir	Sheep Goat Reindeer Musk ox
Vector	None
Vehicle	Contact Infected secretions Fomite Cat-scratch (rare)
Incubation Period	3d - 6d (range 2d - 7d)
Diagnostic Tests	Viral culture (skin lesion or exudate). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Skin pustule or ulcer following contact with sheep or goats; most lesions limited to finger or hand; heals without scarring within 6 weeks.
Synonyms	Contagious pustular dermatitis, Ecthyma contagiosum, Ovine pustular dermatitis. ICD9: 078.89 ICD10: B08.0

Clinical

Human infection is milder than that of sheep, and usually limited to indolent vesicles and pustules on the hands. [1] [2]
- Pustules may attain a size of 1 to 2 cm, and are often associated with low-grade fever and regional lymphadenitis.
- Lesions heal over a period of 2 to 6 weeks, without scarring.
- Bullous lesions [3] , secondary bacterial infection, disseminated orf, Guillain-Barre syndrome [4] and erythema multiforme [5-10] have been described in some cases.

This disease is endemic or potentially endemic to all countries.

Orf in Brazil

Outbreaks of presumed Orf have been reported among sheep and goats in several areas of Brazil. [11]

Notable outbreaks:

1989 (publication year) - An outbreak was reported among goats. [12]

2009 (publication year) - An outbreak of orf (ovine contagious ecthyma) was reported among sheep in Midwest Brazil. [13]

2013 (publication year) - An outbreak of vesicular disease due to Orf virus and a parapoxvirus was reported among dairy cows in midwestern Brazil. [14]

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Ornithosis

Agent	BACTERIUM. Chlamydiaceae, Chlamydiae, Chlamydophila [Chlamydia] psittaci
Reservoir	Parakeet Parrot Pigeon Turkey Duck Cat Sheep Goat Cattle ? Dog
Vector	None
Vehicle	Bird droppings Dust Air Aerosol from cat [rare]
Incubation Period	7d - 14d (range 4d - 28d)
Diagnostic Tests	Serology. Culture (available in special laboratories) rarely indicated.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 10d. Alternatives: Erythromycin 500 mg PO QID X 10d. Azithromycin 1 g, then 0.5 g daily. Clarithromycin 0.5 g BID
Typical Pediatric Therapy	Age < 8 years: Erythromycin 10 mg/kg QID X 10d Age >=8 years: Doxycycline 100 mg PO BID X 10d.
Clinical Hints	Headache, myalgia and pneumonia, often with relative bradycardia, hepatomegaly or splenomegaly; onset 1 to 4 weeks following contact with pigeons, psittacine birds or domestic fowl; case-fatality rate without treatment = 20%.
Synonyms	Chlamydophila abortus, Chlamydophila psittaci, Ornitose, Papegojsjuka, Parrot fever, Psitacosis, Psittacosis, Psittakose. ICD9: 073 ICD10: A70

Clinical

Onset may be insidious or abrupt, and the illness may subclinical, or take the form of nonspecific fever and malaise, pharyngitis, hepatosplenomegaly, and adenopathy. [1]
• Bradycardia and splenomegaly may suggest typhoid at this stage.

A more common presentation consists of atypical pneumonia, with nonproductive cough, fever, headache and pulmonary infiltrates. [2] [3]
• Additional findings may include photophobia, tinnitus, ataxia, deafness, anorexia, vomiting, abdominal pain [4] , diarrhea, constipation, hemoptysis, epistaxis, arthralgia, and rash (Horder's spots) reminiscent of the rose spots of typhoid. [5]
• Fever, pharyngitis, rales and hepatomegaly are noted in over 50% of cases.

Complications include pericarditis, myocarditis, and "culture-negative" endocarditis, ARDS [6] , overt hepatitis, hemolytic anemia, DIC, reactive arthritis, cranial nerve palsy, cerebellar dysfunction, transverse myelitis, meningitis, encephalitis and seizures, thrombophlebitis, pancreatitis and thyroiditis.
• Subclinical infection by *Chlamydophila psittaci* has been implicated in the etiology of chronic polyarthritis. [7]
• Rare instances of abortion have been reported.

Chlamydophila abortus, a related species which affects goats, cattle and sheep, had been associated with rare instances of abortion, stillbirth and even maternal death in humans.

This disease is endemic or potentially endemic to all countries.

Ornithosis in Brazil

Chlamydophila psittaci has been identified in Blue-fronted Amazon parrots (*Amazona aestiva* and Hyacinth macaws (*Anodorhynchus hyacinthinus*) in the Pantanal of Mato Grosso do Sul. [8]

Prevalence surveys:

16.8% of urban pigeons (*Columba livia*, 2010 publication) [9]

Seroprevalence surveys:

4.7% of zoo workers and veterinary practitioners (2009 publication) [10]

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Notable outbreaks:

2007 - An outbreak (27 cases, including 15 veterinarians) in Rio Grande do Sul was associated with a shipment of illegally-smuggled wild birds. [11]

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Oropouche

Agent	VIRUS - RNA. Bunyaviridae, Orthobunyavirus, Simbu group virus: Oropouche virus
Reservoir	Unknown
Vector	Midge (Culicoides paraensis) Mosquito (Culex quinquefasciatus, Aedes serratus, Coquillettidia venezuelensis)
Vehicle	Unknown
Incubation Period	4d - 8d (range 3d - 12d)
Diagnostic Tests	Viral culture (blood, CSF). Serology. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, myalgia, arthralgia, leukopenia and gastrointestinal symptoms; occasionally meningitis; disease lasts 5 to 7 days, however convalescence is prolonged; no deaths have been reported.
Synonyms	Iquitos virus. ICD9: 066.3 ICD10: A93.0

Clinical

Oropouche is characterized by abrupt onset of fever, chills, myalgia, arthralgia, headache, photophobia and leucopenia.
• A rash is present in 5% of cases, and meningitis in 4%. [1]
• No fatal infections have been reported, however relapses and prolonged convalescence are common.

Clinical features associated with infection by a related agent, Iquitos virus, include fever, headache, eye pain, body pain, arthralgias, diarrhea, and chills. [2]

This disease is endemic or potentially endemic to 5 countries.

Oropouche in Brazil

Time and Place:
Oropouche virus was first identified in Brazil in 1960, in a sloth (*Bradypus tridactylus*) and a pool of mosquitoes (*Ochlerotatus serratus*).
- Oropouche is endemic to the Amazon Basin, where it is the second most common arboviral disease (after dengue).
- Epidemics occur during the rainy season with attack rates as high as 60% in rural areas.
- Sporadic single cases are rarely reported. [3]
- Seropositivity was identified among sentinel primates in Mato Grosso do Sul during 2010. [4]

Oropouche fever is the second most common arboviral infection (after dengue) in Brazil. [5]
- Over 36,000 persons were infected during 1961 to 1978.
- An estimated 500,000 had occurred in Brazil as of 2009. [6]

Prevalence surveys:
2.7% of meningoencephalitis cases in the Amazon and Central-Plateau regions (2005 to 2010) [7]

Seroprevalence surveys:
2.9% of inhabitants of Rio Branco, Acre (1999) [8]

The virus was isolated from a monkey (*Callithrix* sp.) in Minas Gerais in 2000.

The principal vector is *Culicoides paraensis*. [9]
- Oropouche virus has also been identified in *Coquillettidia venezuelensis* [10]

Notable outbreaks:

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1961 - An outbreak (11,000 cases, approximate) was reported in Belem, Para State.
1975 - An outbreak was reported in urban centers of Para. [11-13]
1978 to 1980 - An outbreak (130,000 cases in Para and 97,000 in Amazonas) was reported. [14] [15]
1988 - Outbreaks (128 cases) were reported in Maranhao and Goias. [16]
1994 - An outbreak (5,000 cases estimated) was reported in Serra Pelada, Para. [17]
2003 - An outbreak (31 cases) was reported in Para State.
2004 - An outbreak (61 cases) was reported in Para State. [18]
2006 - An outbreak (113 cases) was reported in the Bragantina region, northern Brazil. [19]
2007 to 2008 - An outbreak was reported in Manaus. [20]
2009 - An outbreak (657 cases) was reported in Amapa. [21] [22]

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Osteomyelitis

Agent	BACTERIUM OR FUNGUS. Staphylococcus aureus, facultative gram-negative bacilli, Candida albicans, etc
Reservoir	Endogenous Introduced flora (trauma, surgery)
Vector	None
Vehicle	Trauma Hematogenous Extension from other focus
Incubation Period	Variable
Diagnostic Tests	Radiography, including bone scan. Culture of biopsy material.
Typical Adult Therapy	Systemic antimicrobial agent(s) appropriate to known or suspected pathogen. Surgery as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Limb pain or gait disturbance; obscure fever; prior skin infection; may be hematogenous, or arise from contiguous (soft tissue, joint) infection; X-ray changes are not apparent for at least 10 days in acute infection.
Synonyms	Osteomielite, Osteomielitis, Osteomyelite, Paravertebral abscess. ICD9: 015,730.9 ICD10: M86

Clinical

Osteomyelitis is a self-defined condition characterized by infection of one or more bones.
• Signs and symptoms vary widely, and reflect associated underlying conditions, infecting species and location of the infection. [1-3]

Etiological associations:
• Animal bite: *Pasteurella multocida*
• Diabetes and vascular insufficiency: Usually mixed infection (*Staphylococcus aureus*, *Staphylococcus epidermidis*, Gram-negative bacilli, Anaerobes)
• Hematogenous: Usually single organism (*Staphylococcus aureus*, Enterobacteriaceae)
• Injecting drug user: staphylococci, Gram-negative bacilli, Candida spp.
• Secondary to contiguous infection: Often mixed infection (*Staphylococcus aureus*, Gram-negative bacilli)
• Sickle cell anemia: *Staphylococcus aureus*, *Salmonella* spp.

This disease is endemic or potentially endemic to all countries.

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Otitis media

Agent	BACTERIUM OR VIRUS. Haemophilus influenzae & Streptococcus pneumoniae in most acute cases; RSV, Parainfluenza, et al
Reservoir	Human
Vector	None
Vehicle	None
Incubation Period	Variable
Diagnostic Tests	Clinical findings. Culture of middle ear fluid if available.
Typical Adult Therapy	Antimicrobial agent directed at likely pathogens
Typical Pediatric Therapy	As for adult
Vaccine	Pneumococcal conjugate
Clinical Hints	Acute bacterial otitis media often represents the final stage in a complex of anatomic, allergic or viral disorders of the upper airways; recurrent or resistant infections may require surgical intervention.
Synonyms	Otitis media aguda. ICD9: 382.0 ICD10: H65,H66

Clinical

Signs and symptoms of otitis media consist of local pain and tenderness, with or without fever and signs of sepsis. [1] [2]

This disease is endemic or potentially endemic to all countries.

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Paracoccidioidomycosis

Agent	FUNGUS. Ascomycota, Euascomycetes, Onygenales: Paracoccidioides braziliensis A dimorphic fungus
Reservoir	Soil Dust Armadillo
Vector	None
Vehicle	Air
Incubation Period	1m - 9y
Diagnostic Tests	Fungal culture. Serologic tests are available
Typical Adult Therapy	Sulfamethoxazole/trimethoprim 800/160 mg PO BID X 1 month, then 400/80 mg per day PLUS Itraconazole 200 mg PO daily X 6 months. OR Ketoconazole 400 mg/d X 12m. OR Amphotericin B 0.3 mg/kg/d to total dose 2.0g.
Typical Pediatric Therapy	Sulfamethoxazole/trimethoprim 30/6 mg/kg PO BID X 1 month, then 5/1 mg/kg per day PLUS Itraconazole 3 mg/kg PO daily X 6 months. OR Ketoconazole 5 mg/kg/d X 12m. OR Amphotericin B 0.3 mg/kg/d to total dose 30 mg/kg.
Clinical Hints	Acute or chronic pulmonary disease; verrucous or ulcerated mucocutaneous lesions are present in most cases, often with regional lymphadenopathy; the face, mouth and nose are principal sites of infection.
Synonyms	Brazilian blastomycosis, Lutz-Splendore-Almeida's disease, Paracoccidioides braziliensis, Paracoccidioides lutzii, South American blastomycosis, Tropical granulomatous blastomycosis. ICD9: 116.1 ICD10: B41

Clinical

Paracoccidioidomycosis is characterized by a progressive, subacute to chronic granulmatous process which begins in the lungs [1]
- Infection spreads to the oral or nasal mucosa (with prominent regional lymphadenopathy) [2]
- The gastrointestinal tract [3] [4] skin or viscera, and central nervous system are occasionally involved [5-7]
- The disease may be limited to oral lesions without any evidence of pulmonary disease. [8] [9]
- Odynophagia and dysphagia are common in patients with pulmonary infection. [10]

Two main clinical forms are recognized [11] :
- acute or subacute infection which affects young persons of both sexes and involves primarily the reticuloendothelial system
- chronic multifocal or unifocal infection of the respiratory tract [12] [13] , skin and mucous membranes is most commonly identified in adult males.

Rare complications of paracoccidioidomycosis have included spontaneous pneumothorax [14] and fatal acute respiratory distress syndrome. [15]

Extrapulmonary disease is found in over 70% of cases and may involve skin, mucous membranes, lymph nodes, muscles [16] , joints [17] [18] , tendons [19] , bones [20] , adrenals [21] , bone marrow [22] [23] , abdominal organs [24] or central nervous system (10% to 13%). [25-27]
- Unlike the presentation of leishmaniasis, oropharyngeal lesions are painful and bleed easily. [28]
- There is often a red infiltrated hard base with exuberant or papillomatous hemorrhagic granulomatous lesions.
- Edema of the lips and involvement of the gingivae is common. [29] [30]
- Laryngeal [31] and osseous [32] lesions may be misdiagnosed as malignancy.
- Bacterial superinfection of ulcerative lesions is more common that with oral ulcers due to mucocutaneous leishmaniasis
- Infection of the uterine cervix may be incidentally diagnosed by pap smear [33]
- Concomitant tuberculosis is often present.
- Skin lesions may mimic those of tuberculoid leprosy [34] and roentgenographic findings may suggest a diagnosis of sarcoidosis. [35]
- Rare cases of protein-losing enteropathy [36] , hypercalcemia [37] and adrenal insufficiency have been reported. [38]

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This disease is endemic or potentially endemic to 11 countries.

Paracoccidioidomycosis in Brazil

Brazil accounts for 80% of reported cases of paracoccidioidomycosis in the world.

Time and Place:
Paracoccidioidomycosis is most common in Mato Grosso [39] , Minas Gerais [40] , Rio Grand do Sul, Rio de Janeiro and Sao Paulo [41] [42] ; and rare in Manaus and Amazonas.
- The rate in Sao Paulo was 1 per 100,000 in 1970; 1.6 to 3.7 per 100,000 during 1960 to 1999 (Ribeirao Preto),

3,181 fatal cases were reported during 1980 to 1995 - 1.45 per 1 million population per year. [43]
- During 1996 to 2006, an estimated 1,853 patients died of paracoccidioidomycosis in Brazil. [44]
- 207 deaths were ascribed to the disease in 1995.
- 551 fatal cases were registered in Parana state during 1980 to 1998 (3.48 per million per year) [45]
- Epidemiological features in a series of 1,000 cases treated in Ribeirao Preto during 1960 to 1999 - see reference [46]

Report of *Paracoccidioides lutzii* infection (2 cases) in the Amazon region (2012 publication) [47]

Exported cases:
- 42 importations of paracoccidioidomycosis into non-endemic countries were reviewed in 1985 - including 17 from Brazil. [48]
- In 1959, a case in Switzerland was confirmed in a man who had returned from Brazil. [49]
- A case report published in 2002 described infection of a German national in Brazil. [50]
- Additional countries reporting importations from Brazil have included Morocco, Japan, Austria, Bulgaria, the United Kingdom, Italy, Portugal, Spain, Lebanon, and the United States.

Prevalence surveys:

1.4% of HIV-positive patients in Ribeirao Preto, Sao Paulo (2009 publication) [51]

12.2% of HIV-positive patients in a region endemic for paracoccidioidomycosis (2011 publication) [52]

Systemic fungal diseases were found in 4.6% of AIDS patients in Cuiaba, Mato Grosso - *Cryptococcus neoformans* in 50% of these, *Cryptococcus neoformans var. gattii* 1.6%, *Cryptococcus* spp in 6.6%, *Histoplasma capsulatum* 38.3% and *Paracoccidioides brasiliensis* 3.3% (2005 to 2008) [53]

41.7% of infectious granulomatous laryngitis cases (2007 publication) [54]

44.1% of *Cebus* sp. And 60% of *A. caraya* (New World monkeys) in Parana (2007 publication) [55]

Seroprevalence surveys:

5% of Xacriaba Indians (Minas Gerais)

21% of blood donors from an endemic region

27% of healthy persons in northwestern Parana (2003 publication) [56]

0.9% of blood donors in Sao Paulo (2002 publication) [57]

44% of healthy school children in the Amazon region of Mato Grosso State (skin test-positive, 1996).

53.6% of urban dogs in Uberaba, Minas Gerais (2009 publication) [58]

37% of sheep in Guarapuava, Parana State (2011 publication) [59]

55% of free-range chickens in Mato Grosso do Sul, and 16% in Parana State (2010 publication) [60]

Natural infection of nine-banded armadillos (*Dasypus novemcinctus*) from Sao Paulo has been documented. [61]

Coinfection by *Paracoccidioides* is present in 0.09% of AIDS patients (1992).
- A series of ten coinfected patients was published in 2003. [62]

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Paragonimiasis

Agent	PARASITE - Platyhelminthes, Trematoda. Paragonimus westermani, P. heterotremus, P. skrjabini, P. miyazakii, P. africanus, et al.
Reservoir	Human Dog Cat Pig Wild carnivore, Snail (Semisulcospira, Thiara, etc)
Vector	None
Vehicle	Fresh-water crab (at least 8 species) Crayfish (Cambaroides)
Incubation Period	6w - 6m
Diagnostic Tests	Identification of ova in sputum or stool. Serologic and skin tests are available.
Typical Adult Therapy	Praziquantel 25 mg/kg TID X 2d. OR Bithionol 40 mg/kg every other day X 10 doses. OR Triclabendazole 10 mg/kg/d X 2
Typical Pediatric Therapy	As for adult
Clinical Hints	Pulmonary infection with bloody or "rusty" sputum, central nervous system disease (eg, meningitis or seizures) and eosinophilia; subcutaneous nodules occasionally observed; parasite may survive for decades in human host.
Synonyms	Alaria, Endemic hemoptysis, Lung fluke, Oriental lung fluke, Paragonimus, Poikilorchis, Pulmonary distomiasis. ICD9: 121.2 ICD10: B66.4

Clinical

The acute phase of parasitic invasion and migration is accompanied by diarrhea, abdominal pain, fever, urticaria, hepatosplenomegaly, wheezing, cough, pleuritic pain, and eosinophilia [1] or hypereosinophilia. [2]
• Later, pulmonary manifestations include cough, expectoration of discolored sputum, hemoptysis, and chest roentgenographic abnormalities. [3] [4]
• Pulmonary infection may mimic lung cancer, both clinically and radiologically [5] [6]
• Paragonimiasis is a common cause of persistent pleural effusion in endemic regions [7] ; such collections may suggest the diagnosis of chylothorax. [8] [9]
• Extrapulmonary infection may involve the brain (less than 1% of cases) [10-12] , epidural space [13] , subcutaneous tissues (most commonly the trunk and thighs) [14] , hepatobiliary system [15] , colon [16] or other organs. [17-20]
• Subcutaneous disease is found in 10% of patients with *P. westermani* infection, and 20% to 60% of those with *P. skrjabini* (*P. szechuanensis*) infection.
• Rare instances of disseminated infection with septic shock have been reported. [21]

This disease is endemic or potentially endemic to 47 countries.

Paragonimiasis in Brazil

The first case of paragonimiasis in Brazil was reported in 2007, in the southeastern region. (Salvador-Bahia, 2007 publication) [22]

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Parainfluenza virus infection

Agent	VIRUS - RNA. Paramyxoviridae: Respirovirus - Human Parainfluenza virus 1 and 3. Rubulavirus - Human Parainfluenza virus 2 and 4.
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	3d - 8d
Diagnostic Tests	Viral culture (respiratory secretions). Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Upper respiratory infection - often croup or laryngitis. The disease is most common during infancy; older children develop a 'cold-like' illness; the infection is complicated by pneumonia in 7% to 17% of cases.
Synonyms	Parainfluenza, Sendai. ICD9: 078.89,480.2 ICD10: J12.2

Clinical

Clinical forms of Parainfluenza virus infection include 'the common cold,' otitis media, croup (acute laryngotracheobronchitis) [1] , 'flu-like illness' [2] , bronchiolitis [3] and pneumonia.

This disease is endemic or potentially endemic to all countries.

Parainfluenza virus infection in Brazil

Prevalence surveys:

5.8% of acute respiratory infections in the age group < 5 years (1984 to 1986)
0.4% of childhood hospitalizations for lower respiratory tract infection (Sao Paulo, 1996 to 1996) [4]
1.5% of acute respiratory infections in children below age 5 (Rio Grande do Sul, 1990 to 1992) [5]
12% of outpatients with influenza-like illness (2000 to 2011) [6]
8.9% of children (age below age 5 years) in Sao Paulo with lower respiratory tract infection (2003) [7]
8.42% of children below age 5 years hospitalized with respiratory infection (2005 to 2006) [8]
6.3% of children below age 5 years with acute respiratory disease in Uberlandia (2001 to 2004) [9]
3.3% of outpatient and 5.6% of hospitalized patients with respiratory infection (Curituba, 2005 publication) [10]
30% of outpatients with influenza-like illness (2000 to 2011) [11]
20% of fatal respiratory infection in children (1985 to 2005) [12]
1.5% of influenza-like illness with proven viral etiology among adults in Sao Paulo (2001 to 2003) [13]

Notable outbreaks:

1982 (publication year) - An outbreak of Parainfluenza virus infection was reported in a hospital in Rio de Janeiro. [14]

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Parvovirus B19 infection

Agent	VIRUS - DNA. Parvoviridae, Parvovirinae: Erythrovirus B19
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	4d - 14d (range 3d - 21d)
Diagnostic Tests	Serology. Nucleic acid amplification (testing should be reserved for the rare instance of complicated infection).
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Erythema infectiosum (erythema of cheeks; lacelike or morbilliform rash on extremities); febrile polyarthralgia, or bone marrow aplasia/hypoplasia may be present.
Synonyms	Duke's disease, Erythema infantum febrile, Erythema infectiosum, Erythema simplex marginatum, Erythrovirus B19, Fifth disease, Fourth disease, Funfte Krankheit, Parascarlatina, Parvovirus 4, Parvovirus B19, Sticker's disease. ICD9: 057.0 ICD10: B08.3

Clinical

Acute infection:
Erythema infectiosum is a mild childhood illness characterized by a facial rash ("slapped cheek" appearance), and a reticulated or lacelike rash on the trunk and extremities. [1]
• Papular-purpuric gloves-and-socks syndrome [2] , or localized and generalized petechial rashes may occur in some cases. [3-8]
• Reappearance of the rash may occur for several weeks following nonspecific stimuli such as change in temperature, sunlight, and emotional stress.
• The patient is otherwise well at rash onset but often gives a history of a systemic prodrome lasting 1 to 4 days.
• In some outbreaks, pruritis has been a common clinical feature. [9]
• Rubella-like, morbilliform [10] , vesicular and purpuric [11] rashes have also been reported.
• Asymptomatic infection has been reported in approximately 20% of children and adults.
• Rare instances of hepatosplenomegaly [12] and heart failure have been reported. [13]
• Co-infection with parvovirus and other hepatitis viruses may result in fulminant hepatic failure [14]

Joint manifestations:
In some outbreaks, arthralgias and arthritis have been commonly reported. [15]
• Infection may produce a symmetrical peripheral polyarthropathy.
• The hands are most frequently affected, followed by the knees and wrists.
• Symptoms are usually self-limited but may persist for several months.
• Joint symptoms, more common in adults, are encountered in approximately 20% of cases [16] and may occur as the sole manifestation of infection.

Instances of seizure [17] , coma, encephalitic ataxia or chorea [18] [19] , meningoencephalitis [20] , autonomic or sensory neuropathy [21] , cranial nerve palsy [22] , myocarditis [23] [24] , severe endothelialitis (Degos-like syndrome) [25] , myositis [26] , and acute hepatitis have been reported. [27-30]
• Sequelae remain in 22% of patients with neurological involvement [31]
• A distinct form of Parvovirus infection known as "papular-purpuric gloves and socks syndrome" is characterized by fever and edematous rash, often associated with conjunctivitis and arthritis [32]
• Additional complications may include glomerulonephritis [33] [34] , Melkersson-Rosenthal syndrome and hemophagocytic lymphohistiocytosis [35] [36]
• Hepatic dysfunction may be present in some cases. [37]

Parvovirus B19 infection and hematological disease:
Parvovirus B19 is the primary etiologic agent causing Transient Aplastic Crisis (TAC) in patients with chronic hemolytic

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anemias (e.g., sickle cell disease, hemoglobin SC disease, hereditary spherocytosis, alpha-thalassemia, and autoimmune hemolytic anemia) and occasionally follows anemia due to blood loss. [38]
• Patients with TAC typically present with pallor, weakness, and lethargy and may report a nonspecific prodromal illness during the preceding 1 to 7 days.
• Few patients with TAC report a rash.
• In the acute phase, patients usually have a moderate to severe anemia with absence of reticulocytes; and bone marrow examination shows a hypoplastic or an aplastic erythroid series with a normal myeloid series.
• Recovery is indicated by a return of reticulocytes in the peripheral smear approximately 7 to 10 days after their disappearance.
• TAC may require transfusion and hospitalization and can be fatal if not treated promptly.

A false positive serological reaction toward Epstein-Barr virus has been reported in Parvovirus B19 infection. [39]

A Parvovirus B19-related severe chronic anemia associated with red cell aplasia has been described in transplant recipients [40] , patients on maintenance chemotherapy for acute lymphocytic leukemia, patients with congenital immunodeficiencies, and patients with human immunodeficiency virus (HIV)-related immunodeficiency. [41]

Infection of the intestinal mucosa may produce symptoms of inflammatory bowel disease. [42]

Intrapartum infections:
Intrauterine infections can lead to specific or permanent organ defects in the fetus (e.g. heart anomalies, eye diseases, micrognathy, chronic anemia, myocarditis, hepatitis, meconium peritonitis and central nervous system anomalies). [43-45]
• Thrombocytopenia is reported in 46% of cases [46]
• Rare cases of transient neonatal leukoerythroblastosis have been reported [47]
• In most reported B19 infections occurring during pregnancy, the fetus has not been adversely affected; however, in some cases B19 infection has been associated with fetal death. [48-50]
• The risk of fetal death attributable to maternal parvovirus infection is estimated at less than 10%.
• Fetal death most commonly occurs from the 10th through the 20th weeks of pregnancy.
• Although maternal infection appears to be common in late pregnancy, hydrops is relatively rare. [51]

A related member of the family Parvovirinae, Human Bocavirus, is discussed under 'Respiratory viruses • miscellaneous'

This disease is endemic or potentially endemic to all countries.

Parvovirus B19 infection in Brazil

Prevalence surveys:

28.15 to 1.8% of acute rash disease in Rio de Janeiro State (2002 publication) [52]
0.36% of exanthematous disease in Manaus (2005 publication) [53]
3.3% of patients suspected to have measles and/or rubella (Pernambuco, 2001 to 2004) [54]
9.2% of patients with maculopapular rash (Niteroi, Rio de Janeiro State, 1994 to 1998) [55]
5.7% of patients with fever and rash in Campinas (2003 to 2004) [56]
30% of patients with fever and rash, initially diagnosed as measles (Sao Paulo, 2000 to 2004) [57]
15.0% of patients with cytopenia of unknown origin, vs. 12.7% of controls (Sao Paulo, 2006 to 2009) [58]
18.6% of patients with sickle-cell disease and beta-thalassemia major, vs. 1% of healthy blood donors (2012 publication) [59]

Seroprevalence surveys:

62.8% of HIV-positive patients in Rio de Janeiro (2009 publication) [60]
65% of patients with sickle-cell disease and 60% of healthy blood donors (2012 publication) [61]

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Pediculosis

Agent	PARASITE - Insecta. Anoplura: Pediculus humanus, Phthirus pubis.
Reservoir	Human
Vector	Louse
Vehicle	Contact
Incubation Period	7d
Diagnostic Tests	Identification of adults and "nits."
Typical Adult Therapy	Permethrin 1%; or malthion 0.5%; or lindane OR Ivermectin 200 mcg/kg PO
Typical Pediatric Therapy	Permethrin 1%; or malthion 0.5%; or lindane OR Ivermectin 200 mcg/kg PO (> 15 kg body weight)
Clinical Hints	Pruritus in the setting of poor personal hygiene; adults or nits may be visible; note that the body louse (*Pediculus humanus* var. corporis; not the head louse) transmits diseases such as epidemic typhus, trench fever and relapsing fever.
Synonyms	Crab louse, Lausebefall, Pediculose, Pediculus capitus, Pediculus corporis, Pedikulose, Phthirus pubis, Pidocci. ICD9: 132 ICD10: B85

Clinical

Most louse infestations are asymptomatic, with only 15% to 36% of patients complaining of pruritis.
• The principal clinical finding consists of presence of the lice themselves, and their eggs ('nits'). [1] [2]

This disease is endemic or potentially endemic to all countries.

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Pentastomiasis - Armillifer

Agent	PARASITE - Pentastomid worm. Armillifer moniliformis and Porocephalus taiwana in Asia Armillifer [Porocephalus] armillatus and A. grandis in Africa
Reservoir	Amphibian Reptile Rodent
Vector	None
Vehicle	Snake meat Water Vegetation
Incubation Period	Unknown
Diagnostic Tests	Identification of larvae in tissue.
Typical Adult Therapy	Excision as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Intestinal obstruction, pneumonia and jaundice - related to mechanical obstruction by parasite; crescentic intraabdominal or intrathoracic calcifications on x-ray or chest or abdomen.
Synonyms	Armillifer, Porecephalus. ICD9: 128.8 ICD10: B83.8

Clinical

Symptoms are related to mechanical pressure and may include intestinal obstruction or "acute abdomen."
- Pneumonia, jaundice, pericarditis and pleurisy have also been reported.
- C-shaped or coiled calcifications may be visible on X-ray. [1] [2]
- Two fatal infections in children had been reported to 2003. [3]

This disease is endemic or potentially endemic to 19 countries.

Pentastomiasis - Armillifer in Brazil

The first case report of Armillifer infection in Brazil was published in 2006. [4]

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Pentastomiasis - Linguatula

Agent	PARASITE - Pentastomid worm. Linguatula serrata
Reservoir	Herbivore
Vector	None
Vehicle	Meat (liver or lymph nodes of sheep/goat)
Incubation Period	Unknown
Diagnostic Tests	Identification of larvae in nasal discharge.
Typical Adult Therapy	No specific therapy available
Typical Pediatric Therapy	As for adult
Clinical Hints	Pharyngeal or otic itching, cough, rhinitis or nasopharyngitis which follows ingestion of undercooked liver.
Synonyms	Linguatula, Marrara syndrome. ICD9: 128.8 ICD10: B83.8

Clinical

Infestation ("halzoun" or "marrara syndrome") is associated with pain and itching in the throat or ear, lacrimation, cough, hemoptysis, rhinorrhea or hoarseness. [1] [2] (Halzoun is also associated with infection by *Dicrocoelium dendriticum*) [3]
• Complications include respiratory obstruction, epistaxis, facial paralysis or involvement of the eye.

This disease is endemic or potentially endemic to 184 countries.

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Pericarditis - bacterial

Agent	BACTERIUM. Streptococcus pneumoniae, Staphylococcus aureus, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Ultrasonography and cardiac imaging techniques. Culture of pericardial fluid (include mycobacterial culture).
Typical Adult Therapy	Antimicrobial agent(s) appropriate to known or anticipated pathogen. Drainage as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, chest pain and dyspnea; patients are acutely ill and have overt signs such as venous distention, and an enlarged cardiac 'shadow'; concurrent pneumonia or upper respiratory infection may be present; case-fatality rate = 20%.
Synonyms	Bacterial pericarditis, Pericardite. ICD9: 074.23,074.2,115.03,420 ICD10: I30

Clinical

Viral pericarditis often follows a prodrome of upper respiratory infection.
• Typical findings include fever and chest pain. [1] [2]
• The pain may be pleuritic or positional (ie, exacerbated by bending forward) and associated with signs and symptoms of congestive heart failure.
• Concurrent myocarditis, pneumonia or pleuritis are often present.

This disease is endemic or potentially endemic to all countries.

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Perinephric abscess

Agent	BACTERIUM OR FUNGUS. Escherichia coli, other facultative gram negative bacilli, Candida albicans, et al
Reservoir	Human
Vector	None
Vehicle	None
Incubation Period	Variable
Diagnostic Tests	Urine and blood culture. Renal imaging (CT, etc).
Typical Adult Therapy	Antimicrobial agent(s) appropriate to known or anticipated pathogen. Surgery as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Unexplained fever, leukocytosis and flank pain; patients are typically over age 50, often diabetic; consider in the patient with nonresponsive 'pyelonephritis' or a renal mass (by examination or x-ray).
Synonyms	

Clinical

Symptoms may be overt or subtle, and limited to unexplained fever; indeed, 33% of such lesions are first diagnosed at autopsy.

- Typical patients are female and over the age of 50. [1-3]
- Diabetes and evidence for preceding or current urinary tract infection or bacteremia (including endocarditis) may be present.

This disease is endemic or potentially endemic to all countries.

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Perirectal abscess

Agent	BACTERIUM. Various (often mixed anaerobic and aerobic flora)
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture of drainage material.
Typical Adult Therapy	Surgical drainage and antibiotics effective against fecal flora
Typical Pediatric Therapy	As for adult
Clinical Hints	Anal or perianal pain with fever and a tender mass suggest this diagnosis; granulocytopenic patients commonly develop small, soft and less overt abscesses - often due to *Pseudomonas aeruginosa*.
Synonyms	

Clinical

Perirectal abscess is a self-defined illness usually associated with overt local pain, swelling, tenderness and fluctuance. [1]
• Abscesses in neutropenic patients are often more subtle, and may present as unexplained fever without marked local findings.

This disease is endemic or potentially endemic to all countries.

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Peritonitis - bacterial

Agent	BACTERIUM. Various (often mixed anaerobic and aerobic flora)
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture of blood and peritoneal fluid. Peritoneal fluid cell count may also be useful.
Typical Adult Therapy	Antimicrobial agent(s) appropriate to known or anticipated pathogens. Surgery as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Abdominal pain and tenderness, vomiting, absent bowel sounds, guarding and rebound; diarrhea may be present in children; search for cause: visceral infection or perforation, trauma, underlying cirrhosis (spontaneous peritonitis) etc.
Synonyms	Acute peritonitis, Bacterial peritonitis, Peritonite. ICD9: 567 ICD10: K65

Clinical

Bacterial peritonitis following trauma, infection or perforation of an abdominal viscus is usually overt clinically. [1]

Spontaneous bacterial peritonitis is somewhat more subtle, and should be suspected when unexplained deterioration occurs in a patient with ascites or chronic liver disease. [2] [3]
• As many as 30% of patients are asymptomatic, and the remainder present with fever, chills, abdominal pain, diarrhea, increasing ascites, encephalopathy or renal dysfunction.
• Abdominal tenderness, guarding and hypotension may be present.
• Bacteremia is a poor prognostic factor in these patients. [4]

This disease is endemic or potentially endemic to all countries.

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Pertussis

Agent	BACTERIUM. Bordetella pertussis An aerobic gram-negative coccobacillus
Reservoir	Human
Vector	None
Vehicle	Air Infected secretions
Incubation Period	7d - 10d (range 5d - 21d)
Diagnostic Tests	Culture & direct fluorescence (nasopharynx). Alert laboratory when suspected. Serology.
Typical Adult Therapy	Respiratory precautions. Erythromycin 500 mg QID X 10d. Alternatives: Azithromycin, Clarithromycin
Typical Pediatric Therapy	Respiratory precautions: Erythromycin 10 mg/kg QID X 10d. Alternatives: Azithromycin, Clarithromycin
Vaccines	DTaP DTP
Clinical Hints	Coryza, paroxysmal cough, occasional pneumonia or otitis; lymphocytosis; most often diagnosed in young children; epistaxis and subconjunctival hemorrhage often noted; seizures (below age 2); case-fatality rate = 0.5%.
Synonyms	Bordetella holmesii, Bordetella parapertussis, Bordetella pertussis, Coqueluche, Keuchhusten, Kikhosta, Kikhoste, Kinkhoest, Parapertussis, Pertosse, Syndrome coqueluchoide, Tos convulsa, Tos farina, Tosse convulsa, Tussis convulsa, Whooping cough. ICD9: 033 ICD10: A37

Clinical

WHO Case definition for surveillance: [1-3]
Clinical case definition
A person with a cough lasting at least 2 weeks with at least one of the following:
• paroxysms (i.e. fits) of coughing
• inspiratory .whooping.
• post-tussive vomiting (i.e. vomiting immediately after coughing)
• without other apparent cause
Laboratory criteria for diagnosis
• Isolation of *Bordetella pertussis*, or
• Detection of genomic sequences by polymerase chain reaction (PCR)
Case classification
• Suspected: A case that meets the clinical case definition.
• Confirmed: A person with a cough that is laboratory-confirmed.

Acute illness:
Following an incubation period of 7 to 10 days (range 6 to 20) the patient develops coryza and cough (the catarrhal stage).
• After one to two weeks, the cough progresses into the paroxysmal stage. [4] [5]
• Post-tussive vomiting is common, and young children and older infants may exhibit an inspiratory "whoop."
• Among infants younger than six months, apnea is common and the whoop may be absent. [6]
• The paroxysmal stage lasts three to four weeks (range one to six).
• The convalescent stage lasts for two to four weeks.

Complications:
Infants are at increased risk of complications from pertussis, while pertussis among adolescents and adults tends to be milder and may be limited to a persistent cough. [7]
• Over 70% of infants younger than 6 months require hospitalization.
• Complications of pertussis can include secondary bacterial pneumonia (the most common cause of death in pertussis), seizures and encephalopathy. [8]
• Other, less serious complications include otitis media and dehydration.
• Severe coughing can lead to pneumothorax, epistaxis, subdural hematoma, acute carotid dissection with stoke [9], hernia, and rectal prolapse.
• Pertussis in adults is often characterized by unexplained prolonged cough. [10] [11]

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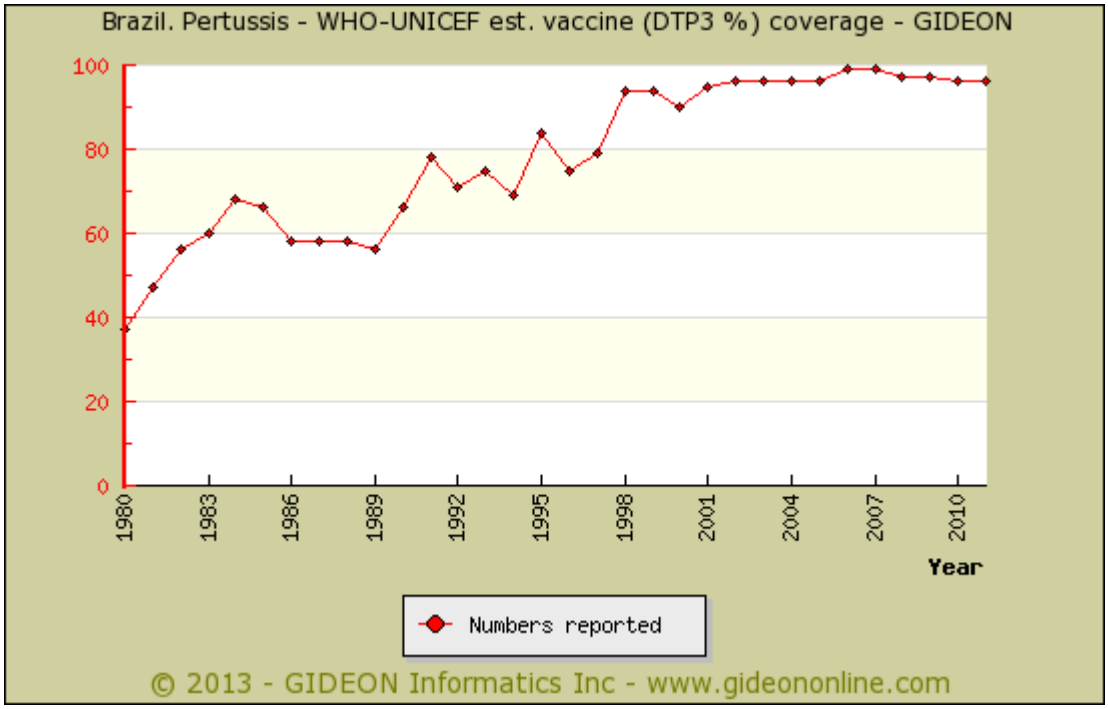
- Pertussis-RSV infection is common. [12]
- Rare cases of acute disseminated encephalomyelitis [13] and hemolytic-uremic syndrome have been ascribed to pertussis [14] [15]
- Human Bocavirus infection may mimic the symptoms of pertussis [16]

Parapertussis is caused by *Bordetella parapertussis*, and shares many of the clinical features of pertussis.
- 70% of infections are asymptomatic.

This disease is endemic or potentially endemic to all countries.

Pertussis in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years



Graph: Brazil. Pertussis - WHO-UNICEF est. vaccine (DTP3 %) coverage

Pertussis was confirmed in 12% of children <1 year of age in 11 countries admitted to pediatric intensive care units (PICU)

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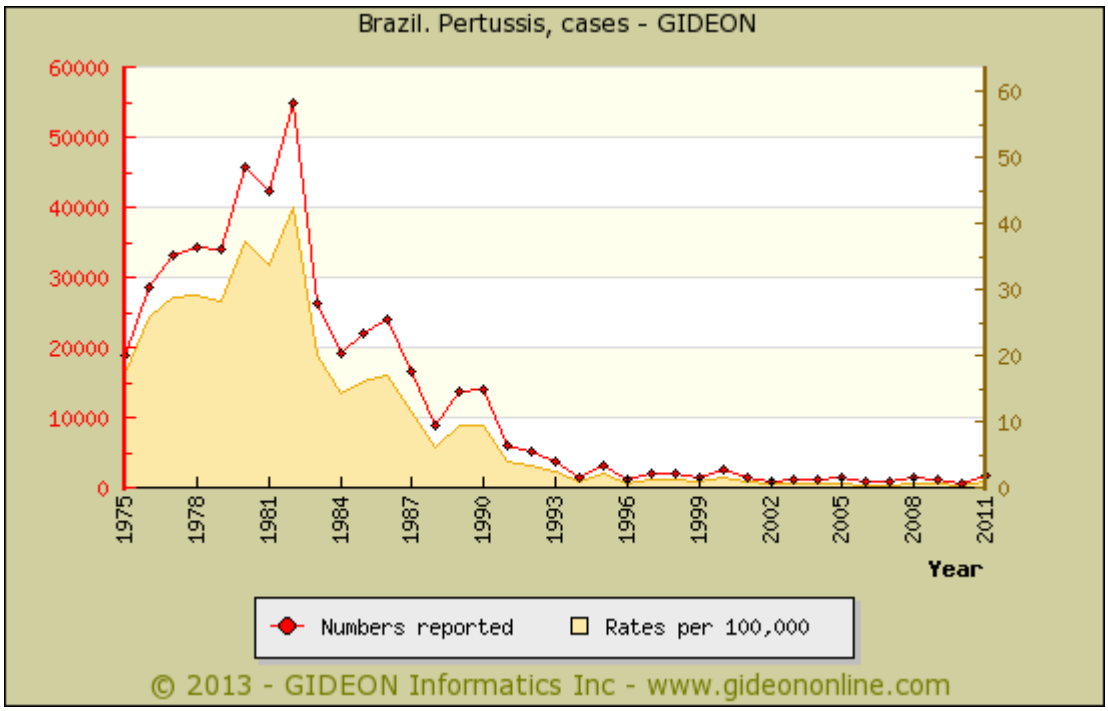
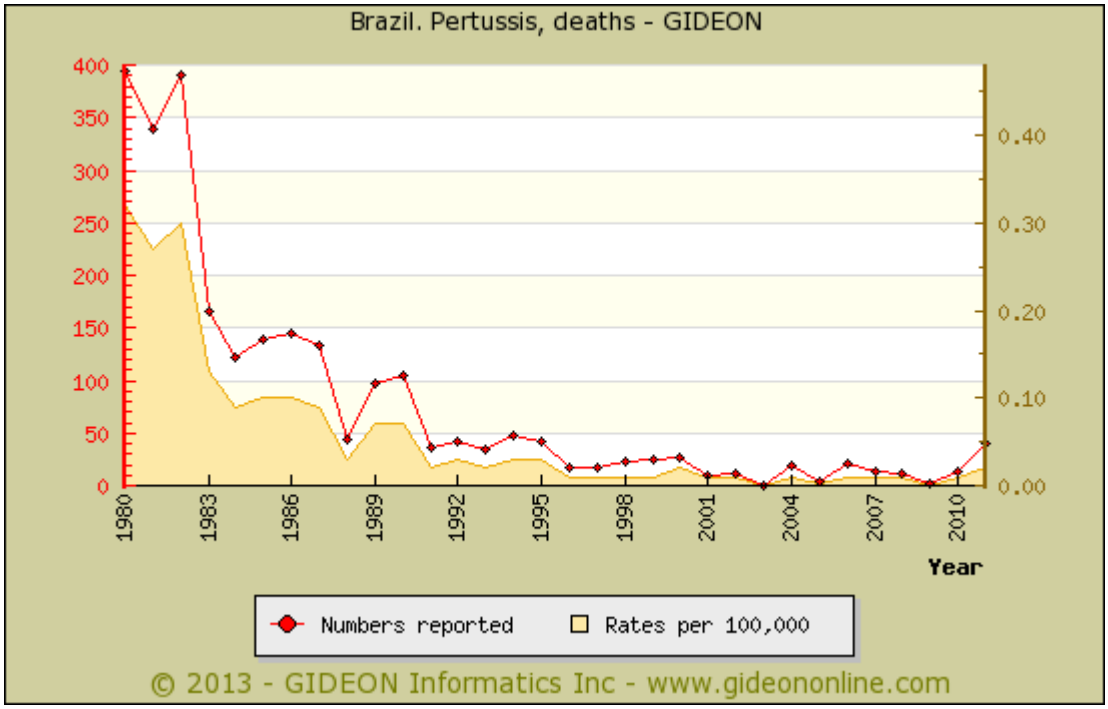
and pediatric wards presenting with respiratory failure, apnea, bradycardia, or cough accompanied by paroxysms, vomiting, whoop or cyanosis - 11% in Brazil (2001 to 2004) [17]



Graph: Brazil. Pertussis, cases

Notes:
1. A total of 326,142 cases were reported during 1980 to 2001.



Graph: Brazil. Pertussis, deaths

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Pharyngeal & cervical space infx.

Agent	BACTERIUM. Streptococcus pyogenes, mixed oral anaerobes, etc.
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Careful examination of region and X-ray (or CT scan). Smear and culture of pus if available.
Typical Adult Therapy	Surgical drainage and parenteral antibiotics effective against oral flora
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, painful swelling and displacement of the tongue, fauces and other intraoral structures; dysphagia, dyspnea or jugular phlebitis may ensue in more virulent infections.
Synonyms	Cervical space infection, descending necrotizing mediastinitis, Lemmier's syndrome, Ludwig's angina, Post-anginal septicemia, Quinsy. ICD9: 682.0,682.1 ICD10: J36,J39.0,J39.1

Clinical

Signs and symptoms reflect the site of infection: [1]
• masticator, buccal, canine or parotid spaces
• submandibular, submaxillary and submandibular spaces (Ludwig's angina)
• lateral pharyngeal, retropharngeal or paratracheal spaces
• peritonsillar tissues (quinsy)
• jugular vein (post-anginal septicemia = Lemmiere's syndrome) [2] [3]

Lemmiere's syndrome is a potentially fatal infection caused by *Fusobacterium necrophorum*.
• The condition is most common among young healthy persons and typically begins with pharyngotonsillitis which spreads to the parapharyngeal spaces to produce septic phlebitis of the internal jugular vein. [4-6]
• Submandibular edema and tenderness along the sternocleidomastoid muscle are noted, usually unilaterally.
• After one to two weeks, the patient develops multiple metastatic abscesses of the lungs, muscles [7] , bones, joints • or rarely, brain.
• Hyperbilirubinemia and mild disseminated intravascular coagulation may be present.
• The case-fatality rate is 4% to 33%, even with appropriate antimicrobial therapy.

This disease is endemic or potentially endemic to all countries.

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Pharyngitis - bacterial

Agent	BACTERIUM. Most often Streptococcus pyogenes; Str. groups B, C, F and G are occasionally isolated
Reservoir	Human
Vector	None
Vehicle	Droplet Rarely food
Incubation Period	1d - 5d
Diagnostic Tests	Throat swab for culture or antigen detection (group A Streptococcus) ASLO titer may not indicate current infection
Typical Adult Therapy	Penicillin G or Penicillin V or other antistreptococcal antibiotic to maintain serum level for 10 days
Typical Pediatric Therapy	As for adult
Clinical Hints	Purulent pharyngitis and cervical lymphadenopathy usually indicate streptococcal etiology; however, viruses (mononucleosis, enteroviruses) and other bacteria (gonorrhea, diphtheria) should also be considered.
Synonyms	Acute pharyngitis, Bacterial pharyngitis, Mal di gola batterica, Oral thrush, Streptococcal pharyngitis, Tonsillitis - bacterial, Vincent's angina. ICD9: 034.0,462 ICD10: J02,J03

Clinical

This is a self-defined condition characterized by erythema and pain in the pharynx, often associated with fever, dysphagia and upper respiratory tract infection. [1]

This disease is endemic or potentially endemic to all countries.

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Pinta

Agent	BACTERIUM. Treponema carateum A microaerophilic gram-negative spirochete
Reservoir	Human
Vector	? Fly (black fly = Simulium)
Vehicle	Contact
Incubation Period	7d - 21d (range 3d - 60d)
Diagnostic Tests	VDRL & FTA (or MHTP) - as in syphilis.
Typical Adult Therapy	Benzathine Penicillin G 1.2 million units IM as single dose
Typical Pediatric Therapy	Benzathine Penicillin G: Weight <14 kg 300,000u IM Weight 14 to 28kg 600,000u IM Weight >28kg 1.2 million u IM
Clinical Hints	Acute, pruritic erythematous papules which evolve to chronic, enlarging dyschromic plaques; a generalized papulosquamous rash may be noted later in the illness; lesions may recur for 10 years in some cases.
Synonyms	Azul, Carate, Empeines, Mal del pinto, Tina. ICD9: 103 ICD10: A67

Clinical

The primary lesion is usually located on exposed areas of the arms or legs, and is accompanied by painless regional lymphadenopathy. [1] [2]

- Secondary lesions ('pintids') appear after several months and may disseminate to other areas of the skin. [3] [4]
- There is no latent stage.
- Late pinta is characterized by skin atrophy and hypopigmentation.

Results of dark field microscopy and serological tests are indistinguishable from those of syphilis.

This disease is endemic or potentially endemic to 7 countries.

Pinta in Brazil

Sporadic infection is reported among the Indians of western Amazonas and along the Rio Negro.

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Pityriasis rosea

Agent	UNKNOWN. Human herpesvirus 7 has been implicated
Reservoir	Unknown
Vector	Unknown
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Clinical features.
Typical Adult Therapy	Supportive; ultraviolet B exposure is suggested
Typical Pediatric Therapy	As for adult
Clinical Hints	3 to 8 week illness; herald patch followed by crops of salmon-colored macules and papules; pruritus; systemic symptoms rare.
Synonyms	

Clinical

Pityriasis rosea is a mild exanthem characterized by oval or round macules or papules which evolve following the appearance of a "herald patch" (80% of cases). [1-5]
• Fine desquamation and pruritus are common.
• Rarely, the condition may recur. [6]
• In Black patients, Pityriasis rosea may present with facial and scalp involvement, post-inflammatory disorders of pigmentation and papular lesions. [7]
• Acral lesions [8] or dermal follicles may predominate in some cases. [9]
• The disease should be distinguished from secondary syphilis • the latter characterized by prominent lymphadenopathy; lack of pruritis and herald patch; and accompanying fever and systemic signs. [10]

This disease is endemic or potentially endemic to all countries.

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Plague

Agent	BACTERIUM. Yersinia pestis A facultative gram-negative bacillus
Reservoir	Rodent Rabbit Cat Wild carnivore
Vector	Flea (Pulex; Xenopsylla)
Vehicle	Air Contact
Incubation Period	2d - 7d (range 1d - 14d)
Diagnostic Tests	Culture (blood, sputum, pus). Fluorescent (DFA) staining of pus. Nucleic acid amplification.
Typical Adult Therapy	Strict isolation. Gentamicin 2 mg/kg IV loading dose, then 1.7 mg/kg Q8h. OR Streptomycin 15 mg/kg q12h X 10d. OR Doxycycline 100 mg PO BID X 10d. OR Chloramphenicol 20 mg/kg PO QID
Typical Pediatric Therapy	Gentamicin 2 mg/kg IV loading dose, then 1.7 mg/kg Q8h OR Streptomycin 10 mg/kg q8h X 10d. OR Chloramphenicol 15 mg/kg PO QID X 10d
Vaccine	Plague
Clinical Hints	Suppurative lymphadenitis; septicemia; hemorrhagic pneumonia; history of animal contact in many cases; case-fatality rates for bubonic plague without therapy are 50% to 60%.
Synonyms	Black death, Black plague, Bubonic plague, Glandular plague, Hemorrhagic plague, Peste, Pneumonic plague, Saint Roch's disease, Yersinia pestis. ICD9: 020 ICD10: A20

Clinical

WHO Case definition for surveillance:
Disease characterized by rapid onset of fever, chills, headache, severe malaise, prostration, with
• bubonic form: extreme painful swelling of lymph nodes (buboes)
• pneumonic form: cough with blood-stained sputum, chest pain, difficult breathing
• Note: Both forms can progress to a septicemic form with toxemia: sepsis without evident buboes rarely occurs.
Laboratory criteria for diagnosis
• Isolation of *Yersinia pestis* in cultures from buboes, blood, CSF or sputum or
• Passive hemagglutination (PHA) test, demonstrating an at least fourfold change in antibody titer, specific for F1 antigen of *Y. pestis*, as determined by the hemagglutination inhibition test (HI) in paired sera.
Case classification
• Suspected: A case compatible with the clinical description. May or may not be supported by laboratory finding of Gram stain negative bipolar coccobacilli in clinical material (bubo aspirate, sputum, tissue, blood).
• Probable: A suspected case with Positive direct fluorescent antibody (FA) test for *Y. pestis* in clinical specimen; or passive hemagglutination test, with antibody titer of at least 1:10, specific for the F1 antigen of *Y. pestis* as determined by the hemagglutination inhibition test (HI); or epidemiological link with a confirmed case.
• Confirmed: A suspected or probable case that is laboratory-confirmed.

Symptoms:
The initial features of plague are nonspecific and include fever, chills, myalgias, pharyngitis, headache.
• Regional lymph nodes are enlarged, painful and extremely tender. [1]
• Additional features, notably in patients with septicemic or pneumomic plague include nausea, vomiting, diarrhea, hematemesis, hematochezia, cough with hemoptysis, dyspnea and signs of meningitis.

Signs:
The physical examination reveals fever, tachycardia, tachypnea, and hypotension.
• Buboes are usually inguinal (60% to 90%), axillary (30%), cervical (10%), or epitrochlear (10%).
• Femoral nodes are involved more frequently than inguinal nodes. [2]
• Nodes are typically no larger than 5 cm, extremely tender, erythematous, and surrounded by a boggy hemorrhagic area.
• A maculopapular lesion may be found at the site of the flea bite.
• Acral cyanosis, ecchymosis, petechiae, and digital gangrene are seen in patients with septicemic plague.
• Signs of septic shock or DIC may also be present.

Plague pneumonia: [3]
Primary plague pneumonia follows an incubation period of 1 to 3 days, with sudden onset of fever, chills, headache and

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malaise. [4]
• Cough is prominent, with copious sputum production, chest pain and dyspnea.
• Profuse hemoptysis is common.
• Physical examination reveals rales and diffuse areas of dullness to percussion. [5]
• Untreated plague pneumonia is virtually always fatal.

Rare instances of gastrointestinal plague have been associated with ingestion of contaminated meat. [6]

This disease is endemic or potentially endemic to 41 countries.

Plague in Brazil

Plague was first reported in Brazil in 1899. [7] [8]

Plague is currently or recently endemic to: [9]
Bahia State:
- Biritinga Municipio
- Candeal Municipio
- Central Municipio
- Conceicao Minicipio
- Feira de Santana Municipio
- Iraquara Municipio
- Irece Municipio
- Itaberaba Municipio
- Jussara Municipio
- Retirolandia Municipio
- Riachao do Jacuipe Minicipio
- Senhor do Bonfim Municipio
- Serrinha Municipio
- Teofilandia Municipio

Ceara State: [10]
- Chapada do Araripe
- The Ibiapaba, Baturite, Machado, Matas, Pedra Branca, and Uruburetama mountains

Paraiba State:
- Araba Municipio
- Barra de S. Rosa Municipio
- Cubati Municipio
- Olivedos Municipio
- Queimadas Municipio
- Remigo Municipio
- Solanea Municipio

Prior activity existed in Alagoas, Bahia, Ceara, Minas Gerais, Paraiba, Pernambuco, Piaui, Rio Grande do Norte.

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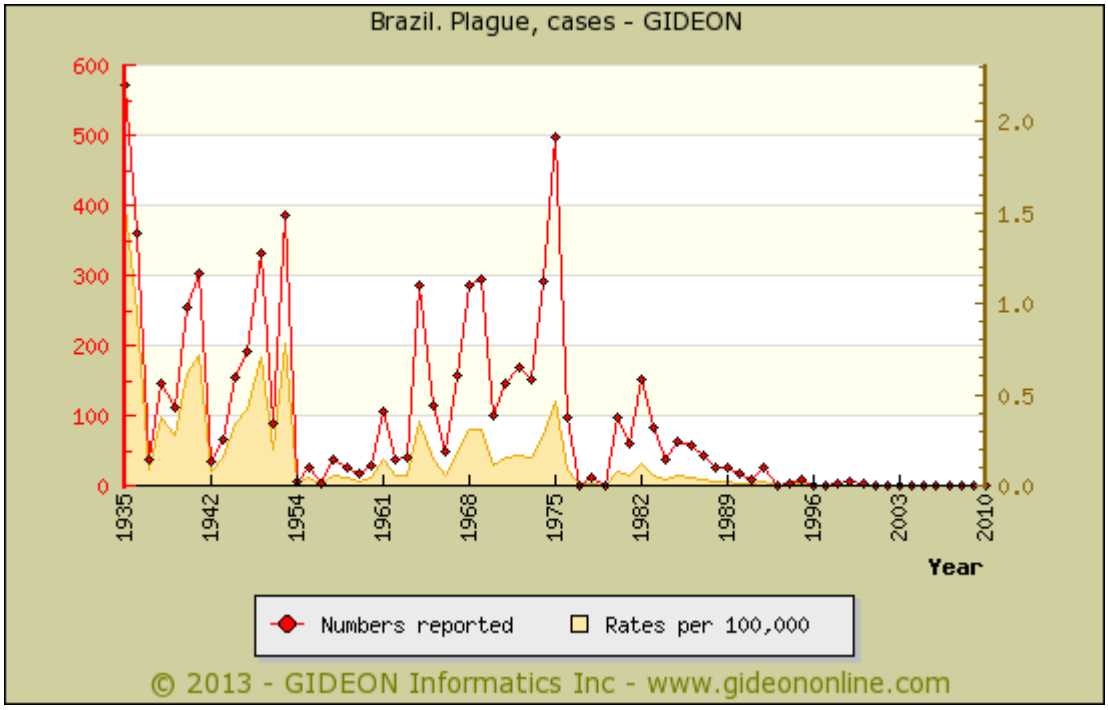

Graph: Brazil. Plague, cases

Notes:
1. 5,638 cases were reported during 1899 to 1929; 535 during 1930 to 1934; 1,223 (490 fatal) during 1935 to 1939; 812 (205 fatal) during 1940 to 1944; 1,040 (182 fatal) during 1945 to 1959. [11]
1. 796 cases were reported during 1980 to 2000 - including 397 from Ceara and 330 from Bahia.
Individual years:
1969 - 99 cases in Bahia and 98 in Ceara.
1995 - All from 6 municipalities of Bahia State.
1997 - From Ceara State.
1998 - From Bahia State (Jussara, Irece and Serrinha).
1999 - From Bahia State (Jussara, Irece and Serrinha).
2000 - From Bahia State (Serrinha).

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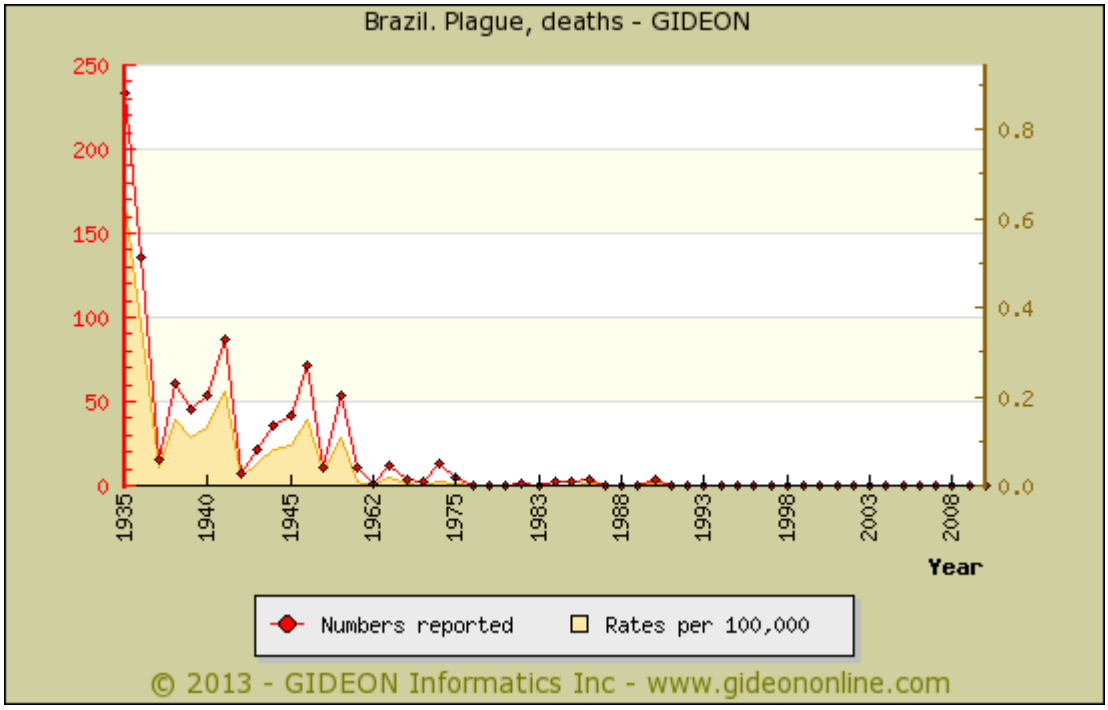

Graph: Brazil. Plague, deaths

Seroprevalence surveys:

0% of humans, dogs and rodents in Bahia (2011 publication) [12]
3.22% of suspected plague cases, 0.29% of rodents, 0.90% of dogs and 0.64% of cats (1983 to 1992) [13]
85% of dogs and 15% of cats in plague foci of Ceara (1997 to 2006) [14]

A local flea species, *Polygenis bohlsi jordani*, is identified as the probable Brazilian vector.

Notable outbreaks:

1899 - An outbreak of bubonic plague was reported in Santos. [15]
1986 - An outbreak of plague was reported in Paraiba state. [16-18]

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Plesiomonas infection

Agent	BACTERIUM. Plesiomonas shigelloides A facultative gram-negative bacillus
Reservoir	Fish Animal Soil Reptile Bird
Vector	None
Vehicle	Water Food
Incubation Period	1d - 2d
Diagnostic Tests	Stool culture - alert laboratory when this organism is suspected. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions. Antimicrobial agent per in-vitro susceptibility (Ciprofloxacin considered 'drug of choice')
Typical Pediatric Therapy	Stool precautions. Antimicrobial agent per in-vitro susceptibility. Fluid replacement
Clinical Hints	Fever, abdominal pain, vomiting and severe diarrhea; symptoms often persist for 2 to 4 weeks; follows ingestion of shellfish or recent travel to developing countries in many cases.
Synonyms	Plesiomonas shigelloides. ICD9: 008.8 ICD10: A04.8

Clinical

The infection is characterized by a self-limited diarrhea, often with blood or mucus in stool. [1]
- Watery diarrhea is most common; however, a cholera-like illness with as many 30 bowel movements per day may occur.
- Associated abdominal pain may mimic that of appendicitis, including enlargement of peritoneal lymph nodes. [2]
- Fecal leucocytes are present.
- As many as 30% of cases continue for over four weeks, and symptoms may persist for as long as 3 months.
- *Plesiomonas* has been rarely associated with fatal meningitis and septicemia [3-13], proctitis [14], cellulitis and dermal abscesses [15], pneumonia [16], pleural effusion [17], osteomyelitis [18], cholecystitis [19], peritonitis [20] [21], salpingitis [22], epididymo-orchitis [23], pancreatitis [24], splenic abscess [25], keratitis [26] and endophthalmitis. [27]
- 21 cases of *Plesiomonas* septicemia had been reported as of 1996. [28]

This disease is endemic or potentially endemic to all countries.

Plesiomonas infection in Brazil

Prevalence surveys:

0% of patients with diarrhea, vs. 0.3% of controls (2009 publication) [29]

Plesiomonas shigelloides is present in water environments of Rio de Janeiro city (1995 publication) [30]

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Pleurodynia

Agent	VIRUS - RNA. Picornaviridae: Coxsackievirus
Reservoir	Human
Vector	None
Vehicle	Air Fecal-oral Fomite
Incubation Period	3d - 5d
Diagnostic Tests	Viral culture (throat, stool). Serology. Nucleic acid amplification.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Sore throat followed by pleuritic chest pain - a late summer illness in temperate regions; pain is often recurrent and appears in 'waves' - local pressure on affected area may elicit identical pain; usually resolves within one week.
Synonyms	Balme disease, Bamie disease, Bornholm disease, Devil's grip, Drangedal disease, Epidemic benign dry pleurisy, Epidemic myalgia, Sylvest's disease. ICD9: 074.1 ICD10: B33.0

Clinical

Pleurodynia is characterized by a prodrome of upper respiratory tract infection, followed by abrupt onset of pleuritic chest pain. [1]
• The pain may be severe and lead to a misdiagnosis of myocardial infarction.
• Some patients present with abdominal pain suggestive of peritonitis.
• Important diagnostic features include appearance of cases in clusters (often in late summer to autumn) and lack of leucocytosis or other findings suggestive of pneumonia or peritonitis.

This disease is endemic or potentially endemic to all countries.

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Pneumocystis pneumonia

Agent	FUNGUS. Ascomycota ?, Archiascomycetes, Pneumocystidales: Pneumocystis jiroveci (now separate from Pneumocystis carinii)
Reservoir	Human
Vector	None
Vehicle	? Air
Incubation Period	4d - 8w
Diagnostic Tests	Identification of organisms in induced sputum, bronchial washings, tissue. Serology. Nucleic acid amplification.
Typical Adult Therapy	Therapy: Sulfamethoxazole/trimethoprim 25 mg/5 mg/kg QID X 14d. OR Pentamidine 4 mg/kg/d X 14d. OR Dapsone + Trimethoprim. OR Atovaquone OR Primaquine + Clindamycin Prophylaxis - similar, but at altered dosage. Dapsone also used.
Typical Pediatric Therapy	Therapy: Sulfamethoxazole/trimethoprim 25 mg/5 mg/kg QID X 14d. OR Pentamidine 4 mg/kg/d X 14d. OR Dapsone + Trimethoprim. OR Atovaquone OR Primaquine + Clindamycin Prophylaxis - similar, but at altered dosage.
Clinical Hints	Dyspnea, hypoxia and interstitial pneumonia; usually encountered in the setting of severe immune suppression (AIDS, leukemia, etc); roentgenographic findings (typically bilateral alveolar pattern) may follow symptoms only after several days.
Synonyms	PCP, Pneumocystis carinii, Pneumocystis jiroveci. ICD9: 136.3 ICD10: B59

Clinical

P. jiroveci infection often presents as a self-limiting upper respiratory tract infection in infants, predominantly in the age group 1.5 to 4 months of age.

The major presenting symptoms are shortness of breath, fever, and a nonproductive cough. [1]
- Sputum production, hemoptysis and chest pain are rarely encountered. [2]
- Tachypnea and tachycardia are usually prominent
- Children may demonstrate cyanosis, flaring of the nasal alae, and intercostal retractions.

Lung auscultation is usually not helpful, with rales present in only 1/3 of adults with this disease.
- The x-ray usually shows bilateral diffuse infiltrates extending from the perihilar region. [3]
- Other findings can unilateral infiltrates, nodules, cavities, pneumatoceles, hilar lymphadenopathy and pleural effusion.
- Patients receiving aerosolized pentamidine as prophylaxis have an increased incidence of apical infiltrates and pneumothorax.
- Impaired oxygenation is common.

Extrapulmonary infection by *P. jiroveci* may occur in as many as 3% of infected patients and is reported as an unexpected finding at autopsy.
- The main sites of involvement are lymph nodes, spleen, liver, bone marrow, gastrointestinal tract, eyes [4] , thyroid, adrenal glands, and kidneys.
- The clinical correlate of these findings is rapidly progressive multisystem disease, an enlarging thyroid mass, pancytopenia, retinal infiltrates, pleural effusion, splenic lesions, and calcifications in the spleen, liver, adrenal, or kidney.
- Rare instances of intestinal pseudotumor [5] and cutaneous infection have been reported. [6]

This disease is endemic or potentially endemic to all countries.

Pneumocystis pneumonia in Brazil

Pneumocystosis was the cause of death in 8% of autopsied AIDS patients (Amazonas, 1996 to 2003) [7]

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Pneumonia - bacterial

Agent	BACTERIUM. Streptococcus pneumoniae, Klebsiella pneumoniae ssp pneumoniae, other aerobic and facultative gram negative bacilli, etc.
Reservoir	Human
Vector	None
Vehicle	Droplet Endogenous infection
Incubation Period	1d - 3d
Diagnostic Tests	Culture of sputum, blood. Analyze ("grade") sputum cytology to assess significance of culture.
Typical Adult Therapy	Antimicrobial agent(s) appropriate to known or suspected pathogen
Typical Pediatric Therapy	As for adult
Vaccine	Pneumococcal
Clinical Hints	Rigors ("shaking chills"), pleuritic pain, hemoptysis, lobar infiltrate and leukocytosis; empyema and lung abscess suggest etiology other than pneumococcus; foul sputum with mixed flora may herald anaerobic (aspiration) pneumonia.
Synonyms	Bacterial pneumonia, Empiema, Empyeem, Empyem, Empyema, Empyeme, Lung abscess, Neumonia, Pleurisy, Pneumococcal infection - invasive, Pneumococcal pneumonia, Polmonite batterica, Streptococcus pneumoniae, Streptococcus pneumoniae - invasive. ICD9: 481,482,483,484 ICD10: J13,J14,J15,J17,J18,J85,J86

Clinical

The designation "Pneumonia • bacterial" in this module is generic, and includes a large variety of etiological agents and anatomical presentations (ie, empyema, lung abscess, lobar• vs. broncho-pneumonia, etc.)
• The clinical features of bacterial pneumonia are largely determined by the infecting species and clinical setting. [1-4]
• All forms are characterized by fever, chest pain, productive cough, and physical or roentgenographic evidence for pulmonary consolidation.

Etiological associations:
• AIDS: *Pneumocystis jiroveci*, Mycobacteria (non-tuberculous), Tuberculosis, Nocardiosis, Cryptococcosis, Cytomegalovirus
• Animal contact: Q-fever, Ornithosis
• Aspiration: Oral Anaerobes; if nosocomial, Enterobacteriaceae, *Acinetobacter*, *Pseudomonas*
• Cystic fibrosis (Fibrocystic disease) • *Burkholderia cepacia*
• Drowning ("near-drowning"): *Pseudoallescheria boydii*
• Endocarditis: *Staphylococcus aureus*
• Immunosuppression: *Aspergillosis*, Cryptococcosis, Nocardiosis, *Pneumocystis jiroveci*, Cytomegalovirus
• Infant: see Respiratory syncytial virus, Parainfluenza virus, Respiratory viruses • misc.
• Influenza: Influenza virus, *Streptococcus pneumoniae*, *Staphylococcus aureus*
• Myeloma: *Streptococcus pneumoniae*
• Nosocomial pneumonia: Enterobacteriaceae, *Acinetobacter*, *Pseudomonas*, *Staphylococcus aureus*
• Pulmonary alveolar proteinosis: *Nocardia*
• Traveler or tourist: Histoplasmosis, Legionellosis, Melioidosis

This disease is endemic or potentially endemic to all countries.

Pneumonia - bacterial in Brazil

Bacterial pneumonia was the cause of death in 17% of autopsied AIDS patients (Amazonas, 1996 to 2003) [5]

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Poliomyelitis

Agent	VIRUS - RNA. Picornaviridae, Picornavirus: Polio virus
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Dairy products Food Water Fly
Incubation Period	7d - 14d (range 3d - 35d)
Diagnostic Tests	Viral culture (pharynx, stool). Serology. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions; supportive
Typical Pediatric Therapy	As for adult
Vaccines	Poliomyelitis - injectable Poliomyelitis - oral
Clinical Hints	Sore throat, headache, vomiting and myalgia followed by flaccid paralysis; meningeal involvement in 1% of cases - paralysis in only 0.1%. paralysis tends to be more extensive in adult patients.
Synonyms	Acute flaccid paralysis, Heine-Medin disease, Infantile paralysis, Kinderlahmung, Kinderverlamming, Paralisi infantile, Paralisis flaccida, Paralisis flacida aguda, PFA (Paralisis Flacidas Agudas), Polio, Poliomyelite, Poliomyelitt. ICD9: 045 ICD10: A80

Clinical

CDC (The United States Centers for Disease Control) case definition for surveillance:
For surveillance purposes, the CDC (The United States Centers for Disease Control) case definition of paralytic poliomyelitis requires, "Acute onset of a flaccid paralysis [1] of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss."
• A 'confirmed case' requires persistence of the neurological deficit 60 days after onset of initial symptoms, fatal illness or unknown follow-up status.

The WHO Case definition for surveillance includes any child under fifteen years of age with acute, flaccid paralysis or any person with paralytic illness at any age when poliomyelitis is suspected. [2]

Poliomyelitis is typically a late summer illness in temperate climates, and often begins as a mild upper respiratory tract infection.
• In some cases, the disease follows vaccination (live vaccine) or recent contact with a vaccinee.
• Patients have been known to excrete virus for as long as ten years following an episode of poliomyelitis [3]
• Antecedent injection in a given site may precipitate paralytic poliomyelitis in the same limb. [4-9]

90% to 95% of poliomyelitis infections are asymptomatic.
• Symptoms include fever, sore throat, headache, vomiting and still neck.
• Paralysis is typically asymmetrical, and most often involves the lower extremities.
• Bulbar paralysis or encephalitis may occur in patients in the absence of limb paralysis.
• 4% to 8% experience minor symptoms, and 1% to 2% develop paralysis.
• Paralysis is most common in the very young and very old, following minor blunt trauma to a limb, and among persons who had undergone tonsillectomy.
• The case/fatality rate for paralytic poliomyelitis in 2% to 10%.

This disease is endemic or potentially endemic to 87 countries. Although Poliomyelitis is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Poliomyelitis in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]

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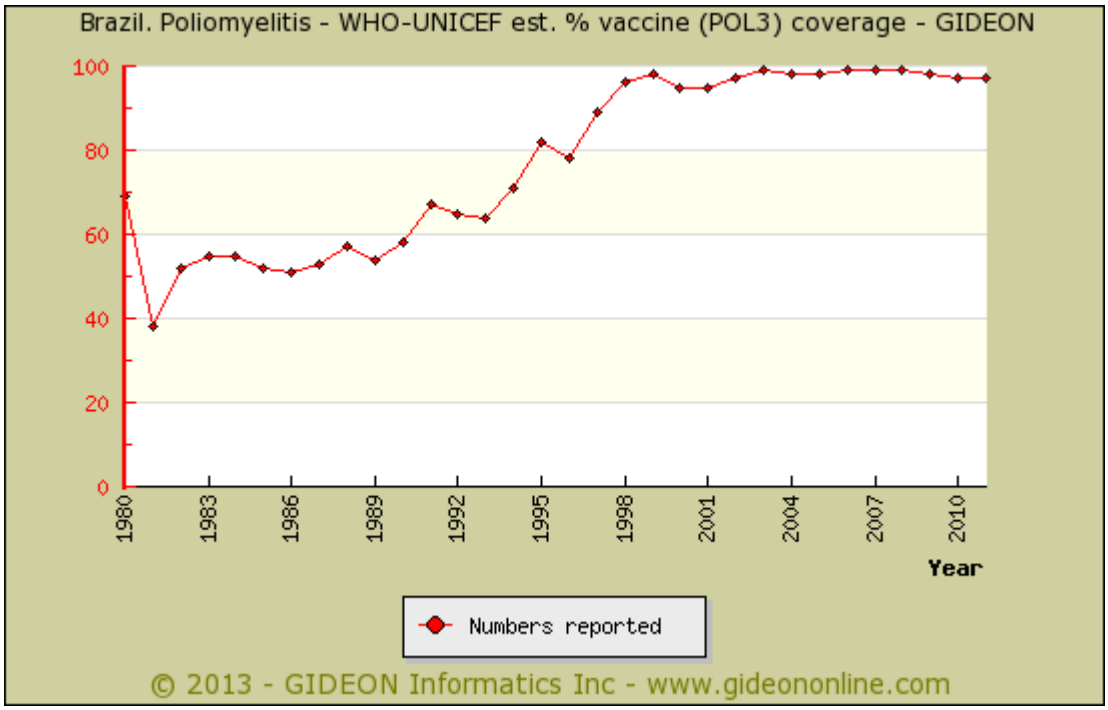
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years



Graph: Brazil. Poliomyelitis - WHO-UNICEF est. % vaccine (POL3) coverage

Seroprevalence surveys:

88.1% of persons in Sao Paulo toward poliovirus type 1, 88.8% toward type 2 and 61.6% toward type 3 (1999 to 2005) [10]

94.6% of persons in Sao Paulo toward poliovirus type 1, 98.8% toward type 2 and 91.9% toward type 3 (estimated, 2002 publication) [11]

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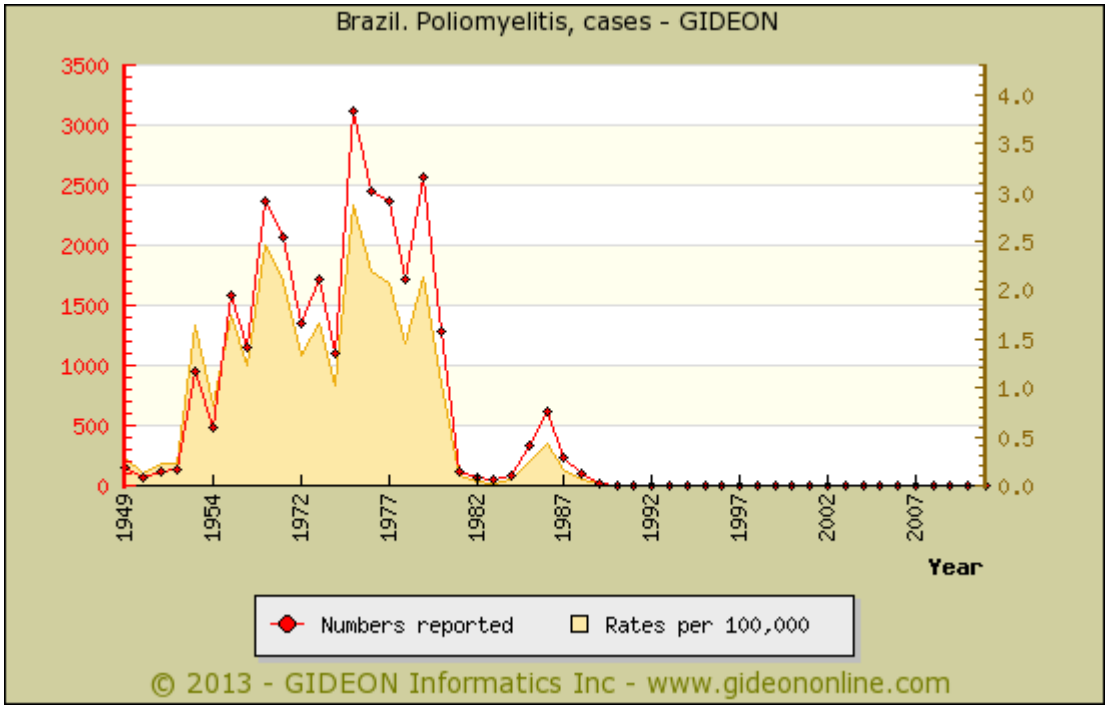
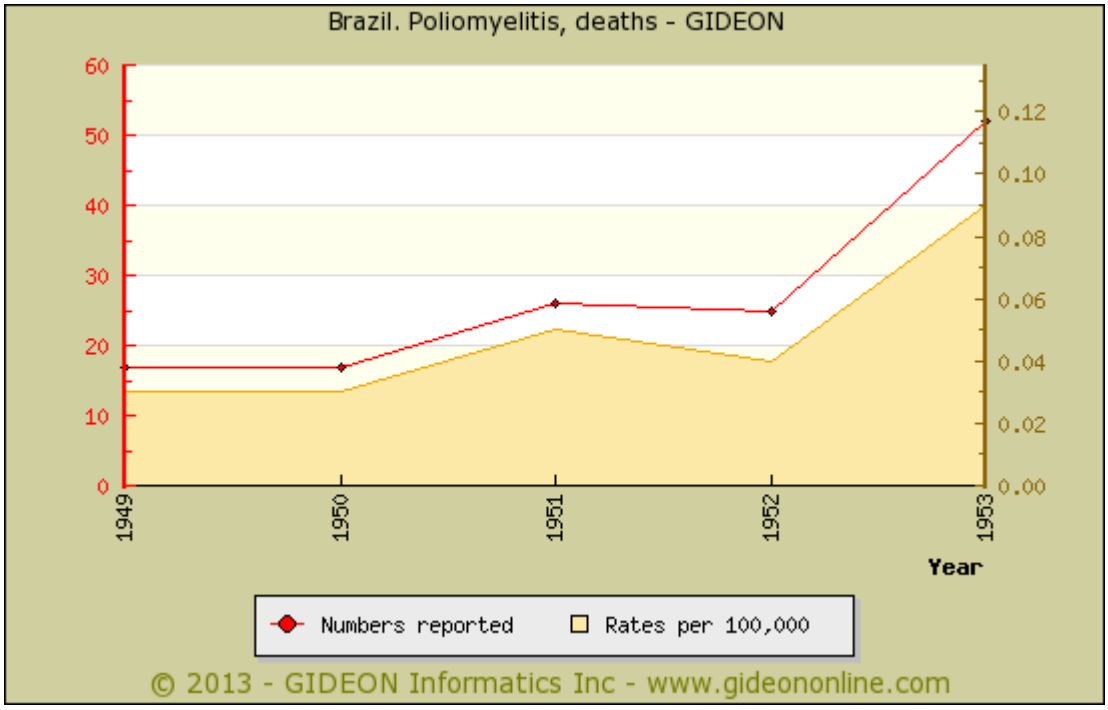


Graph: Brazil. Poliomyelitis, cases

Notes:

1. Historical data from references [12] [13]
2. The last wild-virus case was reported in 1989, and natural infection was declared eradicated as of 1991.



Graph: Brazil. Poliomyelitis, deaths

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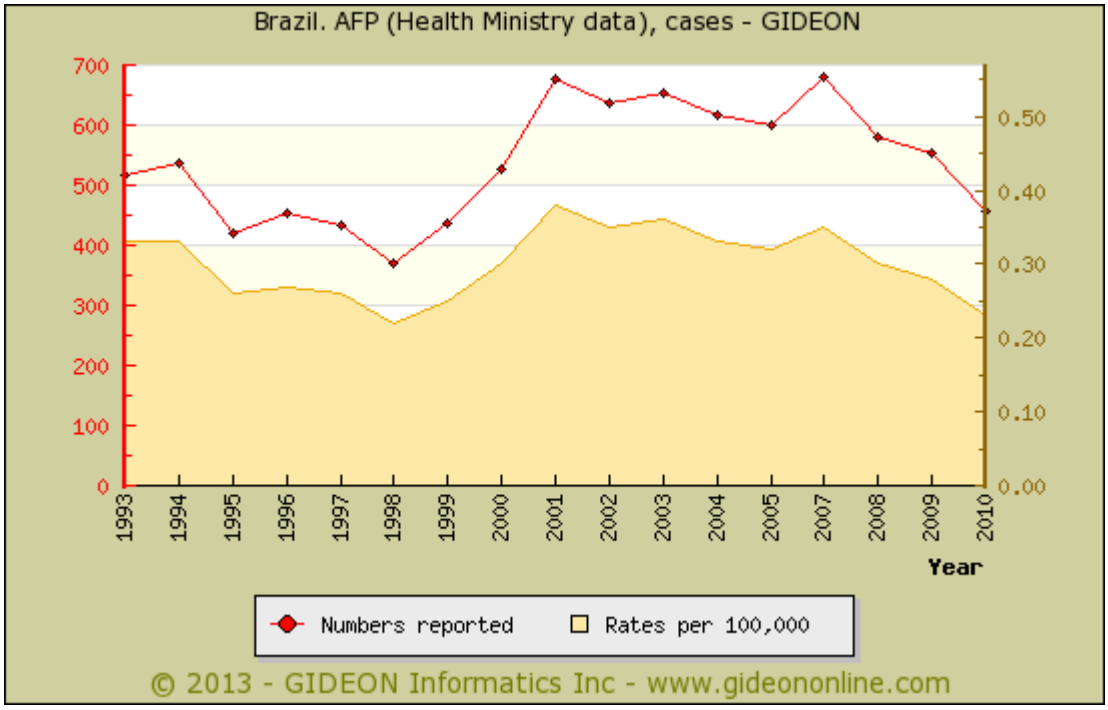
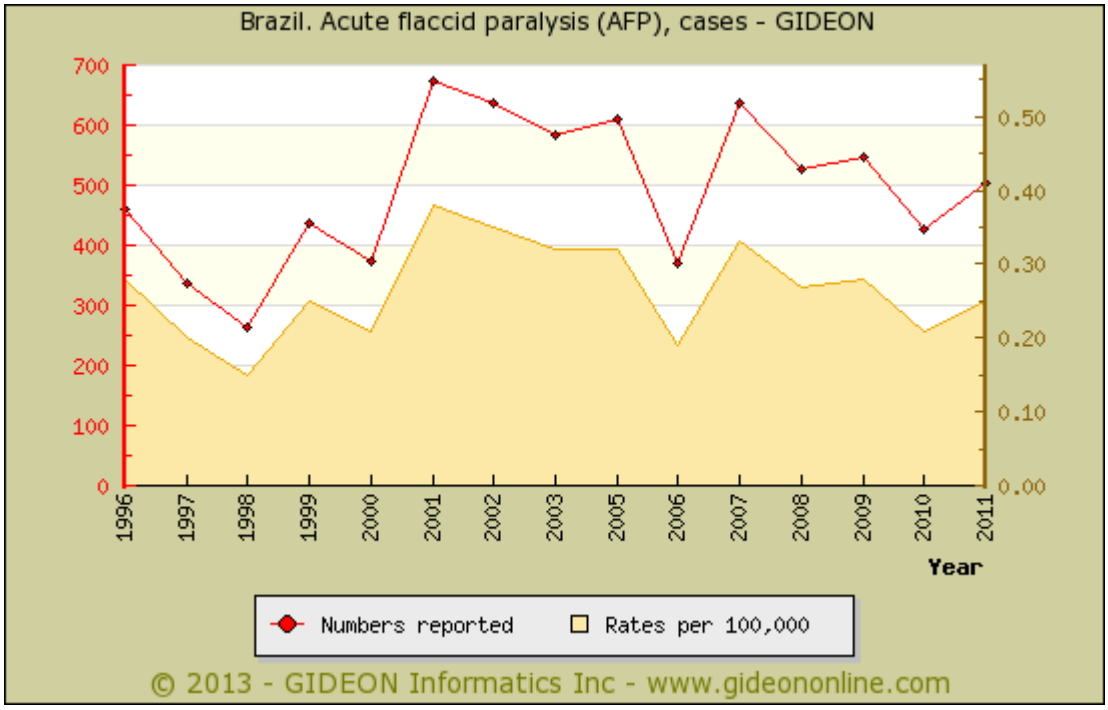


Graph: Brazil. AFP (Health Ministry data), cases

Notes:
1. 54 cases of vaccine-associated paralytic disease were reported during 1989 to 1991; 10 during 1995 to 2001. [14]
2. Enterovirus 71 accounted for 21.7% of acute flaccid paralysis episodes during 1989 to 1990.
3. 3,692 cases of Acute Flaccid Paralysis (AFP) were reported by the Health Ministry during 1993 to 2000.



Graph: Brazil. Acute flaccid paralysis (AFP), cases

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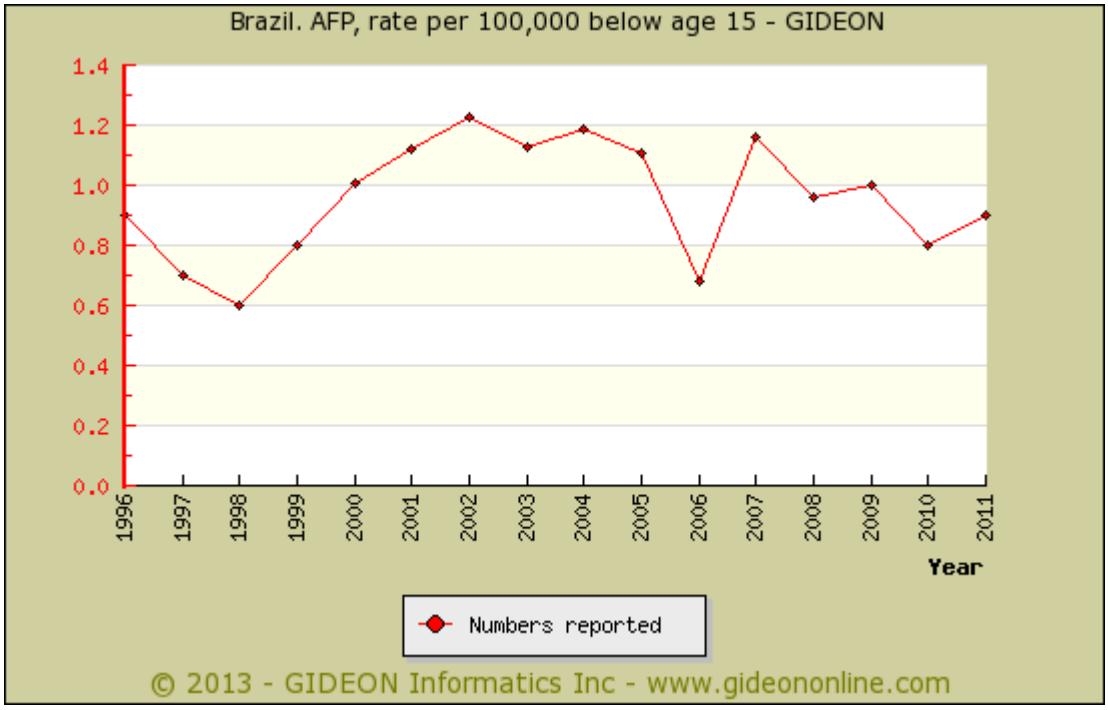

Graph: Brazil. AFP, rate per 100,000 below age 15

Notable outbreaks:

1962 (publication year) - An outbreak of poliomyelitis was reported in Patos de Minas. [15]

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Protothecosis and chlorellosis

Agent	ALGA. Prototheca wickerhamii; rarely Pr. zopfii, Pr. cutis Achloric algae Chlorella spp. contain chloroplasts
Reservoir	A rare animal pathogen (cat, dog, cattle). Chlorella spp. are reported to infect domestic and wild mammals.
Vector	None
Vehicle	Water Sewage Food Local trauma
Incubation Period	Unknown
Diagnostic Tests	Culture on fungal media. Biopsy. Nucleic acid amplification.
Typical Adult Therapy	Surgical excision. There are anecdotal reports of successful therapy with Amphotericin B, Ketoconazole and Itraconazole (latter 200 mg/day X 2 months) or voriconazole
Typical Pediatric Therapy	As for adult (Itraconazole 2 mg/kg/day X 2 months)
Clinical Hints	May follow immune suppression or skin trauma; dermal papules, plaques, eczematoid or ulcerated lesions; olecranon bursitis; systemic infection also reported.
Synonyms	Chlorellosis, Prototheca, Protothecosis. ICD9: 136.8 ICD10: B99

Clinical

Four forms of disease are reported:
• cutaneous infection
• olecranon bursitis
• disseminated
• onychomycosis [1-3]

The incubation period of protothecosis is unknown; however, infections which have followed trauma have appeared after approximately two weeks. [4]
• Most cases presented as a single painless, slowly progressive, well-circumscribed plaque or papulonodular skin lesion that may become eczematoid or ulcerated. [5-7]
• Soft tissue lesions favor the olecranon bursa; sites of minor trauma or corticosteroid injection; or surgical wounds which have been exposed to soil or water. [8] [9]
• Lesions enlarge gradually over weeks to months, with no tendency for healing.
• Other presentations have included tenosynovitis [10] ; algemia complicating immune-suppression [11] ; nasopharyngeal ulcerated lesion followed prolonged intubation, and infection of ambulatory peritoneal catheters.
• Skin lesions in HIV-infected patients are similar to those of healthy patients
• Peritonitis due to *P. wickerhamii* has been reported in peritoneal dialysis patients. [12]
• A case of subacute endocarditis due to *Prototheca wickerhamii* has been reported. [13]

A single case of *Chlorella* wound infection has been reported. [14]

This disease is endemic or potentially endemic to all countries.

Protothecosis and chlorellosis in Brazil

Two cases of cutaneous Protothecosis had been reported in Brazil to 2002 - the most recent from Bahia. [15]
- A third case report was published in 2006. [16]
- A rare instance of onychoprotothecosis has been reported. [17]

Prototheca zopfii is identified as an agent of canine infection [18] and bovine mastitis. [19] [20]
- *Prototheca wickerhamii* has been reported to infect goats in this country. [21] [22]

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Notable outbreaks:

2001 (publication year) - An outbreak of bovine mastitis due to *Prototheca zopfii* was reported in a dairy herd. [23]

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Pseudocowpox

Agent	VIRUS - DNA. Poxviridae, Parapoxvirus: Pseudocowpox virus
Reservoir	Cattle
Vector	None
Vehicle	Contact
Incubation Period	5d - 14d
Diagnostic Tests	Viral culture (skin lesion or exudate). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Umbilicated nodule on the hand following contact with cattle; mild regional lymphadenopathy.
Synonyms	Bovine papular stomatitis, Farmyard pox, Milker's nodule, Noduli mulgentinum, Sealpox. ICD9: 051.1 ICD10: B08.0

Clinical

Pseudocowpox is mild and self-limited and characterized by a red-to-blue dermal nodule associated with minimal lymphadenopathy. [1]

This disease is endemic or potentially endemic to all countries.

Pseudocowpox in Brazil

An outbreak of 'cowpox' in Mato Grosso do Sul during 1997 to 1998 involved 18 human cases.

An outbreak of 'cowpox' in Rio de Janeiro State during 1999 involved 10 cows and 3 humans. [2]

(Some experts suggest that the infection in these outbreaks was in fact pseudocowpox)

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Pyodermas (impetigo, abscess, etc)

Agent	BACTERIUM. Various (Staphylococcus aureus & Streptococcus pyogenes predominate)
Reservoir	Human
Vector	None
Vehicle	Endogenous & contact with infected secretions
Incubation Period	Variable
Diagnostic Tests	Clinical diagnosis usually sufficient. Aspiration of lesion for smear and culture may be helpful in some cases.
Typical Adult Therapy	Antibiotic directed at likely pathogens (Group A Streptococcus and Staphylococcus aureus)
Typical Pediatric Therapy	As for adult
Clinical Hints	Impetigo characterized by vesicles which progress to pustules ('honey-colored pus'); highly contagious; may be complicated by acute glomerulonephritis.
Synonyms	Acne vulgaris, Carbonchio, Carbuncle, Folicolite, Follicolite, Folliculite, Folliculitis, Follikulitis, Foroncolosi, Foronculose, Foruncolosi, Furunculosis, Furunkulose, Furunulose, Hydradenitis, Impetigine, Impetigo, Paronychia, Pyoderma. ICD9: 680,684,686 ICD10: L01,L02,L08.0,L73.2

Clinical

Impetigo is characterized by multiple superficial lesions caused by group A-hemolytic streptococci and/or *Staphylococcus aureus*. [1]
• The lesions consist of pustules that rupture and form a characteristic honey-colored crust.
• Lesions caused by staphylococci are associated with tense, clear bullae (bullous impetigo.).
• Ecthyma is a variant of impetigo that usually presents as punched-out ulcers on the lower extremities.
• Streptococcal impetigo is most common among children 2 to 5 years of age, and epidemics may occur in settings of poor hygiene, lower socioeconomic status or tropical climates.
• The most important complication of impetigo is poststreptococcal glomerulonephritis.

Folliculitis is most often caused by *Staphylococcus aureus*. [2]
• Blockage of sebaceous glands may result in sebaceous cysts, which may present as extensive abscesses or become secondarily infected.
• Infection of specialized sweat glands (hidradenitis suppurativa) occur in the axillae.
• Chronic folliculitis is a hallmark of acne vulgaris, in which normal flora (e.g., *Proprionibacterium acnes*) may play a role.
• Diffuse folliculitis may herald infection by *Pseudomonas aeruginosa* or *Aeromonas hydrophila* [3] , in waters that are insufficiently chlorinated and maintained at temperatures above 37 C. Although such Infection is usually self-limited, bacteremia and septic shock have been reported.

Erysipelas is caused by *Streptococcus pyogenes* and is characterized by abrupt onset of "fiery-red" superficial swelling of the face or extremities.
• The lesion is typically recognized by the presence of well-defined indurated margins, particularly along the nasolabial fold; rapid progression; and intense pain.
• Flaccid bullae may develop on the second or third day of illness; but extension to deeper soft tissues is rare.
• Desquamation occurs between the fifth and tenth days of illness.

Cellulitis is characterized by local pain, erythema, swelling, and heat.
• Cellulitis may be caused by any of a wide variety of bacteria or yeasts; however, *S. aureus* or *S. pyogenes* are most often implicated.
• A history of preceding trauma, insect bite, needle insertion or surgery is often present.
• Cultures of biopsy specimens or aspirates are positive in only 20% of cases.
• Infection by *S. aureus* often spreads out from a localized infection (abscess, folliculitis) or foreign body.
• Streptococcal cellulitis tends to be more diffuse and rapid in onset, and associated with lymphangitis and fever.
• Streptococci also cause recurrent cellulitis in the setting of lymphedema resulting from elephantiasis or lymph node damage.
• Recurrent staphylococcal cutaneous infections are encountered in patients with "Job's syndrome" (eosinophilia and elevated serum levels of IgE); and nasal carriers of staphylococci.

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Cellulitis associated with animal bites is commonly caused by *Pasteurella multocida*, *Staphylococcus intermedius* and *Capnocytophaga canimorsus* (formerly DF-2) and is discussed separately in this module under 'Pasteurellosis, etc.'
• Human bites contain a variety of anaerobic organisms (*Fusobacterium*, *Bacteroides*), aerobic and anaerobic streptococci, and *Eikenella corrodens*.
• *Aeromonas hydrophila* causes an aggressive form of cellulitis following minor trauma in marine environments.
• *P. aeruginosa* is the most common cause of ecthyma gangrenosum and infection following penetrating injuries to the foot.
• Gram-negative bacillary cellulitis, (including *P. aeruginosa* infection) is common among hospitalized, immunocompromised patients.

This disease is endemic or potentially endemic to all countries.

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Pyomyositis

Agent	BACTERIUM. Usually Staphylococcus aureus
Reservoir	Human
Vector	None
Vehicle	Hematogenous
Incubation Period	Variable
Diagnostic Tests	Ultrasonography or CT scan.
Typical Adult Therapy	Antibiotic directed at confirmed or suspected pathogen (usually Staphylococcus aureus); drainage
Typical Pediatric Therapy	As for adult
Clinical Hints	Pain, swelling and "woody" induration of a large muscle (usually lower limb or trunk) associated with fever and leukocytosis; often follows trauma to the involved region; lymphadenopathy uncommon; leucocytosis in most cases.
Synonyms	Tropical pyomyositis. ICD9: 040.81 ICD10: M60.0

Clinical

The initiating lesion may be overt blunt or penetrating trauma; however, some cases may represent complications of viral or parasitic myositis. [1]
• An increasing percentage of reported patients have been HIV-positive. [2]
• 20 to 50% of patients with pyomyositis recall recent blunt trauma or vigorous exercise involving the area of infection; and most infections involve a single muscle or muscle group.
• Rare cases of pyomyositis have been associated with spinal epidural abscess [3] or Lemmiere's syndrome [4]
• The major muscles of the lower extremities and trunk muscles are most often infected [5] ; however, virtually any muscle can be involved. [6-11]

Onset is often subacute with fever, swelling with or without erythema, mild pain and minimal tenderness. [12]
• The involved area is indurated or has a wooden consistency.
• 10 to 21 or more days later, the patient complains of fever, with muscle tenderness and swelling.
• Overlying skin is intact and warm, usually without erythema.
• There is no regional lymphadenitis.
• At this point, pus can be aspirated from the involved muscle.
• Eventually, manifestations of sepsis appear, with local erythema, tenderness and fluctuance. [13]
• Additional symptoms may reflect compression of contiguous structures. [14] [15]
• Septicemia, ARDS and rapidly progressive or fatal infections are also encountered. [16] [17]

Leukocytosis is present.
• Eosinophilia suggests a diagnosis of 'tropical myositis' but is thought to represent the presence of concurrent parasitic infection.

The clinical features of pyomyositis may mimic those of leptospirosis. [18] \

This disease is endemic or potentially endemic to all countries.

Pyomyositis in Brazil

6 cases of pyomyositis were treated in a hospital in Sao Paulo during 1985 to 1992. [19]

82 cases (2 fatal) of pyomyositis were treated in an Amazonian hospital during 2002 to 2006. [20]

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Pythiosis

Agent	PROTOCTISTA. Oomycota, Peronosporales: Pythium insidiosum
Reservoir	Horse Cattle Dog Cat Human Aquatic plants
Vector	None
Vehicle	Direct inoculation Trauma
Incubation Period	Unknown
Diagnostic Tests	Biopsy, Culture in fungal media. Nucleic acid amplification.
Typical Adult Therapy	Excision. Amphotericin B has been used, but not proven. Minocycline; Tigecycline; and combinations of Terbinafine + caspofungin or an azole are effective in-vitro. Immune therapy (injection of Pythium antigens) has been successful in animal models.
Typical Pediatric Therapy	As for adult
Clinical Hints	Gradual onset of soft tissue infection and arterial necrosis or obstruction, usually in the lower extremities in tropical or semitropical climate; rarely destructive keratitis. Most common in patients with thalassemia or aplastic anemia.
Synonyms	Bursattee, Equine phycomycosis, Florida horse leech, Hyphomycosis destruens, Kunkers, Pythium insidiosum, Swamp cancer. ICD9: 136.8 ICD10: B99

Clinical

Infection is acquired through direct contact or trauma, and presents as granulomatous cutaneous and subcutaneous lesions.
• Patients often have underlying thalassemia. [1]
• Vascular pythiosis is characterized by ascending purulent infection and thrombosis of major arteries, usually of the lower extremities. [2] [3]
• If a major vessel is involved, the patient usually dies within weeks. [4]
• Symptoms include intermittent claudication, resting calf pain and other signs of arterial insufficiency.
• Fever and weight loss are not described.

Ocular disease presents as ulcerative keratitis which may progress to endophthalmitis and require enucleation. [5-7]

Pythium insidiosum is not susceptible to antifungal agents, and excision is the only proven form of therapy.
• Saturated solutions of potassium iodide have been recommended for localized skin infections.
• Immune therapy consisting of injection of *Pythium* antigen into infected patients has been successful in animal models, and has been used for human disease in Thailand.

This disease is endemic or potentially endemic to 5 countries.

Pythiosis in Brazil

The first case of human pythiosis in Latin America was reported in Brazil in 2002 - nonfatal infection of a leg, in Sao Paulo. [8]

A series of 27 clinical isolates was reported in Santa Maria (2007 publication) [9]

Pythiosis has been reported in Brazilian equines [10] , calves [11] , dogs [12] , rabbits [13] and sheep. [14] [15]
- The equine pathogen appears to be identical to that which infects humans. [16]

Prevalence surveys:
8.1% of sheep undergoing necropsy (rhinofacial infection, 2013 publication) [17]

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Q-fever

Agent	BACTERIUM. Coxiella burnetii Intracellular organism related to Rickettsiae
Reservoir	Cattle Sheep Goat Bird Fish Rodent Rabbit Tick Bandicoot Marsupial Dog Cat
Vector	None
Vehicle	Air Dust Infected secretions Dairy products
Incubation Period	18d - 21d (range 4d - 40d)
Diagnostic Tests	Serology. Culture possible in specialized laboratories. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg BID X 2w OR Fluoroquinolone Add Hydroxychloroquine 600 mg per day if endocarditis
Typical Pediatric Therapy	Age < 8 years: Erythromycin 10 mg/kg QID X 2 weeks Age >= 8 years: Doxycycline 100 mg BID X 2 weeks
Vaccine	Q fever
Clinical Hints	Headache, myalgia, cough and hepatic dysfunction; hepatosplenomegaly, 'F.U.O.' and endocarditis encountered; proximity to farming or animals during 2 to 4 weeks preceding illness; most infections resolve in 1 to 2 weeks; case-fatality rate = 1.5%.
Synonyms	Balkan grippe, Coxiella burnetii, Febbre australiana, Febre Q, Nine Mile fever, Q-Fieber, Q-koorts, Query fever, Red River fever. ICD9: 083.0 ICD10: A78

Clinical

The typical clinical presentation of Q-fever (pneumonia vs. hepatitis) seems to vary from region to region. [1] [2]

Q-fever is often asymptomatic or mistaken for an acute viral illness.
- Q-fever may be mistaken for Legionnaires' disease [3]
- After an incubation period of 2 to 3 weeks, the patients develops fever, headache, and myalgias. [4]
- Cough is present in 25% to 70%, and hepatosplenomegaly in 30% to 50%.
- An evanescent rash may appear in 5% of cases.
- The blood CRP is elevated; however leukocytosis is present in only 20% of cases. [5]
- Acute thrombocytosis may also be encountered. [6]
- False-positive tests toward a variety of non-related agents and conditions may be encountered: anti-nuclear antibody (ANA), smooth muscle antibody, rheumatoid factor, Epstein-Barr Virus, Cytomegalovirus, Mycoplasma pneumoniae, Parvovirus, *Bordetella pertussis*, *Rickettsia conorii* and *Rickettsia typhi*. [7]

The frequency of pneumonitis is highly variable (10% to 60%) [8] [9] ; and clinical and radiological features are non-specific. [10-12]

- Neurological complications may include encephalitis, brachial plexopathy [13] , Guillain-Barre syndrome [14] , status epilepticus and pseudotumor cerebri [15]
- Several cases of Q-fever uveitis have been reported. [16] In one case, a patient developed anterior uveitis accompanied by exudative bilateral inferior retinal detachment and optic disk edema. [17]

Occasionally, the illness may be prolonged, with severe pneumonitis [18] [19] and hepatic involvement. [20-22]
- Independent risk factors for development of chronic Q fever include valvular surgery, vascular prosthesis, aneurysm, renal insufficiency, and older age. [23]
- Chronic fatigue is common following Q-fever, and in some cases may actually represent persistent infection. [24-28]
- Although the acute disease is usually self-limited, Q-fever endocarditis may occasionally develop 3 to 20 years following the acute infection and is often fatal. [29] [30]
- Pericarditis [31-34] , myocarditis [35] [36] , aortitis [37] , optic neuritis [38] , uveitis [39-41] , disseminated intravascular coagulation [42] , hemophagocytic syndrome [43-45] , bleeding phenomena (melena, epistaxis, petechiae) [46] , autoimmune hemolytic anemia [47] , osteomyelitis [48] [49] , recurrent subcutaneous abscesses and nodules [50] , spontaneous abortion [51] [52] , prosthetic joint infection [53] , cholecystitis [54] and tubulointerstitial nephritis [55] have been reported as complications of Q-

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fever. [56-58]
• Over 80% of patients with Q-fever endocarditis have a history of underlying valvular disease.
• Additional vascular complications of Q-fever include aneurysm rupture, aorto-enteric fistulae and lower-limb embolisation. [59] [60]
• Q fever may mimic Kawasaki disease [61] , lupus erythematosus [62] or Crimean-Congo hemorrhagic fever. [63]

This disease is endemic or potentially endemic to all countries.

Q-fever in Brazil

Sporadic cases of Q-fever are reported. [64-66]

Seroprevalence surveys:

3.9% of healthy persons in Minas Gerais (2005 publication) [67]
40% of abattoir workers with exposure to hides, dust and corrals (Belo Horizonte, 1975 publication) [68]
3.2% of HIV-positive males and 6.3% of HIV-positive females in Jacarepagua, Rio de Janeiro (2009 publication)
31.8% of cattle in Caixas (1953 to 1954) [69]
30% of free-living jaguars (*Panthera onca*) in Pantanal (2011 publication) [70]

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Rabies

Agent	VIRUS - RNA. Rhabdoviridae, Mononegavirales, Lyssavirus: Rabies virus. Other human Lyssaviruses = Mokola, Duvenhage, European Bat (EBL)
Reservoir	Dog Fox Skunk Jackal Wolf Cat Raccoon Mongoose Bat Rarely rodent or Rabbit
Vector	None
Vehicle	Saliva Bite Transplants Air (bat aerosol)
Incubation Period	1m - 3m (range 4d to 19 years !)
Diagnostic Tests	Viral culture & direct immunofluorescence of saliva, CSF, corneal smears. Serology. Nucleic acid amplification.
Typical Adult Therapy	Strict isolation; supportive. The Milwaukee protocol (prolonged deep sedation and support) has been successful in some cases. See Vaccines module for pre- and post-exposure schedules
Typical Pediatric Therapy	As for adult
Vaccines	Rabies Rabies immune globulin
Clinical Hints	Follows animal bite (rarely lick) - often after months: agitation, confusion, seizures, painful spasms of respiratory muscles, progressive paralysis, coma and death; case-fatality rate > 99%.
Synonyms	Aravan, Australian bat lyssavirus, Ballina, BBLV, Bokeloh bat lyssavirus, Duvenhage, EBL, European bat Lyssavirus, Hondsdolheid, Hydrophobia, Ikoma lyssavirus, Irkut, Khujand, Lyssa, Mokola, Pteropus lyssavirus, Rabia, Rage, Raiva, Saint Hubert's disease, Shimoni bat virus, Tollwut, West Caucasian bat, Wutkrankheit. ICD9: 071 ICD10: A82

Clinical

WHO Case definition for surveillance:
• An acute neurological syndrome (encephalitis) dominated by forms of hyperactivity (furious rabies) or paralytic syndromes (dumb rabies) that progresses towards coma and death, usually by respiratory failure, within 7 to 10 days after the first symptom if no intensive care is instituted.
• Bites or scratches from a suspected animal can usually be traced back in the patient medical history.
• The incubation period may vary from days to years [1] but usually falls between 30 and 90 days.
Laboratory criteria for diagnosis
One or more of the following
• Detection of rabies viral antigens by direct fluorescent antibody (FA) in clinical specimens, preferably brain tissue (collected post mortem)
• Detection by FA on skin or corneal smear (collected ante mortem)
• FA positive after inoculation of brain tissue, saliva or CSF in cell culture, in mice or in suckling mice
• Detectable rabies-neutralizing antibody titer in the CSF of an unvaccinated person
• Identification of viral antigens by PCR on fixed tissue collected post mortem or in a clinical specimen (brain tissue or skin, cornea or saliva)
• Isolation of rabies virus from clinical specimens and confirmation of rabies viral antigens by direct fluorescent antibody testing
Case classification
Rabies:
• Suspected: A case that is compatible with the clinical description.
• Probable: A suspected case plus history of contact with suspected rabid animal.
• Confirmed: A suspected case that is laboratory-confirmed.
Rabies exposure:
• Possibly exposed: A person who had close contact (usually a bite or scratch) with a rabies-susceptible animal in (or originating from) a rabies-infected area.
• Exposed: A person who had a close contact (usually a bite or scratch) with a laboratory-confirmed rabid animal.

The initial symptoms of rabies are often limited to low grade fever and pain or paresthesia at the site of inoculation.
• Progressive encephalitis then ensues. [2]
• "Furious rabies" is characterized by hyperactivity, fluctuating level of consciousness, aerophobia and hydrophobia.
• Bizarre behavior and lack of focal neurological signs are typical.

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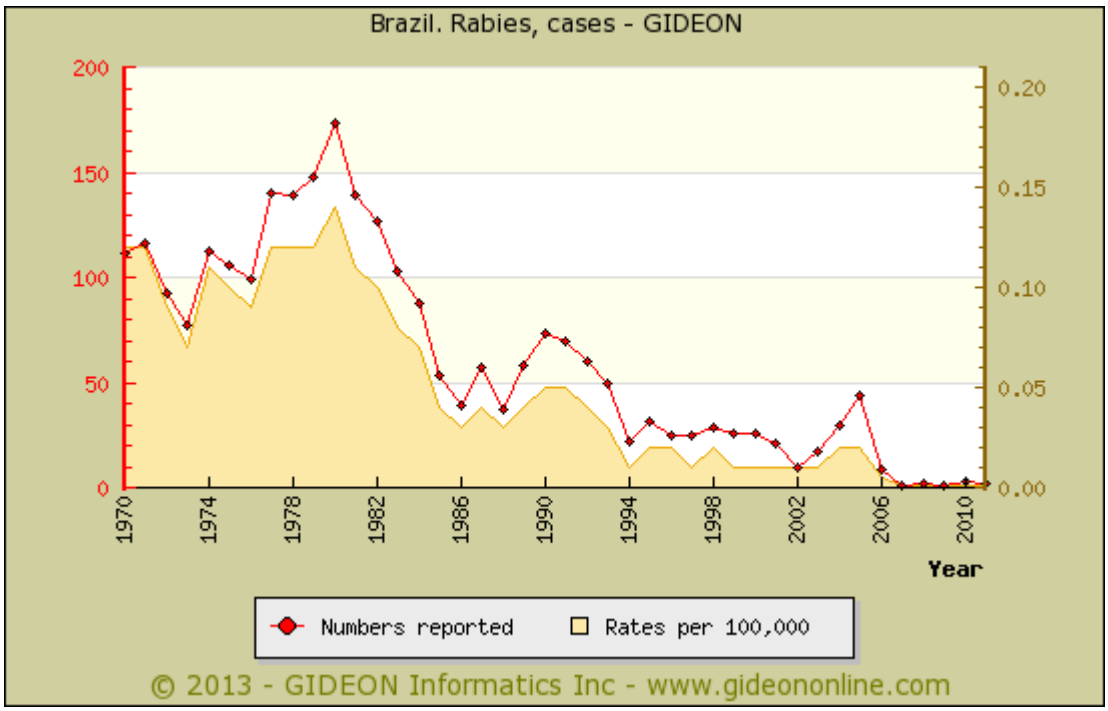
• Hydrophobia may manifest as 'jerky' inspiratory spasms progressing to opisthotonus, generalized seizures or cardiorespiratory arrest.
• Similar reactions may be elicited by fanning the patient ("aerophobia).
• Paralytic ("dumb") rabies is characterized by progressive flaccid paralysis, with fasciculation and pain in the affected muscles.
• Minor sensory disturbances may be present. Such patients may survive for as long as one month, ultimately dying of bulbar and respiratory paralysis.
• Rare instances of survival have been documented. [3-16]
• In Africa, rabies is often mis-diagnosed as cerebral malaria.

This disease is endemic or potentially endemic to 151 countries.

Rabies in Brazil



Graph: Brazil. Rabies, cases

Notes:
1. Rates of 0.05 to 0.1 per 100,000 per year are reported.
2. 1,310 cases of human rabies were reported during 1980 to 2000 (including 59 in Rio de Janeiro and 59 in Sao Paulo); 743 during 1986 to 2004. [17]
3. The average incidence was 100 per year during 1970 to 1979; 124.0 per year during 1980 to 1984; and 41.4 per year during 1985 to 1990.
4. 15% of rabies cases reported during 1987 to 1996 were acquired from bats.
5. Eight cases of human rabies reported in Ceara during 1991 to 1998 were acquired from marmosets (*Callithrix jacchus*). [18]

Individual years:

1988 - 61.1% of cases were reported from rural areas. Included two cases from cats.

1990 - 70% of human cases were reported from the northeast, and 15.1% were acquired from bats.

2008 - Included one case of survival from rabies in Pernambuco. [19-22]

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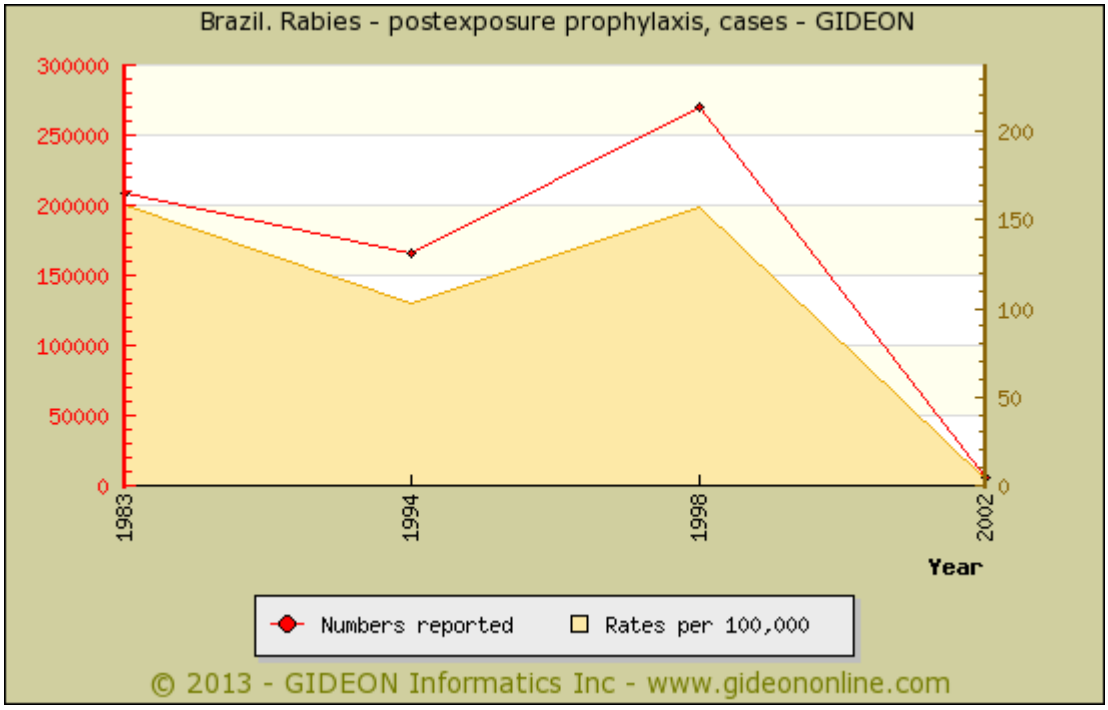
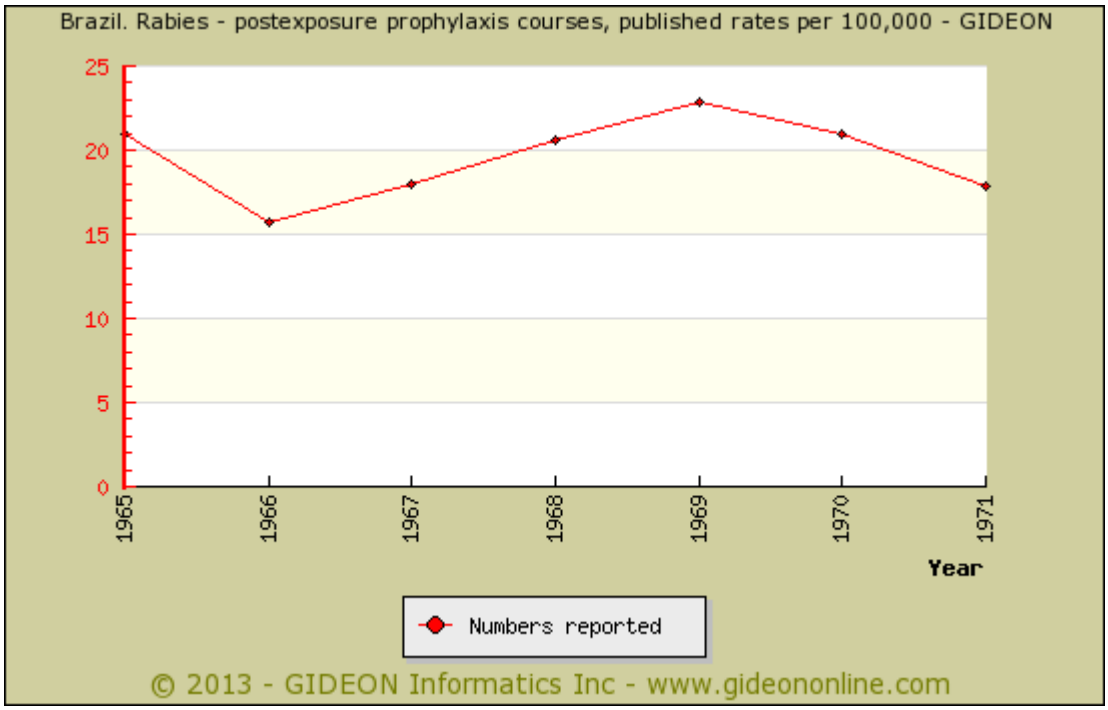


Graph: Brazil. Rabies - postexposure prophylaxis, cases

Notes:
1. 2,223 courses of post-exposure prophylaxis were administered in Porto Alegre during July to December 2006. [23]



Graph: Brazil. Rabies - postexposure prophylaxis courses, published rates per 100,000

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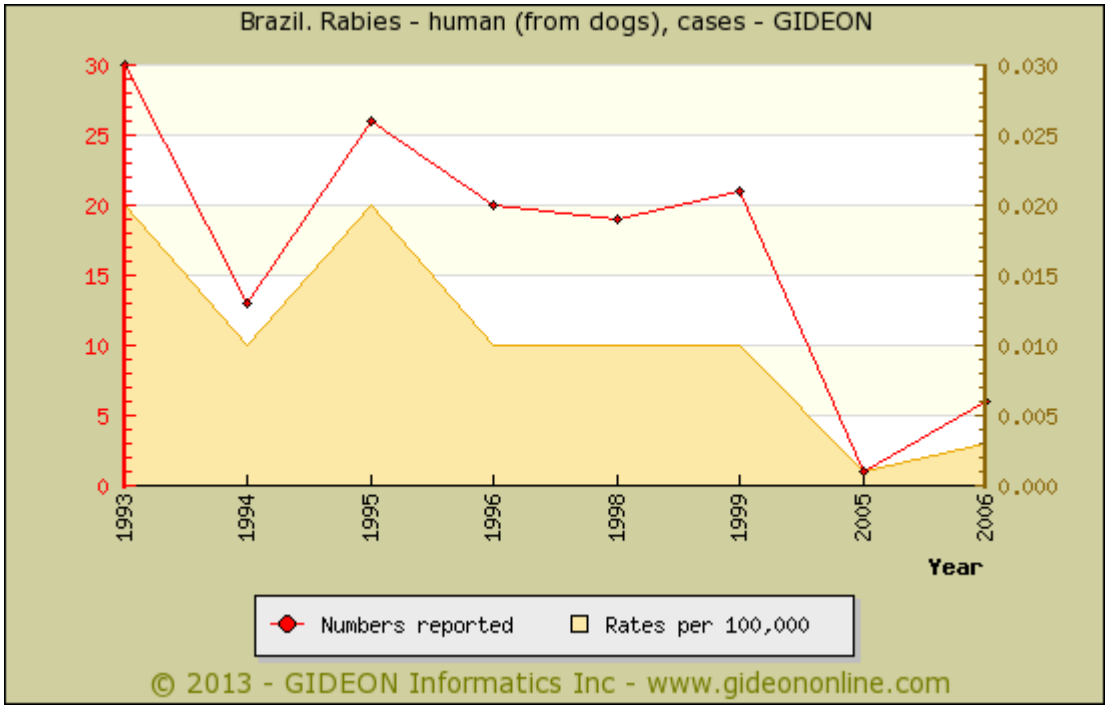
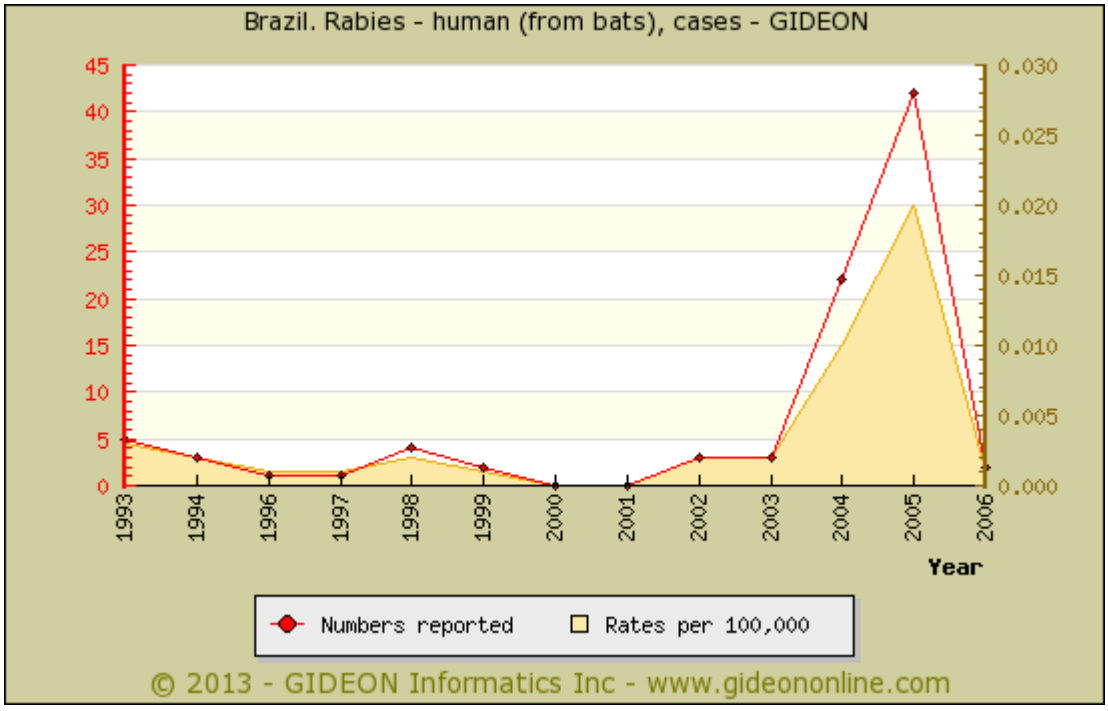


Graph: Brazil. Rabies - human (from dogs), cases



Graph: Brazil. Rabies - human (from bats), cases

Notes:
1. 76 cases of human rabies acquired from vampire bats (*Desmodus rotundus*) were reported during 1980 to 1996 - including two clusters (3 and 2 cases) in Bahia State during 1991 to 1992. [24]

2. 80 cases of bat-associated rabies cases were reported during 1996 to 2006. [25]

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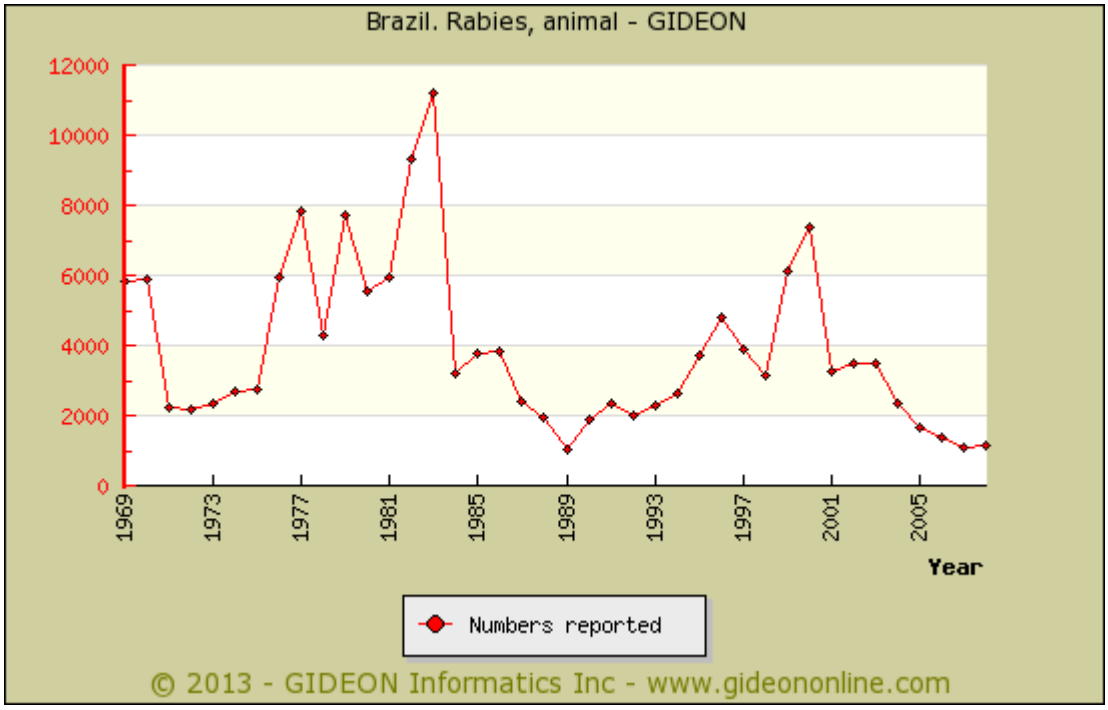

Graph: Brazil. Rabies, animal

Notes:
1. During 1990 to 2005, 173 cases were reported among crab-eating foxes (*Cerdocyon thous*), 25 common marmosets (*Callithrix jacchus*) and 6 crab-eating raccoons (*Procyon cancrivorous*)
2. 4.9% of animals in Aracatuba were found to be rabid during 1993 to 2007. [26]
Individual years:
1970 - Included 647 farm animals.
1994 - 49% ruminants.
1998 - Included 1,103 ruminants and 118 cats.

Neutralizing antibodies have been demonstrated in a variety of wild animals in Sao Paulo City - primates, marsupials, carnivores, edentates and rodents.
- Rabies virus appears to circulate among dogs, vampire bats, cattle, crab-eating foxes (*Cerdocyon thous*) and humans in the Ceara region. [27]

Seroprevalence surveys:

12.3% of free-ranging wild carnivores captured in the Pantanal and Cerrado (2000 to 2006) [28]

11% of free-ranging capuchin monkeys (*Cebus apella nigritus*) in a fragmented, environmentally protected, rural area of southeastern Brazil (2012 publication) [29]

0.8% of bats in Sao Paulo State (1988 to 2003) [30]

1% of nonhematophagous bats in the western region of Sao Paulo were found to be seropositive toward rabies virus (2006 to 2008) [31]

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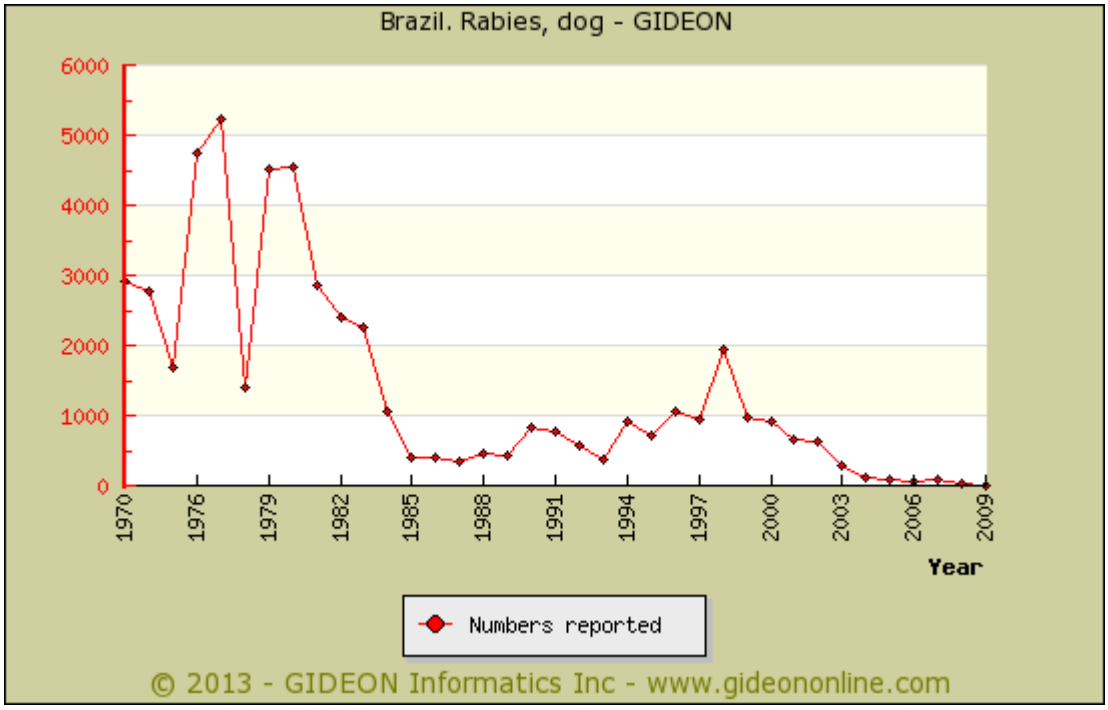

Graph: Brazil. Rabies, dog

Notes:
1. There is evidence that dog rabies arrived to Brazil during the late 19th or early 20th centuries. [32]

Bats and rabies:

- Rabies is associated with vampire bats [33] and frugivorous bat (*Artibeus* spp.). [34]
- 721 bat attacks on humans were reported in the Rio Negro region during 2004 to 2006. [35]
- During 1996 to 2009, rabies was identified in 41 bat species, belong to 25 genera and three families: Phyllostomidae, Vespertilionidaea and Molossidae. [36]
- 1.9% of bats from Southeastern Brazil were found to be infected with rabies virus; the 10 species infected were: *Artibeus lituratus*, *Artibeus* spp., *Myotis nigricans*, *Myotis* spp., *Eptesicus* spp., *Lasiurus ega*, *Lasiurus cinereus*, *Nyctinomops laticaudatus*, *Tadarida brasiliensis*, *Histiotus velatus*, *Molossus rufus*, *Eumops* spp. and *Desmodus rotundus*. (State of Sao Paulo, 2002 to 2003) [37]
- 0.2% of bats in northwestern Sao Paulo State were found to carry rabies in 1997; 1.6% in 2001 - with highest rates in *Artibeus lituratus*. [38]
- Rabies has been detected in bats (*Nyctinomops laticaudatus*) in Rio de Janeiro (2007 publication) [39] ; a vampire bat (*Desmodus rotundus*) in the Ubatuba municipality (2007 publication) [40] ; various bats (*Eumops perotis* [41] , *Molossus molossus*, *Molossops neglectus* and *Myotis riparius*) in Sao Paulo [42] ; *Molossus molossus* in Pernambuco [43] ; a frugivorous bat (*Artibeus lituratus*) in Minas Gerais [44] ; *Eptesicus furinalis* bats in Jundiai City, Sao Paulo (2011 publication) [45] ; and a hairy-legged vampire bat (*Diphylla ecaudata*) [46]
- Rabies was confirmed in eleven (5.5%) of non-hematophagous bats submitted for testing from Rio de Janeiro during 2001 to 2011. [47]
- Seropositive bat species in Sao Paulo have included *Glossophaga soricina*, *Histiotus velatus*, *Desmodus rotundus*, *Artibeus lituratus*, *Nyctinomops macrotis*, *Tadarida brasiliensis*, *Carollia perspicillata*, *Eumops auripendulus*, *Nyctinomops laticaudatus*, *Sturnira lilium* and *Eumops perotis* (1988 to 2003). [48]
- During 2006 to 2008, 1% of nonhemotophagous bats in the western region of Sao Paulo were found to be seropositive toward rabies virus. [49]

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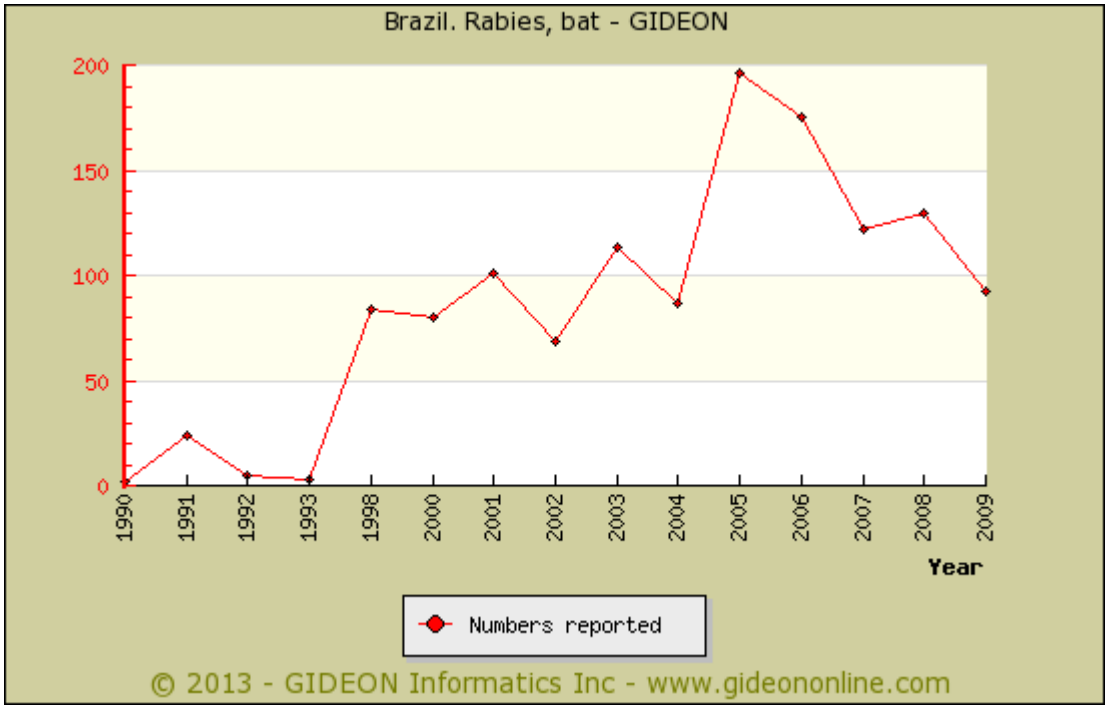

Graph: Brazil. Rabies, bat

Notable outbreaks:

1960 - An outbreak (18 cases) of rabies in Fortaleza was caused by contamination of rabies vaccine by an active virus. [50] [51]

1995 to 1996 - An outbreak (78 cases) of rabies was reported among dogs and cats in Ribeirao Preto, Sao Paulo. [52-55]

2004 to 2005 - Outbreaks (21 cases) of human rabies from vampire bats were reported in Portel (15 cases) and Viseu (6 cases), Para State. [56-59]

2005 - An outbreak (16 cases) of human rabies from bats was reported in Turiassu township (Maranhao). [60] [61]

2011 to 2012 - An outbreak (47 cases) of cattle rabies was reported in Parana. [62] [63]

2012 - An outbreak (1,400 cases estimated) of bovine rabies was reported in Rio Grande do Sul. [64]

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Rat bite fever - spirillary

Agent	BACTERIUM. Spirillum minus An aerobic gram-negative spirochete
Reservoir	Rat Mouse Cat
Vector	None
Vehicle	Bite
Incubation Period	7d - 21d (range 5d - 40d)
Diagnostic Tests	Dark-field exam of wound. Animal inoculation.
Typical Adult Therapy	Amoxicillin/clavulanate 875/125 mg PO BID X 7d. OR Procaine Penicillin G 600,000u IM q12h X 7d. OR Doxycycline 200 mg BID X 7d
Typical Pediatric Therapy	Amoxicillin/clavulanate 10 mg/kg PO BID X 7d OR Procaine Penicillin G 25,000u/kg IM q12h X 7d
Clinical Hints	Lymphadenopathy, myalgia, maculopapular rash and recurrent fever beginning 1 to 3 weeks after rat bite; infection resolves after 3 to 6 days; case-fatality rate = 6%.
Synonyms	Sodoku, Spirillosis, Spirillum minor, Spirillum minus. ICD9: 026.0 ICD10: A25.0

Clinical

Most patients present with a recent rat bite wound, which may later form an ulcer with local swelling, pain and skin changes.
- Regional lymphatics and lymph nodes are enlarged and tender.
- Fever rises to as high as 40 C, with accompanying rigors.
- After 3 days, fever ends in 'crisis,' followed by a quiescent interval of 5 to 10 days.
- One or more relapses follow, and are associated with a purple papular exanthem on the chest and arms.
- Additional findings include generalized hyperreflexia, arthralgia, myalgia and hyperesthesia.
- The fatality rate without treatment is 10%.

Features which may distinguish spirillary [S] from streptobacillary [B] rat bite fever include the following: [1] [2]

incubation	-	S up to 30 days
	-	B up to 10 days
bite wound	-	S may produce a chancre
	-	B heals promptly
relapses	-	S regular
	-	B intermittent
rash	-	S generalized macular
	-	B macular, pustular or petechial
arthritis	-	S rare
	-	B common [3]

This disease is endemic or potentially endemic to all countries.

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Rat bite fever - streptobacillary

Agent	BACTERIUM. Streptobacillus moniliformis A facultative gram-negative bacillus
Reservoir	Rat Squirrel Weasel Turkey
Vector	None
Vehicle	Infected secretions Bite Dairy products
Incubation Period	3d - 10d (range 1d - 22d)
Diagnostic Tests	Culture of blood or joint fluid. Nucleic acid amplification.
Typical Adult Therapy	Amoxicillin/clavulanate 875/125 mg PO BID X 7d. OR Doxycycline 100 mg PO BID X 7d
Typical Pediatric Therapy	Amoxicillin/clavulanate 10 mg/kg TID X 7d. OR (if age>8 years) Doxycycline 2 mg/kg PO BID X 7 days (maximum 200 mg/day)
Clinical Hints	Headache, myalgia, maculopapular rash and arthralgia or arthritis; history of a rat bite during the preceding 1 to 3 weeks in most cases; case-fatality rate = 10%.
Synonyms	Haverhill fever, Streptobacillosis, Streptobacillus moniliformis. ICD9: 026.1 ICD10: A25.1

Clinical

Most patients present with a recent rat bite wound, which may later form an ulcer with local swelling, pain and skin changes. [1]

• Symptoms include fever, prostration, marked myalgia and muscle tenderness, headache and a generalized morbilliform rash • most marked on the hands and feet. [2]
• Generalized lymphadenopathy is present, and migratory arthropathy is often present.
• Fever resides in 5 to 10 days, but may relapse repeatedly over a period of weeks to months.

One or more relapses follow, and are associated with a purple papular exanthem on the chest and arms.
• Additional findings include generalized hyperreflexia, migratory polyarthralgia (over 50% of cases), myalgia and hyperesthesia.
• Arthritis affects more than one joint in 83.3% of patients, involving the knee in most. [3]
• Rare instances of endocarditis [4] [5] , psoas abscess, epidural abscess [6] and spondylodiscitis have been reported. [7]

he fatality rate without treatment is 10%, and results from endocarditis or multiple visceral abscesses.

Features which may distinguish spirillary [S] from streptobacillary [B] rat bite fever include the following: [8] [9]

incubation	-	S up to 30 days
	-	B up to 10 days
bite wound	-	S may produce a chancre
	-	B heals promptly
relapses	-	S regular
	-	B intermittent
rash	-	S generalized macular
	-	B macular, pustular or petechial
arthritis	-	S rare
	-	B common [10]

This disease is endemic or potentially endemic to all countries.

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Relapsing fever

Agent	BACTERIUM. Borrelia spp. A microaerophilic spirochete
Reservoir	Human Tick Rodent
Vector	Tick (Ornithodoros), louse (Pediculus)
Vehicle	Blood products
Incubation Period	7d - 8d (range 2d - 18d)
Diagnostic Tests	Examination of blood smears (thick and thin smears). Some species (B. hermsii) may grow in BSK II medium.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 7d. OR Erythromycin 500 mg QID X 7d
Typical Pediatric Therapy	Chloramphenicol 12.5 mg/kg PO QID X 7d. OR Erythromycin 10 mg/kg QID X 7d
Clinical Hints	Headache, myalgia, hepatosplenomegaly, rash and relapsing illness; louse-borne (vs. tick borne) characterized by higher case fatality rate, fewer relapses and higher incidence of hepatosplenomegaly, jaundice and neurological complications.
Synonyms	Bilious typhoid, Borrelia microti, Borrelia miyamotoi, Borrelia turicatae, Borreliosis, Famine fever, Febbre recidiva, Febbre ricorrente, Febris recurrens, Fiebre recurrente, Lauseruckfallfieber, Mianeh fever, Ruckfall fieber, Tilbakefallsfever, Vagabond fever, Yellow famine fever, Yellow plague. ICD9: 087.9,087.0,087.1 ICD10: A68

Clinical

The clinical manifestations of louse-borne and tick-borne [1] relapsing fevers are similar. [2] [3]
• Louse-borne disease is characterized by a longer incubation period, longer febrile periods and afebrile intervals, and fewer relapses.
• Both types have an acute onset of high fever with rigors, headache, myalgia, arthralgia, photophobia and cough.
• In Africa, tickborne relapsing fever is often mis-diagnosed as malaria. [4]

Physical findings often include conjunctivitis, petechiae, and abdominal tenderness with hepatomegaly and splenomegaly.
• Nuchal rigidity, pulmonary rales, lymphadenopathy, jaundice and ARDS [5] are occasionally encountered.
• Hemorrhagic phenomena are common but rarely severe.
• Iritis and iridocyclitis may lead to permanent impairment of vision. Uveitis is also described. [6]
• A petechial, macular, or papular rash over the trunk may be noted toward the end of the illness.
• As many as 30% of patients develop neurological findings such as coma, cranial nerve palsies, hemiplegia, meningitis, and seizures. [7]
• Rare instances of acute respiratory distress syndrome [8] [9] and dermal eschar [10] have been associated with tick-borne relapsing fever.
• Deaths are ascribed to myocarditis with associated arrhythmias, cerebral hemorrhage or hepatic failure.
• "Tropical thrombophlebitis" has been associated with outbreaks of relapsing fever in South Africa. [11]

Borrelia miyamotoi infection is characterized by fever, headache, myalgia and multiple relapses. [12]
• *B. miyamotoi* meningoencephalitis was reported in an immunocompromised patient. [13]

This disease is endemic or potentially endemic to 118 countries.

Relapsing fever in Brazil

Borrelia mazzottii and *B. venezuelensis* are identified as causes of tick-borne disease.

Louse-borne infection may be present as well.

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Respiratory syncytial virus infection

Agent	VIRUS - RNA. Paramyxoviridae, Pneumovirinae: Human respiratory syncytial virus
Reservoir	Human
Vector	None
Vehicle	Droplet Infected secretions (hands)
Incubation Period	2d - 8d
Diagnostic Tests	Viral culture or DFA (nasal and other respiratory secretions). Serology. Nucleic acid amplification.
Typical Adult Therapy	Ribavirin aerosol 20 mg/ml for 12h/d X 3 to 5d [severe infections]. Effectiveness not proven
Typical Pediatric Therapy	As for adult
Vaccine	RSV immune globulin
Clinical Hints	Rhinorrhea, cough, wheezing, bronchiolitis and respiratory distress; encountered primarily in infancy.
Synonyms	Chimpanzee coryza agent, Respiratory syncytial virus, RSV. ICD9: 079.6,480.1 ICD10: B97.4,J12.1

Clinical

RSV infections are manifested as:
• lower respiratory tract disease (pneumonia, bronchiolitis, tracheobronchitis)
• upper respiratory tract illness, often accompanied by fever and otitis media. [1]

Asymptomatic infection is rare.
• Pneumonia or bronchiolitis occurs in 30% to 71% of patients (89% among closed populations of infants).
• Croup accounts for only 5% to 10% of cases.
• Wheezing [2-4] , rhonchi, rales, and pulmonary infiltrates are encountered with bronchiolitis as well as pneumonia. [5]
• Bronchiolitis is characterized by wheezing and hyperaeration of the lung.
• RSV infection in adults is usually mild; however severe disease may develop. [6-8]

Lower respiratory tract infection is heralded by nasal congestion and often pharyngitis.
• Fever occurs in young children, with temperatures ranging from 38 to 40C.
• Fever is present for 2 to 4 days; however, the extent and duration of the fever does not correlate with the severity of the disease.
• Fever is frequently absent at the time of admission to the hospital.
• Cough is often a predominant sign.
• The cough may be paroxysmal and associated with vomiting, but without the "whoop" typical of pertussis.
• Laryngitis and hoarseness are not common.

Dyspnea, increased respiratory rate, and retractions of the intercostal muscles are common.
• In bronchiolitis, expiration is prolonged, and the respiratory rate may be remarkably elevated. [9]
• Intercostal retractions are also prominent in bronchiolitis.
• On auscultation, the infant may have crackles and wheezing, which may be present intermittently and may fluctuate in intensity.
• Cyanosis is rare, despite hypoxemia. In most infants, the duration of illness is 7 to 21 days, and hospitalization, if required, averages 3 to 7 days.
• Thrombocytosis is common among children hospitalized with RSV bronchiolitis. [10]
• The severity and / or duration of RSV bronchiolitis is exacerbated by concomitant human metapneumovirus infection. [11-14]
• RSV infection accounts for approximately 5% of bronchiolitis obliterans in children (Beijing, 2001 to 2007) [15]
• Infection in premature infants may result in long term effects on airway function. [16-18]

Otitis media is a common complication of RSV infection in young children. [19-22]
• Viral meningitis [23] , encephalopathy and seizures are also encountered. [24-26]
• Repeated or secondary infections occurring after the first 3 years of life are most commonly manifested as an upper respiratory tract illness or tracheobronchitis.

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• Young adults may present with flu-like illness, pneumonia, chronic cough suggestive of tracheobronchitis or bronchitis, and occasionally with otitis. [27]
• Infection among the elderly is often nosocomially acquired, and may result in pneumonia in 5% to 50% of the cases, with a fatal outcome in up to 20%.
• Additional extrapulmonary manifestations of RSV infection include myocarditis [28] , supraventricular tachycardia, ventricular tachycardias, pericardial effusion [29] , seizures, focal neurological abnormalities, hyponatremia and hepatitis [30]

Signs and symptoms of Human Metapneumovirus (hMPV) infection are similar to those of Respiratory syncytial virus infection [31] [32] , and coinfection by these two agents may be particularly severe. [33-37] Children with hMPV infection are likely to be older than those with RSV, and more likely to present with pneumonia and less likely to present with bronchiolitis. [38]
• Clinical signs of Rhinovirus infection [39] and of Human Bocavirus infection are also similar to those of Respiratory syncytial virus infection; however, hypoxia, and neutrophilia may be more common in Human Bocavirus infection. [40]
• Superinfection of RSV by *Staphylococcus aureus* [41] , *Bordetella pertussis* [42] and other bacteria is not unusual. [43]

This disease is endemic or potentially endemic to all countries.

Respiratory syncytial virus infection in Brazil

Rates of RSV infection are highest during the winter (June through August), with an earlier peak in Rio de Janeiro (southeast) than in the south. [44] [45]

The incidence of RSV infection among children below age 5 years is 14 per 1,000 child years (Rio de Janeiro, 1987 to 1989).

Prevalence surveys:

38% of respiratory infections in the age group < 5 years (1987 to 1989)
17.5% of infants hospitalized for acute lower respiratory tract infection (Sao Paulo, 2004) [46]
31.9% of infants admitted to a hospital in Campinas with acute respiratory infection (2009 publication) [47]
26.4% of children below age 5 years with acute respiratory disease in Uberlandia (2001 to 2004) [48]
37% of children below age 5 years with acute respiratory infection, presenting to an emergency service in northeastern Brazil. (2011 publication) [49]
1.5% of children with acute respiratory illness (Rio de Janeiro and Teresopolis, 2006 to 2007) [50]
41.8% of childhood hospitalizations for lower respiratory tract infection in Sao Paulo (1996 to 1996)
29.3% of children ages 0 to 6 years, hospitalized with acute lower respiratory infection in Sao Jose do Rio Preto, Sao Paulo (2004 to 2006) [51]
47.1% of childhood hospitalizations for lower respiratory tract infection in Rio de Janeiro. [52]
23.1% of children ages <= 3 years hospitalized with community-acquired pneumonia in Belem city (2006 to 2007) [53]
54% of viruses associated with lower respiratory tract infection among hospitalized children below age 3 years (2013 publication) [54]
24.1% of children (age below age 5 years) in Sao Paulo with lower respiratory tract infection (2003) [55]
60.9% of infants hospitalized with respiratory infection. (Sao Paulo, 2007 publication) [56]
63.6% of infants admitted to intensive care unit with acute bronchiolitis (2011 publication) [57]
11% of children attending emergency services (Northeast; 2009 publication)
42% of children admitted to a single institution in Sao Paulo (2009 publication) [58]
29% of respiratory pathogens from hospitalized and 7.7% from daycare children in Sao Jose do Rio Preto, Sao Paulo (2012 publication) [59]
73.03% of children below age 5 years hospitalized with respiratory infection (2005 to 2006) [60]
45.1% of children (age below 5 years) with acute respiratory infection in Butanta (2007 publication) [61]
15.7% of children with tonsillar diseases (2912 publication) [62]
36.7% of fatal respiratory infection in children (1985 to 2005) [63]
26.2% of acute respiratory infections in children below age 5 in Rio Grand do Sul (1990 to 1992). [64]
48% of acute lower respiratory infection in children below age 3, and mixed hMPV / RSV infection for an additional 7% (Aracaju, April to May 2002) [65]
3.8% of influenza-like illness with proven viral etiology among adults in Sao Paulo (2001 to 2003) [66]
31% of outpatients with influenza-like illness (2000 to 2011) [67]
3.3% of outpatient and 19.3% of hospitalized patients with respiratory infection (Curituba, 2005 publication) [68]
0% of elderly adults with acute respiratory viral infections (Botucatu, Sao Paulo, 2002 to 2003) [69]

Notable outbreaks:

1999 - An outbreak of Respiratory syncytial virus infection was reported in Salvador. [70]

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2010 - An outbreak (31 cases) was reported in a hematopoietic stem cell transplantation unit. [71]
2010 - An outbreak (10 cases, 0 fatal) was reported in a neonatal intensive care unit. [72]

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Respiratory viruses - miscellaneous

Agent	VIRUS - RNA and DNA Pneumovirinae: Human Metapneumovirus Coronaviridae: New Haven Coronavirus, HKU1 Parvovirinae: Human Bocavirus
Reservoir	Human
Vector	None
Vehicle	Droplet Infected secretions (on hands)
Incubation Period	Unknown
Diagnostic Tests	Viral culture. Serology. Nucleic acid amplification.
Typical Adult Therapy	NA
Typical Pediatric Therapy	NA
Clinical Hints	Rhinorrhea, cough, wheezing, bronchiolitis and respiratory distress; encountered primarily in infancy.
Synonyms	Acanthamoeba polyphaga mimivirus, Bat reovirus, Bocavirus, Bradford coccus, Cardiovirus, Coronavirus HKU1, Coronavirus NL63, Encephalomyocarditis Virus, HCoV-HKU1, HCoV-NL63, HK23629/07, HKU1, HRV-A, HRV-B, HRV-C, Human Bocavirus, Human Coronavirus NL63, Human CoV 229E, Human CoV OC43, Human metapneumovirus, Human rhinovirus, Kampar, Karolinska Institutet virus, KI virus, Melaka, Metapneumovirus, Mimivirus, New Haven coronavirus, Pulau, Rhinovirus, Small Anellovirus, Tioman virus, Torque tenovirus, Torquetenovirus, Washington University virus, WU polyomavirus, WU virus. ICD9: 079.89 ICD10: B34.2,J12.8

Clinical

For a comprehensive review of newer respiratory viral infections, see [1]

Human Metapneumovirus:

Signs and symptoms of Human Metapneumovirus (hMPV) infection are similar to those of Respiratory syncytial virus infection [2] [3] , and coinfection by these two agents may be relatively severe and / or prolonged. [4-9] Children with hMPV infection are likely to be older than those with RSV, and more likely to present with pneumonia and less likely to present with bronchiolitis. [10]

• Findings include either lower respiratory tract disease (pneumonia, bronchiolitis, tracheobronchitis) or upper respiratory tract illness, often accompanied by fever and otitis media. [11] [12]
• Asymptomatic infection is reported. [13] [14]
• Wheezing, rhonchi, rales, and pulmonary infiltrates are encountered with bronchiolitis, hyperaeration and pneumonia. [15] Severe and potentially-fatal infections are reported. [16]
• Apnea has been reported in newborn infants. [17]
• hMPV has been recovered from the middle ear in patients with otitis media. [18] and is associated with 6% of otitis media cases in children. [19]
• Central nervous system disease has been reported, ranging from febrile seizures to fatal encephalitis. [20-22]
• Reinfection is common. [23-25]
• Although infection in adults is usually mild or asymptomatic [26] , severe disease is reported in elderly adults with underlying disease. [27-29]

New Haven coronavirus:

New Haven coronavirus infection is characterized by fever, cough and rhinorrhea. [30] [31]
• Tachypnea, hypoxia and pulmonary infiltrates may be present.
• The agent has also been identified as a common cause for croup. [32]

Coronavirus infections:

HKU1 (HCoV-HKU1), a human coronavirus, was isolated in Hong Kong in 2005, from two adult patients with pneumonia. [33]
• An additional 6 cases in Hong Kong were characterized by gastroenteritis, fever, otitis and febrile seizures.
• Human Coronavirus OC43 infection is associated with fever, rhinitis, pharyngitis, laryngitis, otitis, bronchitis, bronchiolitis or pneumonia. [34]

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Human Bocavirus:

Human Bocavirus is a common cause of lower respiratory tract infection in children. [35] [36]

- Bocavirus infections, including cases of severe pneumonia, have also been reported in adults. [37]
- Patients are often co-infected by Respiratory syncytial virus, Adenovirus, Influenza virus, Human metapneumovirus or other pathogens. [38]
- Clinical presentation may include fever, cough, rhinorrhea, conjunctivitis, wheezing, respiratory distress, pneumonia or pleural effusion. [39]
- Rarely, severe and life-threatening infection is encountered. [40]
- Human Bocavirus infection may mimic the symptoms of pertussis [41]
- Rare instances of Human Bocavirus encephalitis have been reported. [42]
- Clinical signs are also similar to those of Respiratory syncytial virus infection; however, hypoxia, and neutrophilia may be more common in Human Bocavirus infection. [43]
- Disseminated Bocavirus infection, including diarrhea and viremia, has been reported in a stem cell transplant patient. [44]

Other viruses:

Although Rhinovirus infection is usually associated with the common cold, infection may be associated with severe lower respiratory tract infections [45] , and outbreaks of major and even fatal disease have been reported in chronic care facilities. [46-49]

Melaka virus, a bat-associated Reovirus, has been identified as a cause of fever and acute respiratory tract infection in Malaysia. [50]

Saffold Cardiovirus, a member of the Picornaviridae, has been associated with cases of upper respiratory tract infection in children. [51] [52]

- Human infection by an additional Cardiovirus, Encephalomyocarditis Virus, have been characterized by fever, headache, nausea and dyspnea. (2009 publication) [53] One such patient also experienced weight loss, arthralgia, photophobia, myalgia, chills, vomiting, and abdominal pain.

This disease is endemic or potentially endemic to all countries.

Respiratory viruses - miscellaneous in Brazil

Human metapneumovirus (hMPV) in Latin America was first reported in Brazil in 2002. [54]

Prevalence surveys:

hMPV accounted for 17% of acute lower respiratory infection in children below age 3, and mixed hMPV / RSV infection for an additional 7% (Aracaju, April to May 2002) [55]

hMPV was detected in 6.4% of children hospitalized with an acute respiratory infection (Southern Brazil, 2007 publication) [56]

hMPV was detected in 11.4% of children admitted to a single institution in Sao Paulo (2009 publication) [57]

hMPV accounted for 17.8% of children (age below age 5 years) in Sao Paulo with lower respiratory tract infection (2003) [58]

hMPV was detected in 5.6% of infants admitted to a hospital in Campinas with acute respiratory infection (2009 publication) [59]

hMPV was detected in 5.6% of nasopharyngeal aspirates collected from pediatric patients at two university hospitals (Campinas, 2004) [60]

hMPV was detected in 6.4% of hematopoietic stem cell recipients with respiratory symptoms in 2001, 4.7% in 2002 and 11.1% in 2003 (Sao Paulo) [61]

hMPV was detected in 9.2% of influenza-like illness with proven viral etiology among adults in Sao Paulo, Coronavirus OC43 4.6%, Coronavirus 229E 2.3% (2001 to 2003) [62]

hMPV was detected in 2.4% of children with acute respiratory illness; Human Bocavirus (HBoV) in 2.4%; Human Coronaviruses (HCoV-HKU1 or HCoV-NL63) in 1.5% (2006 to 2007) [63]

hMPV was detected in 12.3% of children (<5 years) with acute respiratory infection (negative for 9 common respiratory viruses) attending a public hospital in Uberlandia (2009 publication) [64]

hMPV was detected in 3.9% of respiratory samples from patients with acute respiratory infection, in Curitaba (2011 publication) [65]

hMPV was detected in 4.1% of children aged <5 years hospitalized for community-acquired pneumonia (Salvador, 2011 publication) [66]

hMPV was detected in 2.5% of hematopoietic stem cell transplant recipients with acute respiratory infection (2009 publication) [67]

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hMPV was detected in 10.6% of hematopoietic stem cell transplant recipients, and 12.1% of immunocompetent adults with severe respiratory infections (2009) [68]

hMPV was detected in 14.5% of children with lower respiratory tract infection, in Porto Alegre; and HBoV in 13.2% (2007 to 2008) [69]

hMPV was found in 2% of elderly adults with acute respiratory viral infections, Rhinovirus 28.6% and RSV 0% (Botucatu, Sao Paulo, 2002 to 2003) [70]

HBoV was found in 23% of children hospitalized for community-acquired pneumonia. (Salvador, 2011 publication) [71]

HBoV accounted for 10.5% of respiratory infections among children below age 5 years in Ribeirao Preto (2005) [72]

HBoV was found in 3.0% of patients with acute respiratory infection in Sao Paulo, in 2005; 3.0% in 2006; 3.0% in 2007 [73]

HBoV was found in 2.44% of children and 0.4% of adult bone-marrow-transplant patients with acute respiratory tract infection (Sao Paulo, 2001 to 2008) [74]

HBoV was found in 2.4% of children with acute respiratory illness, Adenovirus 0.5%, hMPV 2.4% and RSV 1.5% (Rio de Janeiro and Teresopolis, 2006 to 2007) [75]

RSV was found in 63.6% of infants admitted to intensive care unit with acute bronchiolitis, and Rhinovirus in 39% (2011 publication) [76]

RSV was found in 37% of children below age 5 years with acute respiratory infection, presenting to an emergency service in northeastern Brazil, Adenovirus 25%, Rhinovirus 19%, HBoV 19%, hMPV 10% and *Mycoplasma pneumoniae* 10%. (2011 publication) [77]

RSV accounted for 54% of viruses associated with lower respiratory tract infection among hospitalized children below age 3 years, hMPV 32% and Human rhinovirus 21% (2013 publication) [78]

RSV was identified in 31% of outpatients with influenza-like illness, Parainfluenza virus in 30% ad Adenovirus in 12% (2000 to 2011) [79]

Coronavirus HKU1 was found in 3.6% of children with respiratory infection or diarrhea (1995) [80]

Viruses were found in 97.5% of children with tonsillar diseases - adenovirus in 47.1%, human enterovirus in 40.5%, human rhinovirus in 38%, human bocavirus in 29.8%, human metapneumovirus in 17.4% and human respiratory syncytial virus in 15.7% (2912 publication) [81]

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Reye's syndrome

Agent	UNKNOWN
Reservoir	Unknown
Vector	None
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Clinical diagnosis.
Typical Adult Therapy	Electrolyte & glucose management, ? enemas, ? dialysis
Typical Pediatric Therapy	As for adult
Clinical Hints	Vomiting, lethargy, coma, seizures, hepatomegaly, hypoglycemia and elevated blood ammonia concentration; usually anicteric; follows viral infection; aspirin ingestion is often implicated.
Synonyms	Reye syndrome. ICD9: 331.81 ICD10: G93.7

Clinical

Signs and symptoms of Reye's syndrome include protracted vomiting and encephalopathy, in the absence of fever or jaundice. [1] [2]
• Hepatomegaly is present in 50% of cases.
• Twelve hours to 3 weeks following an antecedent viral illness, the patient develops vomiting and lethargy, followed by restlessness, irritability, combativeness, disorientation, delirium, tachycardia, hyperventilation, dilated pupils with sluggish response, hyperreflexia, positive Babinski sign, and appropriate response to noxious stimuli.

Diarrhea and hyperventilation are often the first signs in children below age 2 years.
• Later, obtundation, coma and decorticate rigidity are associated with inappropriate response to noxious stimuli.
• Coma deepens, and the patient is found to have fixed and dilated pupils, loss of oculovestibular reflexes and dysconjugate gaze with caloric stimulation.
• Seizures ensue, with flaccid paralysis, absent deep tendon reflexes, lack of pupillary response and respiratory arrest.

Similar disease (Reye-like syndrome) is caused by inborn errors of metabolism, hypoglycemia, hypoketonemia, elevated ammonia, and organic aciduria. [3]
• A case of encephalopathy and hepatic failure • similar to Reye's syndrome • was related to *Bacillus cereus* food poisoning. [4]

This disease is endemic or potentially endemic to all countries.

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Rheumatic fever

Agent	BACTERIUM. Streptococcus pyogenes A facultative gram-positive coccus
Reservoir	Human
Vector	None
Vehicle	Droplet
Incubation Period	1w - 5w
Diagnostic Tests	Clinical diagnosis.
Typical Adult Therapy	Supportive; salicylates
Typical Pediatric Therapy	As for adult
Clinical Hints	Migratory arthritis, fever, carditis, chorea, subcutaneous nodules, erythema marginatum and leukocytosis; follows overt pharyngitis after 1 to 5 weeks in most cases; acute attack persists for approximately 3 months.
Synonyms	Febbre reumatica. ICD9: 390,391 ICD10: I00,I01,I02

Clinical

Case definition for surveillance:
The CDC (The United States Centers for Disease Control) case definition for surveillance requires evidence for preceding group A streptococcal infection (culture, serology) in addition to two major clinical criteria; or one major and two minor criteria, as follows:

Major clinical criteria:
- carditis
- polyarthritis
- chorea [1] [2]
- subcutaneous nodules
- erythema marginatum. [3]

Minor criteria:
- previous rheumatic fever or rheumatic heart disease
- arthralgia
- fever
- elevation of erythrocyte sedimentation rate [ESR]
- positive C-reactive protein
- leucocytosis
- prolongation of the P-R interval on electrocardiogram.

This disease is endemic or potentially endemic to all countries.

Rheumatic fever in Brazil

151 deaths were ascribed to 'active rheumatic fever' in 1994; 157 in 1995.

72.1% of patients with acute rheumatic fever will later develop chronic valvular disease, with 15.9% progressing to severe chronic mitral or aortic lesions (Minas Gerais). [4]

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Rhinoscleroma and ozena

Agent	BACTERIUM. Klebsiella pneumoniae ssp ozaenae and Klebsiella pneumoniae ssp rhinoscleromatis Facultative gram-negative bacilli
Reservoir	Human
Vector	None
Vehicle	Infected secretions
Incubation Period	Unknown
Diagnostic Tests	Culture. Biopsy. Nucleic acid amplification. Advise laboratory when this diagnosis is suspected.
Typical Adult Therapy	Rhinoscleroma: Streptomycin, often with systemic or topical Rifampin - for 3 to 6 weeks; fluoroquinolones also appear to be effective. Ozena: Ciprofloxacin or Sulfamethoxazole/trimethoprim for 3 months
Typical Pediatric Therapy	As for adult
Clinical Hints	Rhinorrhea associated with a painless intranasal mass; may extend to sinuses or ears.
Synonyms	Klebsiella pneumoniae ssp ozaenae, Ozena, Rhinoscleroma. ICD9: 040.1 ICD10: J31.0

Clinical

The nose is involved in over 90% of cases of rhinoscleroma.
- Findings include fetid discharge, a crusting granulomatous mass and cicatrization. [1] [2]
- The pharynx is involved in 15% to 40%, the larynx in 2% to 2%, the tracheobronchial tree in 15% [3] and the paranasal sinuses in 2% to 25%. [4]
- Rare instances of laryngeal stenosis resulting from rhinoscleroma are reported. [5]
- Standard therapy consists of streptomycin in combination with topical or systemic rifampicin, for at least 3 to 6 weeks.
- Recent studies suggest that fluoroquinolones are also effective.

Ozena (primary atrophic rhinitis) is characterized by progressive atrophy of the nasal mucosa and underlying bone.
- Findings include foul-smelling, thick, dry crusts and greatly enlarged nasal cavities. [6]
- Laryngeal involvement has been reported. [7]
- Ozena may be associated with tracheobronchopathia osteochondroplastica [8]
- Rare instances of disseminated systemic infection are reported. [9]

This disease is endemic or potentially endemic to all countries.

Rhinoscleroma and ozena in Brazil

Sporadic cases of rhinoscleroma are reported. [10-12]

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Rhinosporidiosis

Agent	PROTOCTISTA Rhinosporidium seeberi [may in fact be Microcystis, a cyanobacterium]
Reservoir	Water Soil Vegetation
Vector	None
Vehicle	Aerosol from soil or water
Incubation Period	2w - 6m
Diagnostic Tests	Histology of resected material (organism does not grow in-vitro).
Typical Adult Therapy	Excision
Typical Pediatric Therapy	As for adult
Clinical Hints	Friable, painless vascular masses of nose, conjunctivae and larynx; recurrence is common.
Synonyms	Rhinosporidium seeberi. ICD9: 117.0 ICD10: B48.1

Clinical

Clinical forms include:
• nasal (chronic, painless unilateral obstruction, mucoid discharge) [1]
• conjunctival (usually palpebral) [2] or lacrimal lesion. [3] [4]
• ENT (mucous membrane mass of the epiglottis, tongue, palate, tonsil, uvula, larynx [5] [6], paranasal sinuses. [7]
• urethral (predominantly male), presenting as a painless, friable polyp of the fossa navicularis. [8]

Multiple painless dermal or subcutaneous nodules may be present. [9-14]
• Rarely, skin lesions may be polymorphic [15]
• Additional manifestations have included primary cutaneous lesions [16], osteomyelitis [17-19], bronchial mass lesion [20], obstructive tracheitis [21] and infection of the parotid duct. [22-25]

Relapse occurs in approximately 10% of cases following excision.

Signs of mucosal chromomycosis may mimic those of rhinosporidiosis. [26]

This disease is endemic or potentially endemic to 65 countries.

Rhinosporidiosis in Brazil

Most cases are reported from the southern portion of the country. [27-29]

Infection has been reported in animals. [30]

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Rhodococcus equi infection

Agent	BACTERIUM. Rhodococcus equi An aerobic gram-positive coccobacillus
Reservoir	Farm animal Farm soil
Vector	None
Vehicle	? Inhalation Contact Ingestion
Incubation Period	Unknown
Diagnostic Tests	Culture of blood, body fluids and secretions. Advise laboratory when these organisms are suspected.
Typical Adult Therapy	Vancomycin 500 mg q8h. Alternatives: Erythromycin, Gentamicin, Rifampin
Typical Pediatric Therapy	Vancomycin 10 mg/kg q6h. Alternatives: Erythromycin, Gentamicin, Rifampin
Clinical Hints	Most often encountered as pleuropulmonary infection in an immune-suppressed patient; history of contact with farm or farm animals in 40% of cases.
Synonyms	Rhodococcus. ICD9: 027.9 ICD10: A92.8

Clinical

The clinical features of *Rhodococcus equi* disease are largely determined by the site of infection and clinical substrate in which it occurs. [1] [2]

- 49% of patients are HIV-positive.
- Pulmonary infection predominates among HIV-positive patients [3]
- Extrapulmonary disease (abscesses. septicemia, eye or wound infection, etc) is most common in immunocompetent individuals. [4]

This disease is endemic or potentially endemic to all countries.

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Rickettsia felis infection

Agent	BACTERIUM. Rickettsia felis
Reservoir	Opossum (Didelphis marsupialis) ? Flying squirrel Raccoon Cat Flea ? Dog
Vector	Flea (cat flea = Ctenocephalides felis). Organism has also been found in Pulex irritans
Vehicle	None
Incubation Period	Unknown
Diagnostic Tests	Serology (IFA). Nucleic acid amplification. Note that Weil-Felix reaction may be positive (OX-19).
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3 to 5d. OR Chloramphenicol 500 mg PO QID X 3 to 5d
Typical Pediatric Therapy	Doxycycline 2 mg/kg PO BID X 3 to 5d (maximum 200 mg/day). OR Chloramphenicol 10 mg/kg PO QID X 3 to 5d
Clinical Hints	Fever, headache and myalgia; macular rash present in 20% of patients; history of recent contact with opossum; disease mimics endemic typhus.
Synonyms	California pseudotyphus, Cat flea typhus, ELB agent. ICD9: 081.1 ICD10: A79.8

Clinical

The features of *Rickettsia felis* infection are similar to those of endemic typhus.
• Headache and myalgia predominate.
• The rash is often macular and most prominent on the trunk and abdomen.
• Often the rash is nonspecific, and may be lacking in 50% of patients. In some cases, polymorphous skin lesions, including papules, vesicles, erosions and ulcers are present. [1]
• Major complications are rare.
• The severity of infection has been associated with old age, delayed diagnosis, hepatic and renal dysfunction, central nervous system abnormalities, and pulmonary compromise.
• As many as 4% of hospitalized patients die.

This disease is endemic or potentially endemic to 47 countries.

Rickettsia felis infection in Brazil

Evidence for human infection by *Rickettsia felis* has been demonstrated in two Brazilian patients with febrile rash. [2] [3]

Infection of a dog by *Rickettsia felis* has been documented in a dog from Sao Jose dos Pinhais, Parana State. [4]

Rickettsia felis has been documented in fleas (*Ctenocephalides* spp) in Minas Gerais [5]
- *R. felis* has been documented in dog fleas (*Ctenocephalides felis* and *Ctenocephalides canis*) in a Guarani Indian community in suburban Sao Paulo. [6]
- *Rickettsia felis* was identified in dog ticks (*Rhipicephalus sanguineus*) and fleas (*Ctenocephalides felis*) in Espirito Santo. (2008 publication) [7]
- *Rickettsia felis* was identified in fleas (*Ctenocephalides felis*, *Rhipicephalus sanguineus* and *Amblyomma cajennense*) infesting dogs and horses. [8] [9]

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Rocio

Agent	VIRUS - RNA. Flaviviridae, Flavivirus: Rocio virus
Reservoir	? Wild bird
Vector	Mosquito (Psorophora ferox, Aedes scapularis)
Vehicle	None
Incubation Period	average 12 d (range 7d - 15d)
Diagnostic Tests	Viral culture (brain, serum). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, vomiting, conjunctivitis, pharyngitis and abdominal distention; CNS symptoms may be present; case fatality rate = 5% to 25%. permanent neurological residua reported in 20%.
Synonyms	

Clinical

A prodrome of fever, headache, malaise, vomiting, and conjunctivitis is followed by altered consciousness, motor weakness and cerebellar dysfunction. [1] [2]

- One third of patients progress to coma, and 10% die of the disease. [3]
- Sequelae remain in 20% of survivors.

This disease is endemic or potentially endemic to 1 country.

Rocio in Brazil

Time and Place:

- Rocio encephalitis was first reported in Brazil in 1975.
- Most cases are limited to the coastal plain south of Santos. [4]
- Cases of Rocio infection have been documented in the Rebeira valley region (1989 publication). [5]

Sporadic cases were reported in 1983 and 1987.

Vectors:

- The local vector is *Psorophora ferox*. [6]
- *Aedes scapularius* has also been implicated in transmission. [7]

Seroprevalence surveys:

0% of horses and caimans from the Pantanal (2009) [8]

Seropositive birds have been identified in Sao Paulo State (1978 to 1990) [9]

Notable outbreaks:

1975 - An outbreak (465 cases, 61 fatal) was reported in southern Sao Paulo State in 1975. [10] The epidemic extended southward in 1976, resulting in an additional 825 cases (95 fatal). [11]

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Rocky Mountain spotted fever

Agent	BACTERIUM. Rickettsia rickettsii
Reservoir	Tick Dog Rodent
Vector	Tick (Dermacentor, Amblyomma)
Vehicle	None
Incubation Period	5d - 7d (range 2d - 14d)
Diagnostic Tests	Serology. Direct immunofluorescence or culture of skin lesions. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3 to 5d. OR Chloramphenicol 500 mg PO QID X 3 to 5d
Typical Pediatric Therapy	Doxycycline 2 mg/kg PO BID X 3 to 5d (maximum 200 mg/day). OR Chloramphenicol 10 mg/kg PO QID X 3 to 5d
Clinical Hints	Headache, myalgia, vomiting and a maculopapular or petechial rash (primarily involving the extremities); may be history of a tick bite or dog contact during the preceding 1 to 2 weeks; rash absent in 5%. case-fatality rate (untreated) = 25%.
Synonyms	American spotted fever, Bullis fever, Febre maculosa brasileira, Fiebre manchada, Lone star fever, Rickettsia 364D, Rickettsia amblyommii, Rickettsia canadensis, Rickettsia montanensis, Rickettsia parkeri, Rickettsia rickettsii, Rickettsia texiana, Rickettsiae, RMSF, Sao Paulo fever, Tidewater spotted fever, Tobia fever. ICD9: 082.0,082.8 ICD10: A77.0

Clinical

Rocky Mountain spotted fever usually begins with fever, myalgia, and severe headache, often accompanied by vomiting, abdominal pain, diarrhea and abdominal tenderness. [1-3]

- A rash is encountered in 80% to 85% of cases, and usually appears 3 to 5 days after the onset of fever. [4]
- The rash begins around the wrists and ankles but may start on the trunk or be diffuse at the onset. [5]
- The palms and soles are not always involved, or only involved late in the illness.
- An eschar (tache noire) has been described in some cases [6] ; and dermal necrosis or acral gangrene may develop in a few percent of patients. [7] [8]

Meningitis with CSF pleocytosis (either lymphocytic or polymorphonuclear) occurs in one third of the patients.

- Renal failure is often encountered.
- Other complications may include deafness, autoimmune vasculitis [9] , pneumonia, ARDS, pulmonary edema or myocarditis. [10] [11]
- Laboratory findings include a normal leucocyte count in most cases, with occasional anemia or thrombocytopenia.
- Bleeding diatheses are uncommon.
- Hyponatremia encountered in 50% of patients.

Mortality is correlated with delay in diagnosis and treatment, old age, absence of rash ("spotless fever") or known tick exposure, and onset during winter months.

- Mortality rates are highest among immune-suppressed patients, American Indians, children ages 5 to 9 years, and adults >=70 years of age (United States, 1999 to 2007) [12]

A second agent of 'spotted fever,' *Rickettsia parkeri*, is transmitted by *Amblyomma maculatum* [13] in the southern United States.

- Illness is similar to that caused by *Rickettsia rickettsii*; however, a dermal eschar and vesicular rash may be present. [14]
- *R. parkeri* infection is relatively mild, with lower rates of hospitalization and mortality than those of Rocky Mountain Spotted fever. [15]

Rickettsia amblyommii infection has been associated with a rash at the site of tick attachment. (2006) [16]

Rickettsia 364D infection (vector, *Dermacentor occidentalis*) is associated primarily with the appearance of a single cutaneous eschar. (2010 publication) [17]

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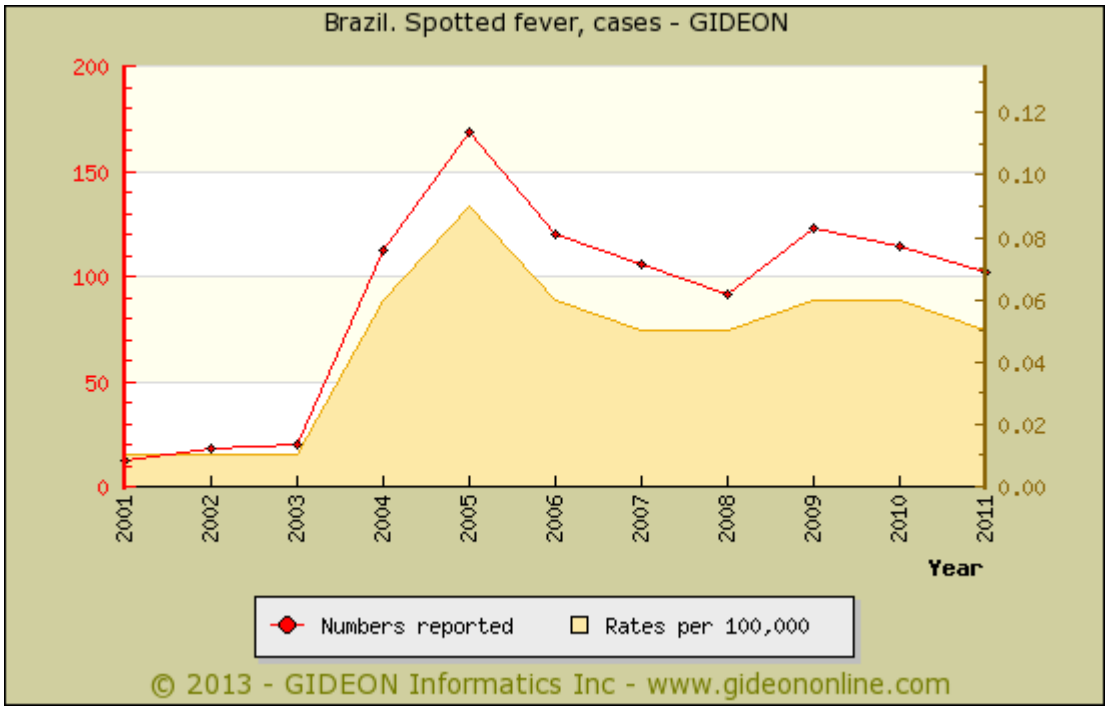
This disease is endemic or potentially endemic to 11 countries.

Rocky Mountain spotted fever in Brazil

Time and Place:
Rickettsial spotted fever is known locally as Brazilian spotted fever, Sao Paulo fever and Febre maculosa brasileira.
- Rickettsial spotted fever was first reported in Brazil in 1920.
- Cases are reported in Minas Gerais [18] , Bahia, Goias, Rio Grande do Sul, Sao Paulo and Espirito Santo. [19]
- The disease has re-emerged in Rio de Janeiro in recent years. [20]



Graph: Brazil. Spotted fever, cases

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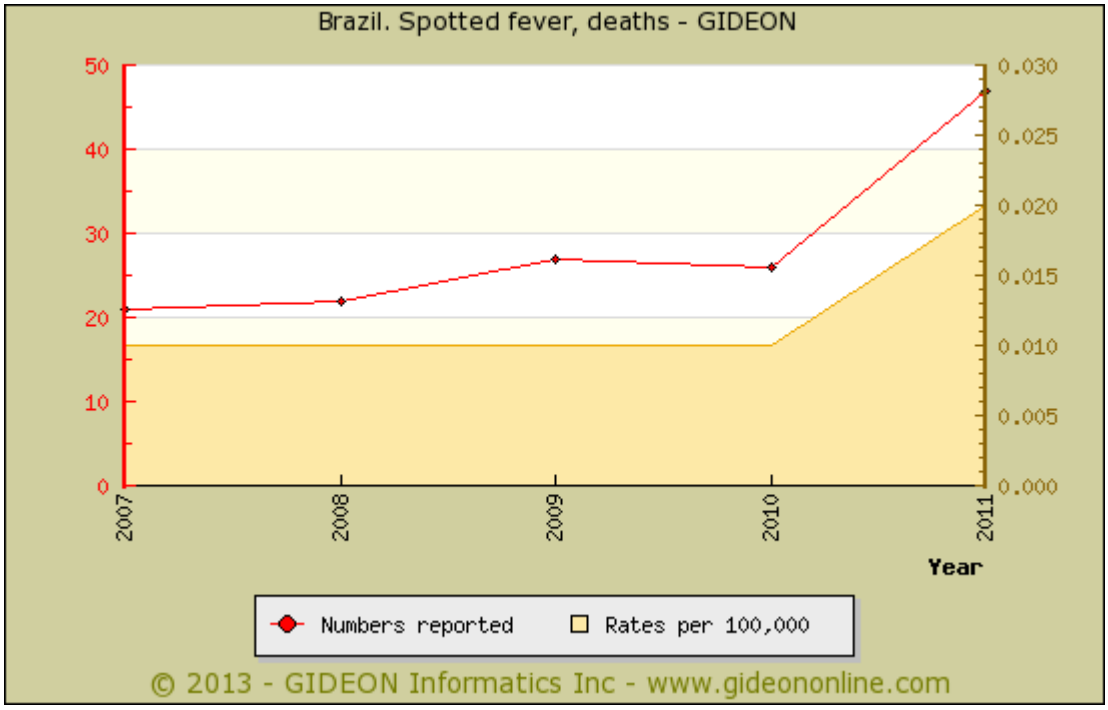

Graph: Brazil. Spotted fever, deaths

Geographical notes:

- Bahia: A case of eschar-associated spotted fever rickettsiosis (species not determined) was reported in 2007. [21]
- Minas Gerais: Outbreaks were reported during 1929 to 1944, but not during 1945 to 1980.
Subsequent outbreaks were reported in Minas Gerais in 1981, 1984, 1992, 1995 and 2000.
92 cases (40% fatal) were confirmed in Minas Gerais during 1981 to 1989; 78 during 1992 to 1997 (including 3 fatal cases in 1996, and 2 in 1997) [22] ; 210 during 1999 to 2009; 6 in 2010. [23]
- Sao Paulo: 67 cases were reported during 1985 to 2000; 224 (including 6 case-clusters) during 2001 to 2008 [24] ; 5,949 suspected cases (206 confirmed) during 2003 to 2006. [25]
17 cases (8 fatal) were reported in Pedreira county, Sao Paulo during 1985 to 1995. [26]
Five fatal cases were reported in Sao Paulo State in 2003. [27]
Eight outbreaks were reported in Sao Paulo State during 2005. [28]
38 cases (2 confirmed, 1 fatal) were reported in Valinhos in 2010; 27 (7 confirmed, 5 fatal) during January to July, 2011. [29]
29 cases were reported in Piracicaba during 2002 to 2011; 1 in 2011; 18 during January to September, 2012. [30]

Prevalence surveys:

1.3% of *Rhipicephalus sanguineus* ticks in metropolitan Sao Paulo are infected by *Rickettsia rickettsii* (2009 publication) [31]

1.3% of *Amblyomma cajennense* in Minas Gerais (1997 publication) [32]
Rickettsia parkeri was identified in 9.7% of *Amblyomma triste* (a non-human biting tick) in Pauliceia County, Sao Paulo (2005). [33]

Seroprevalence surveys:

1.6% of healthy persons in Minas Gerais (2005 publication) [34]
10.6% of healthy students and 4.11% of dogs in the endemic areas of Minas Gerais (1998) [35]
38.4% of humans, 7.6% of dogs and 25.9% of horses (Espirito Santo, *Rickettsia* spp., 2010 publication) [36]
77.3% of horses and 31.3% of dogs in Pedreira Municipality, Sao Paulo State (2001) [37]
25.7% of dogs in southern Brazil (*Rickettsia parkeri*, 2002 to 2003) [38]
69.6% of dogs in metropolitan Sao Paulo (2009 publication) [39]
4.4% of dogs from Sao Jose dos Pinhais, Parana State (2006 to 2007) [40]
47.5% of dogs in the Pantanal region of Mato Grosso State (*Ehrlichia canis*, 2011 publication) [41]
10% of free-living jaguars (*Panthera onca*) in Pantanal (*Rickettsia parkeri*, 2011 publication) [42]

Reservoirs:

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- The principal reservoir hosts are capybara (*Hydrochaeris hydrochaeris*) [43] , horses [44] and occasionally dogs. [45]
- Natural infection has been identified in dogs in Sao Paulo. [46]
- *R. parkeri* antibodies have been documented in dogs from urban areas in the western Brazilian Amazon. (Monte Negro, Rondonia) [47]

Vectors:

The principal vectors are *Amblyomma aureolatum* (areas of Sao Paulo) [48] and *Amblyomma cajennense*. [49] [50]
- *Amblyomma cooperi* has also been implicated (in Sao Paulo). [51]
- Spotted fever-group rickettsiae have been found in *Rhipicephalus sanguineus* in Rio de Janeiro State [52] , and *Rickettsia rickettsii* has been found in a *Rhipicephalus sanguineus* tick from Mato Grosso do Sul. [53]

Notable outbreaks:

1993 (publication year) - An outbreak (6 cases, 4 confirmed) was reported in Espirito Santo. [54]
2004 (publication year) - An outbreak was reported in Campinas. [55]
2004 - An outbreak (3 fatal cases) was reported in Maua (Grande Sao Paulo). [56]
2005 - An outbreak (7 cases, 2 fatal) was reported in a mountain resort near Rio de Janeiro. [57]
2008 - An outbreak (3 fatal cases) was reported in Sao Paulo State. [58]

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Rotavirus infection

Agent	VIRUS - RNA. Reoviridae: Rotavirus
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Water
Incubation Period	12h - 3d
Diagnostic Tests	Stool assay for viral antigen. Serology. Nucleic acid amplification.
Typical Adult Therapy	Stool precautions; supportive
Typical Pediatric Therapy	As for adult
Vaccine	Rotavirus
Clinical Hints	Vomiting, diarrhea and mild fever: the illness lasts approximately 1 week, and is most severe in infancy; fatal cases are associated with dehydration and electrolyte imbalance.
Synonyms	Rotavirus. ICD9: 008.61 ICD10: A08.0

Clinical

Infants and young children present with fever, vomiting, diarrhea, and occasionally dehydration. [1]
• Most hospitalized patients had experienced fever and vomiting for 2 to 3 days, and diarrhea for 4 to 5 days.
• The diarrhea is watery without blood or mucus.
• Leukocytes are detected in the stool in a small percentage of patients.
• Approximately 36% of episodes are characterized by 'dehydrating diarrhea.'
• Viremia is present in over 50% of patients with Rotavirus diarrhea. [2] [3]
• Asymptomatic infection is common. [4]

Infection in immunodeficient children may persist for weeks to months.

Rotavirus infection is not unusual in adults. [5]

Complications:
• Rotavirus infection increases the risk of bacteremia in children with nontyphoid *Salmonella* gastroenteritis [6]
• Rare instances of toxic megacolon have been reported. [7]
• Although intestinal intussusception may occur in some cases [8] , a causal role for Rotavirus infection (ie, as opposed to Rotavirus vaccine [9]) is not established. [10]
• Central nervous system dysfunction may complicate Rotavirus infection, in the form of seizures [11-14] , cerebellitis [15-18] , encephalopathy [19] [20] and death. [21]
• Some reports have linked Rotavirus infections with instances of aseptic meningitis [22] [23] , necrotizing enterocolitis, myositis, liver abscess, pancreatitis [24] [25] , pneumonia, Kawasaki's disease, acute hemorrhagic edema [26] , sudden infant death syndrome and Crohn's disease.

This disease is endemic or potentially endemic to all countries.

Rotavirus infection in Brazil

Time and Place:
The presence of Rotavirus in Brazil was first confirmed in 1976, in Belem. [27]
- Each year, Rotavirus infections are estimated to cause 3,525,053 episodes of diarrhea, 655 853 visits to outpatient healthcare facilities, 92 453 hospitalizations, and 850 deaths of children <= 5 years. [28] [29]
- Over 70% of children are seropositive by age 4 to 5 years.
- Disease rates are highest during March to September, with a peak during June (Minas Gerais). [30]

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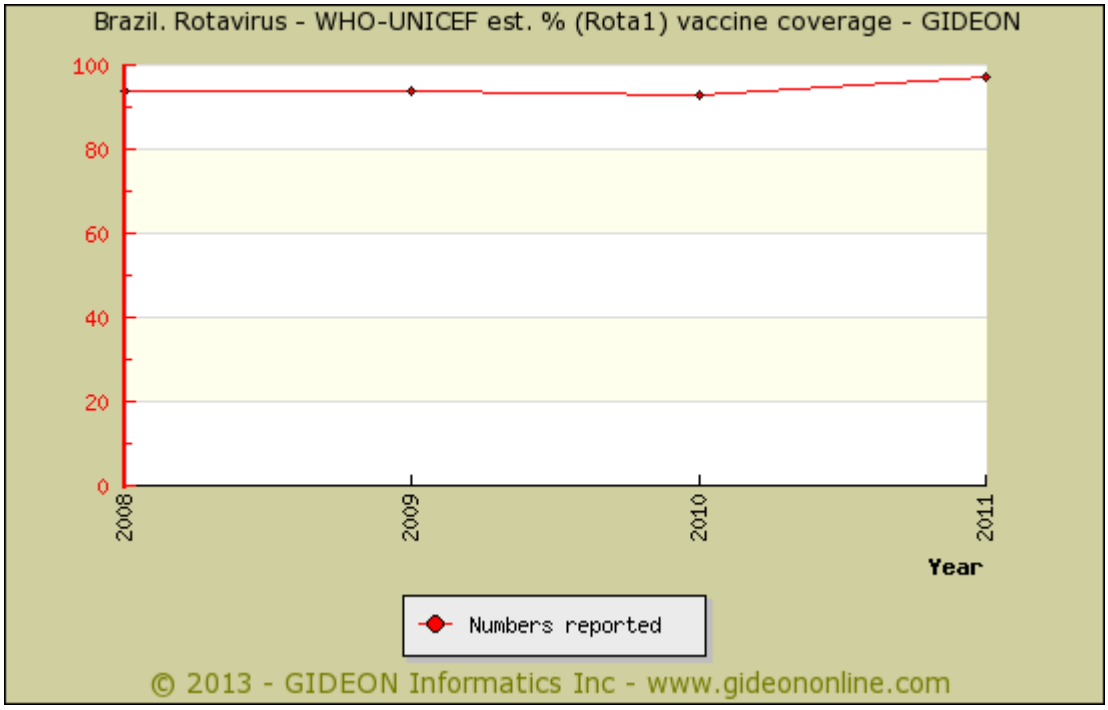
- Introduction of routine Rotavirus vaccination in 2006 was followed by a 26% reduction in pediatric hospitalization for gastroenteritis in 2006, 48% reduction in 2007 [31] and additional decreases during 2008 to 2009. [32-37]

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years



Graph: Brazil. Rotavirus - WHO-UNICEF est. % (Rota1) vaccine coverage

Notes:
1. Analysis of Rotavirus vaccine coverage in Brazil - see reference [38]

Prevalence surveys:
12% to 42% of acute diarrhea in Brazil (0.25 episodes per child per year)
20% of stool samples received by a hospital laboratory in Sao Paulo (2003 to 2005) [39]
19.2% of stool samples from patients with gastroenteritis (Rio de Janeiro, 2005 to 2008) [40]
0% of HIV-positive patients with diarrhea, and 0% of control patients (southeastern Brazil, 2006 to 2007) [41]
1.5% of HIV-positive patients with diarrhea, and 2.5% of HIV-negative patients with diarrhea (1995 to 1999) [42]
56% of patients hospitalized for acute diarrhea (Ponta Grossa, Londrina and Assai, Parana, 2011 publication) [43]
24.9% of children ages <=15 years with acute gastroenteritis (2007 to 2010) [44]

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14.4% of children with diarrhea below age 10 in Goias (1986 to 2000) [45]
29% of children with diarrhea below age 5, nationwide (1998 to 1999)
22% of pediatric hospitalizations for diarrhea in (2010) [46]
7.9% of urban children with diarrhea in Salvador (2000 to 2002) [47]
15% of children with diarrhea in Uberaba, Minas Gerais (2008 publication) [48]
19.3% of children with gastroenteritis, in Pernambuco (2010 publication) [49]
33.8% of stool samples from children below age 2 years, in 2005, 23.7% in 2006, 16.8% in 2007, 22.9% in 2008, and 18.3% in 2009 [50]
35% of children below age 5 years with acute diarrhea, in Recife (2004 to 2005) [51]
25% of children below age 5 years with acute gastroenteritis, in Sao Paulo (2009 publication) [52]
25.8% of children below age 5 years hospitalized for acute gastroenteritis (Sao Paulo, 2004 to 2008) [53]
29.6% of hospitalized children ages ? 5 years with acute gastroenteritis in Sao Paulo, Brazil (2004 to 2009) [54]
43.3% of children below age 5 years hospitalized for acute gastroenteritis (Sao Paulo, Salvador, Goiania, and Porto Alegre, 2005 to 2006) [55]
23.6% of children below age 5 with diarrhea and 10.1% of healthy controls, in Rondonia (2007 publication) [56]
23.6% of children below age 6 years with diarrhea, in Porto Velho (Rondonia, 2006 publication) [57]
16.1% of children in a day-care center, with acute gastroenteritis (Rio de Janeiro, 1994 to 2008) [58]
8.2% of children with acute diarrhea, in Rio de Janeiro (2002 to 2004) [59]
46.9% of children admitted to a pediatric hospital with acute diarrhea (PCR detection) (Salvador, Bahia, 1999, 2000 and 2002) [60]
32.6% of hospitalized and 3.4% of outpatient children ages 0 to 5 years, with diarrhea (Minas Gerais, 1998) [61]
23.2% of hospitalized children with acute gastroenteritis in Campo Grande (2000 to 2004) [62]
28.3% of stool samples from children hospitalized for gastroenteritis (Parana, EIA, 2001 to 2008) [63]
20.5% of children hospitalized with gastroenteritis. (Vitoria city, 2004 to 2006) [64]
36.9% of infants hospitalized with acute diarrhea (Natal, 2008 publication) [65]
6% of children below age 5 years hospitalized with Astrovirus gastroenteritis (Rio de Janeiro, 2004) [66]
4% of children hospitalized with diarrhea were found to have mixed Rotavirus / Norovirus infection (Rio de Janeiro, 2004) [67]
0% of asymptomatic children ages 4 to 14 months, in a day-care center (2012 publication) [68]
0% of recently-captured primates (Red-howler *Alouatta guariba clamitans*; Howler, *Alouatta caraya*; Marmosets, *Callithrix* spp. *Callithrix penicillata*; Black-faced lion tamarin, *Leontopithecus caissara*) in southern Brazil (2012 publication) [69]
44.2% of water samples in the area of Manaus (2007 publication) [70]
24.3% of recreational waters in a lagoon (Rio de Janeiro, 2012 publication) [71]
0% of surface water samples in Arroio Diluvio, Porto Alegre (2009) [72]
65% of surface water samples in Florianopolis, Santa Catarina (2012 publication) [73]
19% of water samples and 8.3% of oyster samples in Florianopolis (2007 to 2008) [74]

Norovirus or enteric Adenovirus coinfection was found in 14% of Rotavirus-positive samples from hospitalized patients (2012 publication) [75]

Seroprevalence surveys:

36% of adults in a suburban community (1998 publication) [76]

Notable outbreaks:

1977 - An outbreak (157 cases) of Rotavirus infection was reported among Tiriyo Indians in Para. [77]
1980 - An outbreak of *Shigella sonnei* and Rotavirus infections was reported in a private school in Rio de Janeiro. [78]
1987 (publication year) - An outbreak of Rotavirus infection was reported among members of a family in Belem, Para. [79]
1992 - An outbreak (132 cases) of Rotavirus infection was reported in Mirassol, Sao Paulo. [80]
1993 - An outbreak of Rotavirus infection was reported in Valentim Gentil, Sao Paulo. [81]
1993 - An outbreak (64 cases) of Rotavirus infection was reported among children attending a day-care centre in Belem. [82]
1994 (publication year) - An outbreak of Rotavirus infection was reported in a day care nursery in Rio de Janeiro. [83]
2003 to 2004 - Outbreaks were reported in Campinas, Sao Paulo. [84]
2005 - An outbreak of Rotavirus gastroenteritis was reported in Rio Branco City, Acre State. [85]
2006 - An outbreak was reported in Bom Jesus City, Piaui State. [86]
2010 - Outbreaks of Rotavirus and Norovirus infection were reported at two correctional facilities. [87]

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Rubella

Agent	VIRUS - RNA. Togaviridae: Rubella virus
Reservoir	Human
Vector	None
Vehicle	Contact Air Transplacental
Incubation Period	16d - 18d (range 14d - 23d)
Diagnostic Tests	Viral culture (throat, urine). Serology. Nucleic acid amplification.
Typical Adult Therapy	Respiratory precautions. Supportive
Typical Pediatric Therapy	As for adult
Vaccines	Rubella Rubella - Mumps Measles-Mumps-Rubella Measles-Rubella
Clinical Hints	Maculopapular rash following a one-day prodrome of coryza and headache; post auricular lymphadenopathy; arthralgia and arthritis encountered in adults; severe thrombocytopenia or encephalitis may follow acute infection.
Synonyms	Epidemic roseola, German measles, Roda hund, Rode hond, Rode hunder, Rodehond, Rosolia, Roteln, Rubeola [Spanish], Three-day measles. ICD9: 056 ICD10: B06

Clinical

CDC (The United States Centers for Disease Control) case definition for surveillance:
For surveillance purposes, the CDC (The United States Centers for Disease Control) case definition of rubella requires, "An illness that has all of the following characteristics:
• acute onset of generalized maculopapular rash
• temperature >37.2 C if measured
• arthralgia/arthritis, lymphadenopathy, or conjunctivitis" [1] Arthropathy may occur in as many as 41% of cases [2]

A "confirmed" case requires either laboratory confirmation or epidemiological link to a laboratory-confirmed case.
• Atypical features may be seen in adults with rubella; ie, hepatitis, conjunctival hemorrhage [3] , uveitis [4] , retinitis [5] and a high incidence of polyarthritis.
• Rare instances of acute hepatic failure [6] and hemophagocytic syndrome [7] are reported.

Congenital rubella should be suspected if any of the following is present in a newborn infant [8] [9] :
• cataracts (45% of cases), congenital glaucoma, pigmentary retinopathy
• congenital heart disease (70%, most commonly patent ductus arteriosus or pulmonary artery stenosis) Both anomalies may appear concurrently in up to 50% of cases [10]
• hearing loss (35% to 60%)
• purpura
• splenomegaly
• jaundice
• microcephaly, mental retardation [11] , meningoencephalitis
• radiolucent bone disease
• duodenal stenosis [12]

The chance of fetal defects from a viremic mother is 40% to 90% during the first trimester. [13]
• Infection also increases the risk for spontaneous abortion and miscarriage by 50%. [14]
• The rate of congenital rubella syndrome during epidemics is 0.5 to 2.2 per 1,000 live births.
• 60% of children with CRS have hearing impairment, 45% congenital heart disease, 27% microcephaly, 25% cataracts, 23% low birth weight (< 2,500 grams), 17% purpura, 19% hepatosplenomegaly, 13% mental retardation and 10% meningoencephalitis.

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Anterior uveitis • differential diagnosis:
Anterior uveitis due to Rubella virus is characterized by younger age at onset and a chronic course, typically associated with cataract at presentation. [15]
• Rubella virus has been implicated in the etiology of Fuchs heterochromic iridocyclitis. [16]
• Anterior uveitis due to Herpes simplex and Varicella-Zoster viruses is more common in adults, and often follows an acute course.
• Herpes simplex anterior uveitis presents with conjunctival redness, corneal edema, a history of keratitis, and the presence of posterior synechiae. Anterior chamber inflammation is common with Herpes simplex virus, while vitritis is more common with Rubella and Varicella-Zoster virus.
• Rubella, Herpes simplex and Varicella-zoster viruses are associated with intraocular pressure of more than 30 mmHg and development of glaucoma (18%-30%; P = 0.686).
• Focal chorioretinal scars were present in 22% of Rubella cases, 0% of HSV and in 11% of VZV uveitis cases.

This disease is endemic or potentially endemic to all countries.

Rubella in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Routine immunization using MMR or MR was introduced in 1992; and routine administration at age 12 months was introduced in 2000.

No cases were reported between and

Prevalence surveys:
20.2% of patients with maculopapular rash (Niteroi, Rio de Janeiro State, 1994 to 1998) [17]
13% of patients with fever and rash, initially diagnosed as measles (Sao Paulo, 2000 to 2004) [18]
5.7% of patients with fever and rash in Campinas (2003 to 2004) [19]
3.9% of dengue-like illness (Federal District, 2008) [20]

Seroprevalence surveys:
89% of pregnant women in Parana (IgG, 1996 to 1998)
99.6% of pregnant women in Parana (2010) [21]
71.6% of pregnant women in Sergipe (2009 publication) [22]
93.1% of pregnant women in Sao Paulo (2011 publication) [23]
61% of pregnant and post-partum women in Forteleza (1997) [24]
92.7.% of 15 to 19 year-olds, 82.4% of 20 to 29 year-olds and 90.7% of 30 to 39 year-olds in Guarantingueta (2009 publication) [25]
83.9% of women ages 12 to 29 years, prior to an immunization campaign; 92.5% following the campaign (2011 publication) [26]

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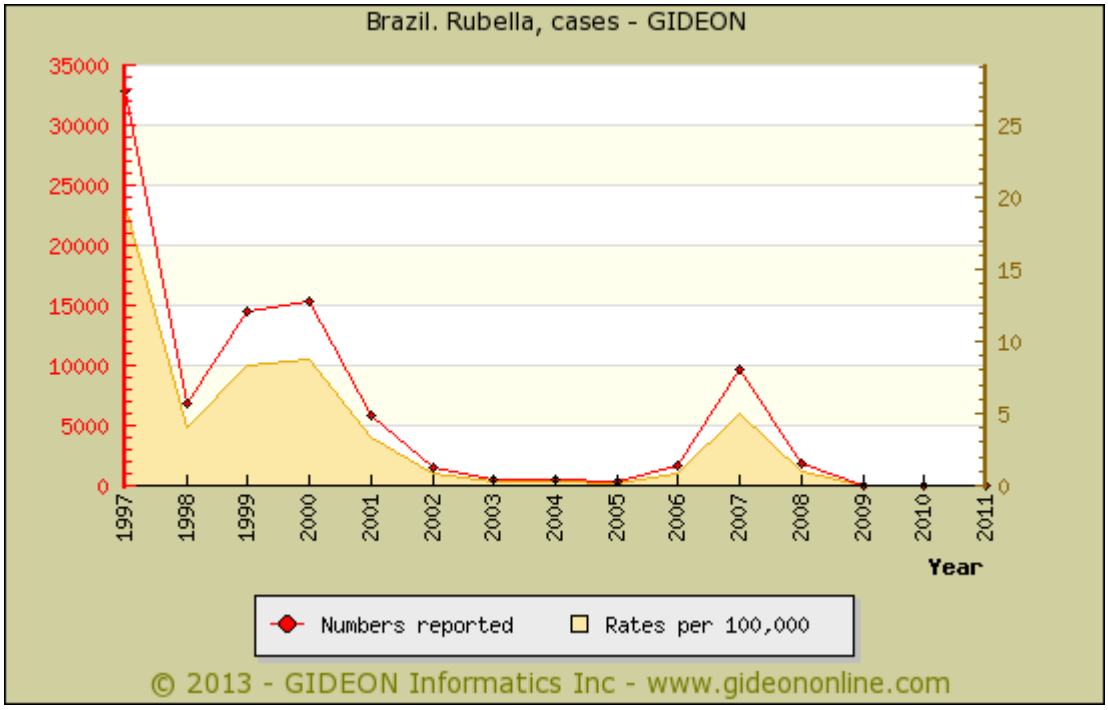
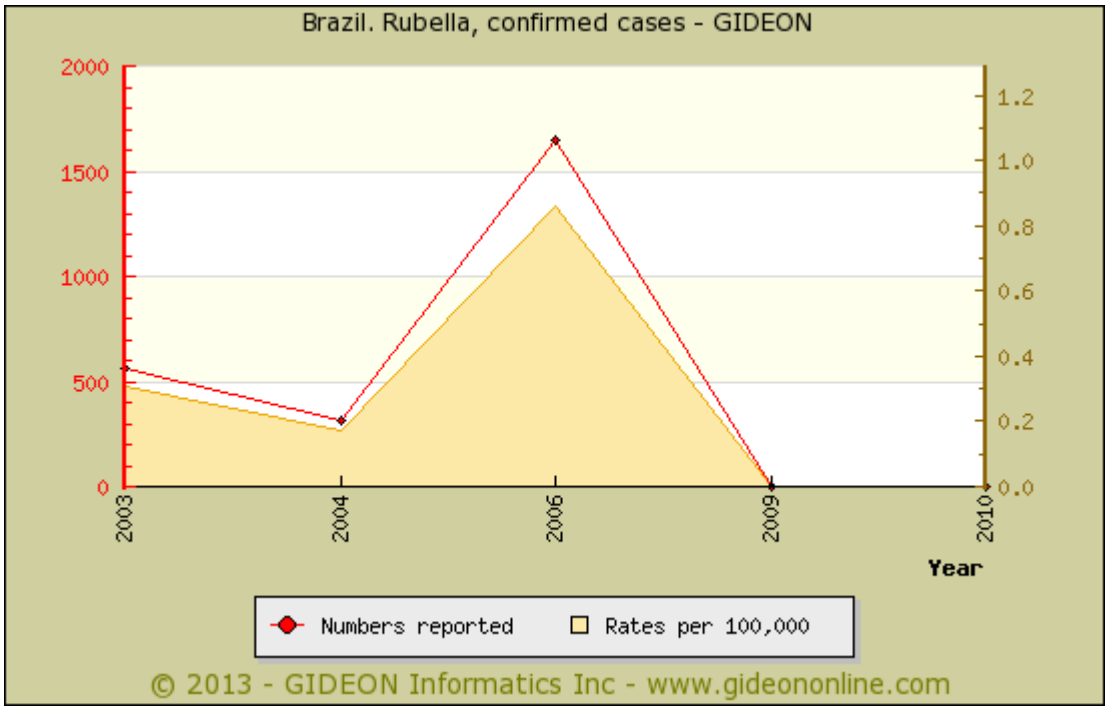


Graph: Brazil. Rubella, cases

Notes:

1. Outbreaks of rubella were reported in Rio de Janeiro in 1968, 1974 and 1981. [27] [28]
2. 68,878 cases were reported during 1997 to 2000 - including 24,841 in Sao Paulo and 5,538 in Rio de Janeiro.
3. 1,266 cases (12 CRS) were confirmed in Paraiba during 1999 to 2005. [29]
4. Officially-reported disease rates were 18.8 per 100,000 in 2000, and 0.9 per 100,000 in 2005 100,000 [30]

16.9% of patients suspected to have measles and/or rubella were found to have Parvovirus B19 infection, and 16.9% dengue. (Pernambuco, 2001 to 2004) [31]



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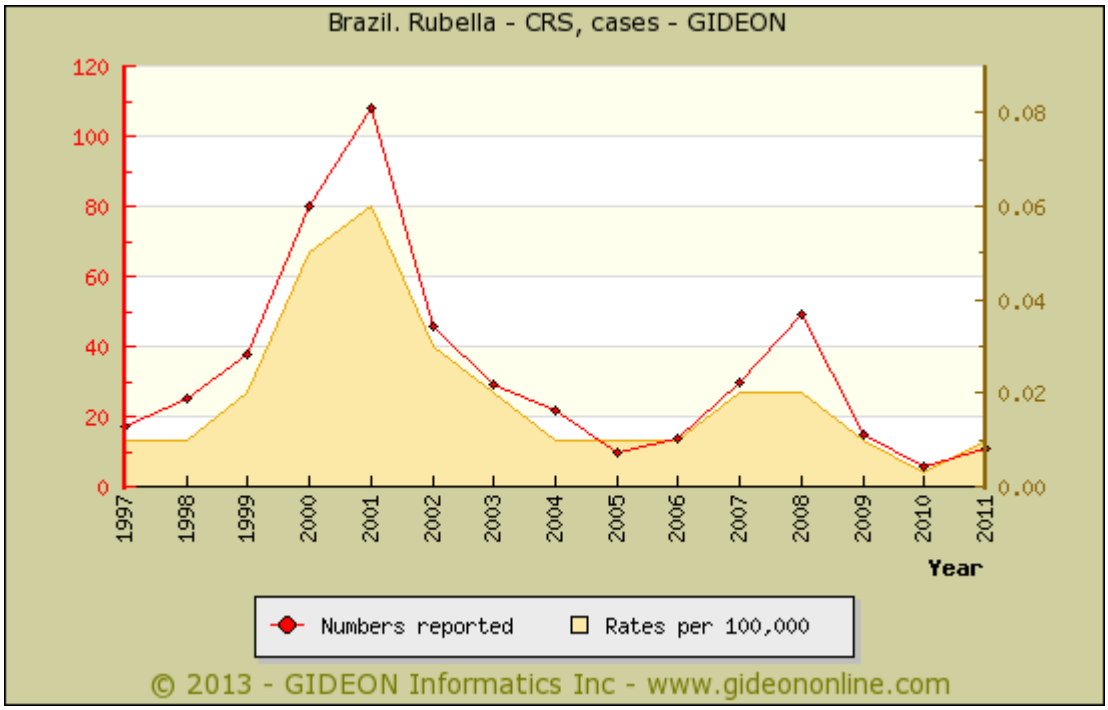
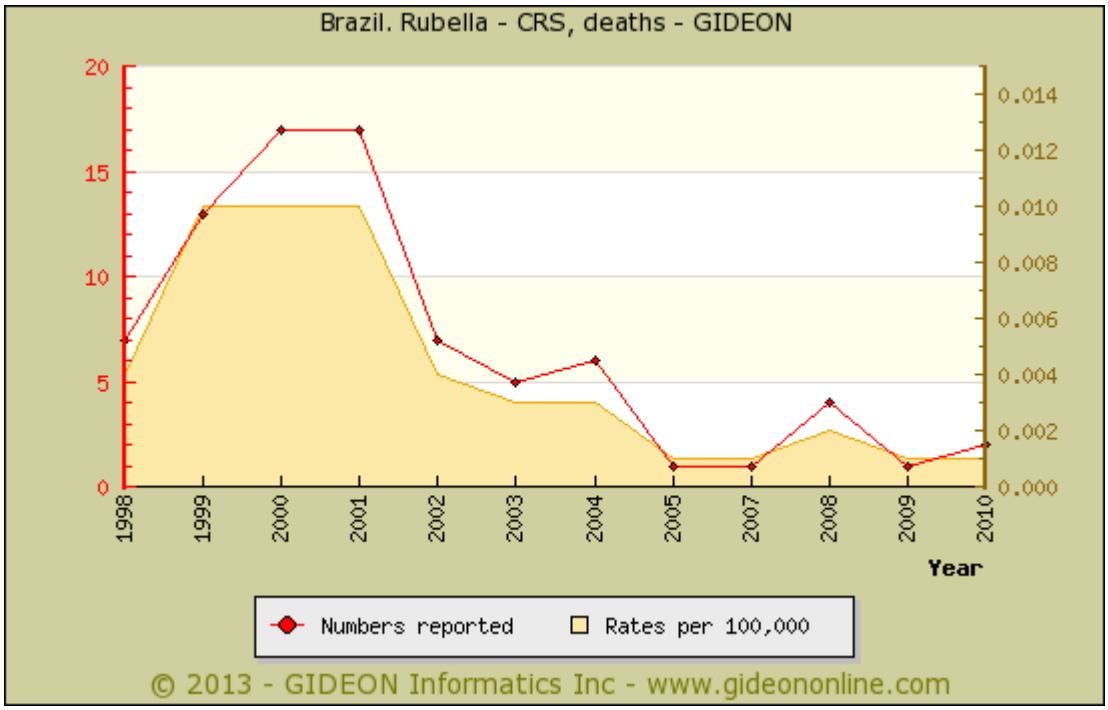
Graph: Brazil. Rubella, confirmed cases



Graph: Brazil. Rubella - CRS, cases

Notes:
1. Congenital rubella syndrome has been a notifiable disease since 1996.
2. The nationwide rate of congenital rubella is approximately 69.3 per 100,000 live births.
3. 876 suspected cases of CRS (132 confirmed) were reported during 1997 to 2000.
4. Rio Branco reported CRS rates of 4.3 per 1,000 live births during 2000 to 2001. [32]



Graph: Brazil. Rubella - CRS, deaths

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Notable outbreaks:

1968 - An outbreak of rubella was reported in Rio de Janeiro. [33]

1974 - An outbreak of rubella was reported in Rio de Janeiro. [34]

1981 - An outbreak of rubella was reported in Rio de Janeiro. [35]

1999 to 2000 - Outbreaks involved 20 of the country's 27 states. [36-38]

2005 - An outbreak (8 cases) was reported in Spain, among expatriates from Brazil. [39]

2007 - An outbreak (2,842 cases) was reported in Rio Grande do Sul. Eight cases of CRS were reported in the state in 2008. [40]

2007 to 2008 - An outbreak was reported in Sao Paulo. [41]

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Salmonellosis

Agent	BACTERIUM. Salmonella A facultative gram-negative bacillus
Reservoir	Mammal Bird Reptile
Vector	None
Vehicle	Food Milk Eggs Poultry Shellfish Meat Vegetables Fruit Fecal-oral Fly
Incubation Period	12h - 36h (range 6h - 5d)
Diagnostic Tests	Culture (stool, blood, infected tissue). Serology.
Typical Adult Therapy	Stool precautions. Therapy not indicated for uncomplicated diarrhea; if necessary, treat per antibiogram
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, chills & watery diarrhea 12 to 24 hours after ingestion of eggs, meat, poultry; fecal leucocytes present; fever resolves in 2 days; but diarrhea persists for up to 7 days (occasionally weeks).
Synonyms	Salmonellosen, Salmonellosi. ICD9: 003 ICD10: A02

Clinical

WHO Case definition for surveillance:
• An illness with the following symptoms: diarrhea, abdominal cramps, fever, vomiting and malaise.
Laboratory criteria for confirmation
• Isolation of *Salmonella* spp. from the stool or blood of a patient.
Case classification
• Suspected: An individual showing one or more of the clinical features.
• Confirmed: A suspected case with laboratory confirmation.

Acute infection:

Salmonella gastroenteritis is usually indistinguishable from that caused by other bacterial and viral pathogens. [1]
• Nausea, vomiting, and diarrhea begin 6 to 48 hours following ingestion of contaminated food or water.
• Incubation periods as long as 8 days have been reported. [2]
• Abdominal cramps and fever as high as 39 C are common.
• The diarrhea is usually characterized as loose, non-bloody stools of moderate volume.
• Voluminous diarrhea, bloody stools, and tenesmus may also occur.

The infection is usually self-limited.
• Fever resolves within 3 days, and diarrhea resolves within 3 to 7 days.
• Stool cultures may remain positive for 4 to 5 weeks after infection, and carriage may persist for as long as one year in fewer than 1% of cases. [3]
• Antibiotic treatment is reserved for unusual and complicated infections: septicemia, neonates, immunosuppressed patients, etc.

Complications:

The spectrum of extraintestinal salmonellosis is similar to that of other gram-negative bacterial infections: osteomyelitis [4-8], meningitis [9-11], endocarditis [12-14], etc.
• Endovascular infections are particularly common, and may result in aneurysms of the aorta and other large vessels. [15] [16]
• *Salmonella* osteomyelitis is common in children with underlying hemoglobinopathies. Pyomyositis has also been reported in such cases. [17]
• Septicemia is often described in patients with schistosomiasis [18-22], lymphoma, lupus erythematosus [23] [24], bartonellosis, malaria and hepatic cirrhosis.
• Rotavirus infection increases the risk of bacteremia in children with nontyphoid *Salmonella* gastroenteritis [25]
• Elderly patients are at risk for complicated or fatal infection. [26]
• Reactive arthritis has been reported in as many as 16.8% of cases [27-29]
• The risk for reactive arthritis following *Salmonella* infection [30] was 1.4/100,000 cases (United States, 2002 to 2004) [31]
• There is evidence that salmonellosis may increase the risk for later development of inflammatory bowel disease. [32]

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This disease is endemic or potentially endemic to all countries.

Salmonellosis in Brazil

Prevalence surveys:

38.4% of diarrhea episodes treated at a tertiary pediatric care facility in Salvador, Bahia (2005 publication) [33]
0.6% of urban children with diarrhea in Salvador (2000 to 2002) [34]
9.3% of children below age 6 years with diarrhea, in Porto Velho (Rondonia, 2006 publication) [35]
7.9% of children with diarrhea in northeastern Brazil (2008 publication) [36]
5% of HIV-positive patients with diarrhea, and 1.7% of control patients (southeastern Brazil, 2006 to 2007) [37]
3% of minimally processed leafy salads collected from retailers. (Sao Paulo, 2007 publication) [38]
10% of untreated and 0% of treated oysters from the Cananeia estuary. (Sao Paulo, 1998 to 1999) [39]
0% of raw milk samples from 4 important milk producing areas of Brazil (2008 publication) [40]
30% of fully-cooked ham sold in supermarkets (Forteleza, 2011 publication) [41]
8% of feral pigeons (*Columba livia*) in Jaboticabal (2011 publication) [42]
87% of Tegu Lizards (*Tupinambis merianae*, 2009 publication) [43]
14.5% of water samples from an integrated aquaculture system (2010 publication) [44]

Notable outbreaks:

1984 (publication year) - An outbreak of *Salmonella* agona infection was reported in a pediatric unit in Rio de Janeiro. [45]

1993 - An outbreak (211 cases) of *Salmonella enterica* infection in a school in Sao Paulo was caused by contaminated "pate" prepared with eggs, potatoes and bread. [46]

1994 (publication year) - An outbreak (6 cases) of *Salmonella* agona infection was reported in a pediatric unit in Rio de Janeiro. [47]

1998 to 1999 - An outbreak (140 cases) of *Salmonella enterica* subsp. *enterica* serotype Infantis was reported in a neonatal unit. [48]

1999 - An outbreak (8 cases) of *Salmonella enteritidis* infection at a health care facility in Sao Paulo was caused by contaminated lyophilized enteral nutrition. [49]

1999 - An outbreak (17 cases, 2 fatal) of *Salmonella enterica* serotype Newport infection in the United States was ascribed to mangoes imported from Brazil. [50]

2004 - An outbreak of salmonellosis was associated with a scientific meeting in Sao Paulo. [51]

2011 to 2012 - An outbreak (55 cases) of *Salmonella* Serotype Newport infection involving England (26 cases), Wales (3), Northern Ireland (1), Scotland (5), Ireland (5) and Germany (15) was related to contaminated water melon slices imported from Brazil. [52]

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Sarcocystosis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Eimeriida: Sarcocystis bovihominis or S. suihominis
Reservoir	Cattle Pig
Vector	None
Vehicle	Meat Water
Incubation Period	9d - 39d
Diagnostic Tests	Identification of cysts in stool.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Diarrhea and abdominal pain of varying severity; muscle pain and eosinophilia occasionally encountered.
Synonyms	Isospora hominis, Kudoa, Sarcocystiasis, Sarcocystis, Sarcosporidiosis. ICD9: 136.5 ICD10: A07.8

Clinical

Human infection follows ingestion of undercooked beef or pork.
- Clinical features are limited to abdominal pain, vomiting, moderate diarrhea or asymptomatic infection of muscle. [1] [2]
- Rare instances of myositis [3] with eosinophilia have also been reported.

This disease is endemic or potentially endemic to all countries.

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SARS and hCoV-EMC

Agent	Virus - RNA. Coronaviridae, Coronavirus.
Reservoir	Human Bat Civet Cat
Vector	None
Vehicle	Droplet, ? Fecal-oral
Incubation Period	3d - 5d (range 2d - 23d)
Diagnostic Tests	Identification of virus through PCR and direct immunofluorescence. Serology (ELISA)
Typical Adult Therapy	Supportive. Isolation (respiratory and other secretions).
Typical Pediatric Therapy	Supportive. Isolation (respiratory and other secretions).
Clinical Hints	Exposure to endemic area or patient; fever (>38 C), cough, respiratory difficulty, headache, myalgia, diarrhea, hepatic dysfunction, CPK elevation, thrombocytopenia. Case-fatality rate 10.9%.
Synonyms	human coronavirus Erasmus Medical Centre, London1 novel CoV 2012, Novel CoV 2012, SARG, Severe Acute Respiratory Syndrome, Sindroma Respiratoria Aguda, Sindrome respiratorio agudo grave, SRA, SRAS, Syndrome Respiratoire Aigu Severe. ICD9: 079.82 ICD10: U04.9

Clinical

The official Clinical Case Definition for SARS is as follows: [1-6]

A person with a history of:
Fever (>=38 °C)
AND One or more symptoms of lower respiratory tract illness (cough, difficulty breathing, shortness of breath)
AND Radiographic evidence of lung infiltrates consistent with pneumonia or RDS; OR autopsy findings consistent with the pathology of pneumonia or RDS without an identifiable cause.
AND No alternative diagnosis can fully explain the illness.

The Laboratory case definition of SARS is as follows:

A person with symptoms and signs that are clinically suggestive of SARS AND with positive laboratory findings for SARS-CoV based on one or more of the following diagnostic criteria:
a) PCR positive for SARS-CoV.
PCR positive using a validated method from:
At least two different clinical specimens (eg nasopharyngeal and stool)
OR The same clinical specimen collected on two or more occasions during the course of the illness (eg sequential nasopharyngeal aspirates)
OR Two different assays or repeat PCR using a new RNA extract from the original clinical sample on each occasion of testing.
b) Seroconversion by ELISA or IFA
Negative antibody test on acute serum followed by positive antibody test on convalescent phase serum tested in parallel
OR Fourfold or greater rise in antibody titer between acute and convalescent phase sera tested in parallel.
c) Virus isolation
Isolation in cell culture of SARS-CoV from any specimen AND PCR confirmation using a validated method.

Acute infection: [7]

The incubation period is generally 3 to 5 days, with a range of 2 to 10 days. [8]
• Most patients complain of headache, sore throat, myalgia and chills.
• Additional findings reported in some patients include rhinorrhea, chest pain, diarrhea [9] [10] , abdominal pain, vomiting and confusion.
• Initial reports described isolated instances of "rash."
• Lymphocytopenia, neutrophilia or neutropenia, thrombocytopenia, and elevations of CPK and hepatic transaminase levels are common.
• Rhabdomyolysis and renal failure were reported in some patients. [11] [12]

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After 3 to 7 days, lower respiratory symptoms occur, particularly a dry, nonproductive cough which may be accompanied by hypoxemia. [13]
• Features of 'atypical pneumonia' develop on the fourth to fifth days of illness. [14]
• Ten to twenty percent of patients described to date have developed signs of adult respiratory distress syndrome. [15]
• Chest roentgenograms reveal generalized interstitial or patchy infiltrates, with areas of consolidation in a few cases. [16-19]
• Sub-clinical and non-pneumonic infections are reported. [20] [21]

Complications: [22]
The overall case-fatality rate is 10.9%; but as high as 50% among those who develop severe respiratory illness.
• Multi-organ dysfunction (bowel, liver, kidney) may occur in severe cases. [23]
• Risk factors for mortality include age >60 years, history of contact with SARS patients within 2 weeks prior to illness onset, health occupation, inferior hospital ranking and longer interval to seeking medical care (longer than 1 day). [24]
• Advanced age, leucocytosis, detectable virus in nasopharyngeal aspirates (PCR) and elevated serum lactic dehydrogenase (LDH) levels have been associated with a poor prognosis.
• The disease appears to be milder among young children. [25]
• Some studies have suggested that concurrent infection by human metapneumoviruses or orthoreoviruses are associated with more severe illness and poor prognosis.
• Nearly 60% of survivors experienced psychiatric disorders (mainly PTSD or depression) [26]

WHO Case Definitions For hCoV-EMC Infection: [27-32]

Case Definition for Case Finding
Severe Respiratory Disease associated with NOVEL CORONAVIRUS
Interim case definition as of 25 September 2012

Patient under investigation:
A person with an acute respiratory infection, which may include fever (greater than or equal to 38 C, 100.4 F) and cough;
AND
suspicion of pulmonary parenchymal disease (e.g. pneumonia or acute respiratory distress syndrome (ARDS)) based on clinical or radiological evidence of consolidation;
AND
travel to or residence in an area where infection with novel coronavirus has recently been reported or where transmission could have occurred;
AND
is not already explained by any other infection or etiology, including all clinically indicated tests for community-acquired pneumonia according to local management guidelines.

Epidemiological criteria:
Close contact within the last 10 days before onset of illness
• with a probable or confirmed case of novel coronavirus infection while the case-contact was ill
OR
• travel to or residence in an area where infection with novel coronavirus has recently been reported or where transmission could have occurred.

Probable hCoV-EMC Case:
A person fitting the definition above of a "Patient Under Investigation with clinical, radiological, or histopathological evidence of pulmonary parenchyma disease (e.g. pneumonia or ARDS) but no possibility of laboratory confirmation either because the patient or samples are not available or there is no testing available for other respiratory infections,
AND
close contact with a laboratory confirmed case,
AND
is not already explained by any other infection or etiology, including all clinically indicated tests for community-acquired pneumonia according to local management guidelines.

Confirmed Novel Coronavirus Case:
A person with laboratory confirmation of infection with the novel coronavirus.

This disease is endemic or potentially endemic to 3 countries. Although SARS and hCoV-EMC is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

SARS and hCoV-EMC in Brazil

One 'probable' case (nonfatal) was reported during a multi-country outbreak - on June 9, 2003. [33]

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Scabies

Agent	PARASITE - Arthropod. Arachnid, Acarina (Mite), Sarcoptiae: Sarcoptes [Acarus] scabiei
Reservoir	Human
Vector	mite
Vehicle	Contact, including Sexual contact
Incubation Period	3d - 42d
Diagnostic Tests	Identification of mites in skin scrapings.
Typical Adult Therapy	Permethrin 5%. OR Lindane. OR Crotamiton 10% OR Ivermectin 150 to 200 ug/kg PO as single dose
Typical Pediatric Therapy	Permethrin 5%. OR Lindane. OR Crotamiton 10% OR Ivermectin 200 mcg/kg PO (> 15 kg body weight)
Clinical Hints	Intensely pruritic papules, vesicles and burrows - interdigital webs, wrists, elbows, axillae, perineal region, buttocks, penis; pruritus most intense at night; severe psoriaform infestation (Norwegian scabies) noted in debilitated patients.
Synonyms	Cheyletiella, Cheyletiella infestation, Escabiose, Escabiosis, Histiostomatid mites, Kratze, Mange, Ornithonyssus, Pyemotes, Sarcoptes scabiei, Sarna, Scabbia, Skabies, Tropical rat mite. ICD9: 133 ICD10: B86

Clinical

The lesions of scabies are usually symmetrical.
• Typical sites include the interdigital webs, buttocks, penis, scrotum, breasts and nipples, axillae and flexor surfaces of the wrists. [1]
• Pruritis is often worse at night.
• Skin lesions consist of burrows, papules or vesicles. [2]
• Exaggerated eczematous patches ('crusted', or Norwegian scabies) [3] [4] may be encountered • notably in institutions for Down's syndrome and leprosy. [5]
• Lesions in children are atypical and tend to involve the buttocks and perineum. [6]
• Complications include secondary infection and acute glomerulonephritis.

Otoacariasis due to Histiostomatid mites has been reported in Saudi Arabia. [7]

This disease is endemic or potentially endemic to all countries.

Scabies in Brazil

Prevalence surveys:
9.8% of persons in a rural community in Alagoas (2005)

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Scarlet fever

Agent	BACTERIUM. Streptococcus pyogenes A facultative gram-positive coccus
Reservoir	Human
Vector	None
Vehicle	Infected secretions Occasionally food
Incubation Period	1d - 4d
Diagnostic Tests	Typical clinical features associated with group A streptococcal pharyngitis.
Typical Adult Therapy	Benzathine Penicillin G 1.2 million units IM as single dose
Typical Pediatric Therapy	Benzathine Penicillin G : Weight <14kg: 300,000 units IM Weight 14 to 28kg: 600,000 units IM Weight >28kg: 1.2 million units IM
Clinical Hints	Overt pharyngitis followed within 24 to 48 hrs by florid erythematous rash.
Synonyms	Escarlatina, Lanhousha, Scarlattina, Scharlach. ICD9: 034.1 ICD10: A38

Clinical

Signs of streptococcal pharyngitis (fever, pharyngeal exudate and pain) are followed by the appearance of a rash within 12 to 24 hours.
• The exanthem appears initially on the trunk and spreads rapidly over the body to finally involve the extremities. [1]
• The exanthem has the texture of sandpaper, and blanches with pressure.
• Pruritis may be present.
• Facial flushing and circumoral pallor are characteristic.

The patient appears ill, with fever, tachycardia, pharyngitis, tender adenopathy and palatal petechiae.
• Within a few days, the rash becomes more intense along skin folds, producing lines of confluent petechiae (Pastia sign).
• The rash begins to fade within 3 to 4 days, with desquamation evident over the face, palms and fingers.
• Skin peeling may persist for as long as a month.

During the first 2 days of illness, the tongue has a white coat through which the red and edematous papillae project ('white strawberry tongue').
• The tongue later desquamates and becomes markedly reddened ('red strawberry tongue').

Complications are those associated with the streptococcal infection itself • spread to regional, retropharyngeal tissues, middle ears, and sinuses; acute rheumatic fever or post-streptococcal glomerulonephritis.
• Septic complications such as meningitis, pyogenic arthritis, and endocarditis, are occasionally encountered.

This disease is endemic or potentially endemic to all countries.

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Schistosomiasis - haematobium

Agent	PARASITE - Platyhelminthes, Trematoda. Strigeida, Schistosomatidae: Schistosoma haematobium
Reservoir	Snail (Bulinus, Planorbarius, Ferrissia) Rarely baboon or monkey
Vector	None
Vehicle	Water (skin contact)
Incubation Period	2w - 6w
Diagnostic Tests	Identification of ova in urine or stool. Serology. Antigen detection.
Typical Adult Therapy	Praziquantel 20 mg/kg PO BID X 1 day
Typical Pediatric Therapy	As for adult
Clinical Hints	Early urticaria, fever and eosinophilia; later, dysuria, hematuria and obstructive nephropathy; often complicated by bladder cancer in advanced cases; parasite may survive for decades in human host.
Synonyms	Bilharziasis, urinary, Egyptian hematuria, Katayama fever [1], Schistosoma guineensis, Schistosoma haematobium, Schistosomal hematuria, Schistosomiasis, Vesicle bilharziasis. ICD9: 120.0 ICD10: B65.0

Clinical

WHO Case definition for surveillance:
Endemic areas (moderate or high prevalence)
• Suspected: Not applicable.
• Probable: Not applicable.
• Confirmed: A person with visible hematuria or with positive reagent strip for hematuria or with eggs of *S. haematobium* in urine (microscope).
Non-endemic areas and areas of low prevalence
• Suspected: A person with visible hematuria or with positive reagent strip for hematuria.
• Probable: Not applicable.
• Confirmed: A person with eggs of *S. haematobium* in urine (microscope).

The clinical features caused by *Schistosoma* species infecting man are similar [1] , and will be discussed together.

Acute infection:
Within 24 hours of penetration by cercariae, the patient develops a pruritic papular skin rash known as swimmer's itch. [The more overt form of Cercarial dermatitis associated with avian schistosomes is discussed elsewhere in this module.]
• One to two months after exposure, an overt systemic illness known as Katayama fever (named for Katayama district, Hiroshima, Japan) begins, heralded by acute onset of fever, chills, diaphoresis, headache, and cough. [2]
• The liver, spleen, and lymph nodes are enlarged, and eosinophilia is present.
• Rare instances of myocarditis have been reported during acute schistosomiasis. [3] [4]
• Although deaths have been described at this point (notably in *S. japonicum* infection) these findings subside within a few weeks in most cases.

Chronic schistosomiasis:
The likelihood of progression to chronic schistosomiasis is related to the extent of infestation.
• Chronic schistosomiasis caused by *S. mansoni*, *S. japonicum*, or *S. mekongi* is characterized by fatigue, abdominal pain and intermittent diarrhea or dysentery.
• Blood loss from intestinal ulcerations may lead to moderate anemia.
• In *S. mansoni*, *S. japonicum*, and *S. mekongi* infections, ova remain in the venous portal circulation and are carried to the liver where they produce granulomata and fibrosis [5] , and block portal blood flow.
• Colonic polyposis is has been associated with infection by *S. mansoni*, *S. japonicum*, and *S. intercalatum*. [6] Retroperitoneal fibrosis has been reported with *S. japonicum* infection. [7]
• Portal hypertension and portosystemic collateral circulation result.
• Although liver function tests remain normal for a long time, hepatosplenomegaly and variceal hemorrhage develop.
• The spleen is firm and may reach massive size.
• Fatal hematemesis is unusual.
• Laboratory tests reveal moderate eosinophilia and anemia related to blood loss and hypersplenism.
• Eventually, hepatic function deteriorates, with late ascites and jaundice.

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In *S. haematobium* infection, ova are located in the bladder and ureters, leading to granuloma formation, inflammation, hematuria, ureteral obstruction, secondary infection and often carcinoma of the bladder. [8-10] Ova are also commonly present in the seminal vesicles and prostate. [11]
• Areas of chronic inflammation, fibrous tissue and calcifications ("sandy patches") in the genital mucosa and bladder contain ova, and are considered pathognomonic for *S. haematobium* infection. [12]
• Genital lesions may present a risk factor for acquisition of HIV infection [13] ; and schistosomal co-infection may accelerate HIV disease progression and facilitate viral transmission to sexual partners. [14]
• Terminal hematuria and dysuria are common symptoms.
• Although best known for damage to the urinary bladder and ureters, the female genitalia are involved in 50% to 70% of women with *S. haematobium* infection • resulting in vaginal deformities and fistulae [15] , hypogonadism, ectopic pregnancy, miscarriage and malignancy. [16-19] *Schistosoma mansoni* is implicated in the etiology of appendicitis [20] , and membranoproliferative glomerulonephritis and amyloidosis [21] ; and may also involve the fallopian tubes [22] [23] or cause ovarian [24] or testicular pseudotumor [25] and acute abdomen associated with granulomatous peritonitis [26] or panniculitis. [27]
• Reinfection or inadequately treated infection may lead to extra-anogenital bilharziasis cutanea tarda. Lesion may typically complicate pre-existing skin conditions. [28]
• Proctitis is occasionally encountered. [29]

S. intercalatum infection is characterized by abdominal pain and bloody diarrhea.

S. mekongi is an important cause of hepatomegaly in endemic areas.

Complications:
The following are some of the many complications described in chronic schistosomiasis.
• Pulmonary schistosomiasis is manifested by symptoms and signs of right ventricular congestion related to blockage of pulmonary capillaries by ova in the course of hepatosplenic schistosomiasis. [30-32]
• Central nervous system schistosomiasis is manifested as delirium, coma, seizures, dysphasia, visual impairment, ataxia, a cerebal mass, generalized encephalopathy, cerebral vasculitis with stroke, or focal epilepsy (notably in *S. japonicum* infection). [33-45]
• Granulomata of *S. haematobium* and *S. mansoni* may involve the spinal cord (most commonly the cauda equina or conus medularis) , producing transverse myelitis. [46-59] Rare instances of cerebral infection by *S. haematobium* have been reported. [60] *Schistosoma mansoni* infection may occasionally involve the bladder, mimicking *S. haematobium* infection or malignancy. [61] *S. mansoni* infection has been implicated in cases of colo-rectal cancer. [62]
• *Salmonella* bacteremia is often reported among persons with hepato-splenic schistosomiasis. [63-67]

This disease is endemic or potentially endemic to 58 countries. Although Schistosomiasis - haematobium is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Schistosomiasis - haematobium in Brazil

Notable outbreaks:

1994 - An outbreak (55 cases) was reported among Brazilians who had attended a peace mission in Mozambique. [68] [69]

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Schistosomiasis - mansoni

Agent	PARASITE - Platyhelminthes, Trematoda. Strigeida, Schistosomatidae: Schistosoma mansoni
Reservoir	Snail (Biomphalaria) Dog Cat Pig Cattle Rodent Horse Non-human primate
Vector	None
Vehicle	Water (skin contact)
Incubation Period	2w - 6w
Diagnostic Tests	Identification of ova in stool or biopsy specimens. Serology. Antigen detection.
Typical Adult Therapy	Praziquantel 20 mg/kg PO BID X one day OR Oxamniquine 15 mg PO X one dose
Typical Pediatric Therapy	Praziquantel 20 mg/kg PO BID X one day OR Oxamniquine 10 mg PO BID X one day
Clinical Hints	Early urticaria, fever and eosinophilia; later, hepatosplenomegaly and portal hypertension; parasite may survive for decades in human host.
Synonyms	Bilharziasis, intestinal, Katayama fever [3], Schistosoma mansoni. ICD9: 120.1 ICD10: B65.1

Clinical

WHO Case definition for surveillance (all forms of intestinal schistosomiasis):
Endemic areas (moderate or high prevalence)
• Suspected: A person with chronic or recurrent intestinal symptoms (blood in stool, bloody diarrhea, diarrhea, abdominal pains) or, at a later stage, hepatosplenomegaly.
• Probable: A person who meets the criteria for presumptive treatment, according to the locally applicable diagnostic algorithms.
• Confirmed: A person with eggs of *S. mansoni*, or *S. japonicum/mekongi* in stools (microscope).
Non-endemic areas and areas of low prevalence
• Suspected: A person with chronic or recurrent intestinal symptoms (blood in stool, bloody diarrhea, diarrhea, abdominal pains) or, at a later stage, hepatosplenomegaly.
• Probable: Not applicable.
• Confirmed: A person with eggs of *S. mansoni* or *S. japonicum* in stools (microscope). A person with positive reaction to immunoblot test.

The clinical features caused by *Schistosoma* species infecting man are similar [1] , will be discussed together.

Acute infection:

Within 24 hours of penetration by cercariae, the patient develops a pruritic papular skin rash known as swimmer's itch. [The more overt form of Cercarial dermatitis associated with avian schistosomes is discussed elsewhere in this module.]
• One to two months after exposure, an overt systemic illness known as Katayama fever (named for Katayama district, Hiroshima, Japan) begins, heralded by acute onset of fever, chills, diaphoresis, headache, and cough. [2]
• The liver, spleen, and lymph nodes are enlarged, and eosinophilia is present.
• Although deaths have been described at this point (notably in *S. japonicum* infection) these findings subside within a few weeks in most cases.

Chronic schistosomiasis:

The likelihood of progression to chronic schistosomiasis is related to the extent of infestation.
• Chronic schistosomiasis caused by *S. mansoni*, *S. japonicum*, or *S. mekongi* is characterized by fatigue, abdominal pain and intermittent diarrhea or dysentery.
• Colonic polyposis is has been associated with infection by *S. mansoni*, *S. japonicum*, and *S. intercalatum*. [3] Retroperitoneal fibrosis has been reported with *S. japonicum* infection. [4]
• Blood loss from intestinal ulcerations may lead to moderate anemia.
• In *S. mansoni*, *S. japonicum*, and *S. mekongi* infections, ova remain in the venous portal circulation and are carried to the liver where they produce granulomata and fibrosis [5] , and block portal blood flow.
• Portal hypertension and portosystemic collateral circulation result.
• Although liver function tests remain normal for a long time, hepatosplenomegaly and variceal hemorrhage develop.
• The spleen is firm and may reach massive size.
• Fatal hematemesis is unusual.
• Laboratory tests reveal moderate eosinophilia and anemia related to blood loss and hypersplenism.

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• Eventually, hepatic function deteriorates, with late ascites and jaundice.

In *S. haematobium* infection, ova are located in the bladder and ureters, leading to granuloma formation, inflammation, hematuria, ureteral obstruction, secondary infection and often carcinoma of the bladder. [6-8] Ova are also commonly present in the seminal vesicles and prostate. [9]
• Genital lesions may present a risk factor for acquisition of HIV infection [10] ; and schistosomal co-infection may accelerate HIV disease progression and facilitate viral transmission to sexual partners. [11]
• Terminal hematuria and dysuria are common symptoms.

S. intercalatum infection is characterized by abdominal pain and bloody diarrhea.

S. mekongi is an important cause of hepatomegaly in endemic areas.

Complications:
The following are some of the many complications described in chronic schistosomiasis.
• Pulmonary schistosomiasis is manifested by symptoms and signs of right ventricular congestion related to blockage of pulmonary capillaries by ova in the course of hepatosplenic schistosomiasis. [12-14]
• Central nervous system schistosomiasis is manifested as delirium, coma, seizures, dysphasia, visual impairment, ataxia, a cerebal mass, generalized encephalopathy, cerebral vasculitis with stroke, or focal epilepsy (notably in *S. japonicum* infection). [15-27]
• Granulomata of *S. haematobium* and *S. mansoni* may involve the spinal cord (most commonly the cauda equina or conus medularis) , producing transverse myelitis. [28-41] Rare instances of cerebral infection by *S. haematobium* have been reported. [42] *Schistosoma mansoni* infection may occasionally involve the bladder, mimicking *S. haematobium* infection or malignancy. [43] *S. mansoni* infection has been implicated in cases of colo-rectal cancer. [44]
• Although best known for damage to the urinary bladder and ureters, the female genitalia are involved in 50% to 70% of women with *S. haematobium* infection • resulting in vaginal deformities and fistulae [45] , hypogonadism, ectopic pregnancy, miscarriage and malignancy. [46-49] *Schistosoma mansoni* is implicated in the etiology of appendicitis [50] , and membranoproliferative glomerulonephritis and amyloidosis [51] ; and may also involve the fallopian tubes [52] [53] or cause ovarian [54] or testicular pseudotumor [55] and acute abdomen associated with granulomatous peritonitis [56] or panniculitis. [57]
• *Salmonella* bacteremia is often reported among persons with hepato-splenic schistosomiasis. [58-62]

This disease is endemic or potentially endemic to 59 countries.

Schistosomiasis - mansoni in Brazil

Time and Place:
- Schistosomiasis is endemic to Para, Maranhao [63] , Piaui, Ceara, Rio Grande do Norte, Paraiba, Pernambuco [64-66] , Alagoas, Sergipe [67] , Bahia [68] , Minas Gerais [69-71] , Espirito Santo, Rio de Janeiro [72] , Parana, Santa Catarina and Distrito Federale.
- Endemic disease has recently spread into Pernambuco.

11% of the land mass lies within the endemic zone.

The number of carriers was estimated at 7.1 million (4.5% of the population) in 1996; 6.3 million (3.9% of the population) in 1997.

Prevalence surveys:
2.2% of school children in Salvador (1997)
44.5% of children in rural Bahia (2006 publication) [73]
37.7% of persons in an endemic area of Bahia (2012 publication) [74]
57.4% of patients with asthma in Bahia (2012 publication) [75]
0% of urban children with diarrhea in Salvador (2000 to 2002) [76]
30.2% of children in the Suburbio Ferrovaiario region of Salvador (2006 publication) [77]
36.73% of school children in Americaninhas, Minas Gerais (2008 publication) [78]
24.9% of students ages 7 to 15 years in Alagoas (2010 publication) [79]
38.5% of individuals in a community in Minas Gerais were coinfected with hookworm and *Schistosoma mansoni* (2009 publication) [80]
13.3% of school children in the rainforest zone of Pernambuco (2004 to 2006) [81]
82.1% of persons in Sao Lourenco da Mata, rural Recife (Pernambuco, 1990 publication) [82]
50.3% of persons in southeastern Brazil (2008 publication) [83]
20% of urban residents of Belo Horizonte (1996) A paper describing an outbreak (17 cases) of acute infection from a swimming pool in Belo Horizonte was published in 2004.
60.8% of the population of Capitao Andrade (Minas Gerais) in 1973; 36.2% in 1984; 27.3% in 1994; 19.4% in 2003. [84]

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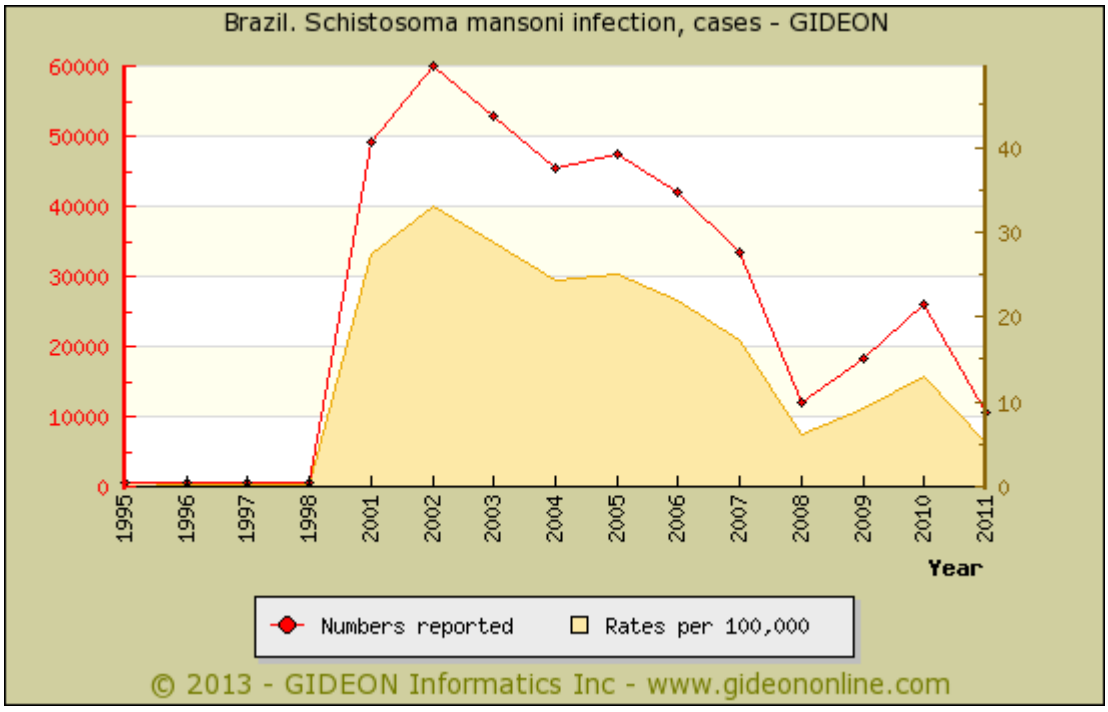
16% of persons in a low-endemic area of Ceara (2012 publication) [85]
11.6% of the population of Sumidouro, Rio de Janeiro in 1995; 8.8% in 1996; 12.2% in 1998; 5.9% in 1999; 3.2% in 2000 [86]
1% of the population of Sabugo, Paracambi, Rio de Janeiro (2010 publication) [87]
13.6% of persons in Sergipe in 2005; 11.2% in 2006; 11.8% in 2007; 10.6% in 2008 [88]
5.3% of persons in endemic areas of Minas Gerais (1984 to 2007) [89]
45.3% of persons in Americaninhas, Minas Gerais (2004) [90]
45.2% of persons in Americaninhas, Minas Gerais (2004) [91]
34.7% of persons in Piau and Coronel Pacheco, Minas Gerais (stool PCR, 2012 publication) [92]
3.2% of persons and 8.3% of *Biomphalaria glabrata* snails - in Tutoia, Maranhao (2008) [93]
4% of renal biopsies in Salvador (2003 to 2009) [94]
5.7% of *Biomphalaria* snails in Jaboatao dos Guararapes, Pernambuco (2006 to 2007) [95]
32.1% of *Biomphalaria* snails in 2000, 15.7% in 2010 - in Pernambuco [96]
33.3% of *Biomphalaria glabrata*, 47.2% of *Biomphalaria tenagophila* and 8.3% of *Biomphalaria straminea* - in Minas Gerais (2005 to 2009) [97]

Seroprevalence surveys:
48% of persons from Rio de Janeiro (2007 publication) [98] Only 20% of seropositive persons had ova in stool

30% of patients with hepatosplenic schistosomiasis were found to be seropositive for anti-HBc and/or HBsAg, and 7.4% for anti-HCV (Recife, 2008) [99]



Graph: Brazil. *Schistosoma mansoni* infection, cases

Notes:
1. 164,938 cases were reported in 1980.

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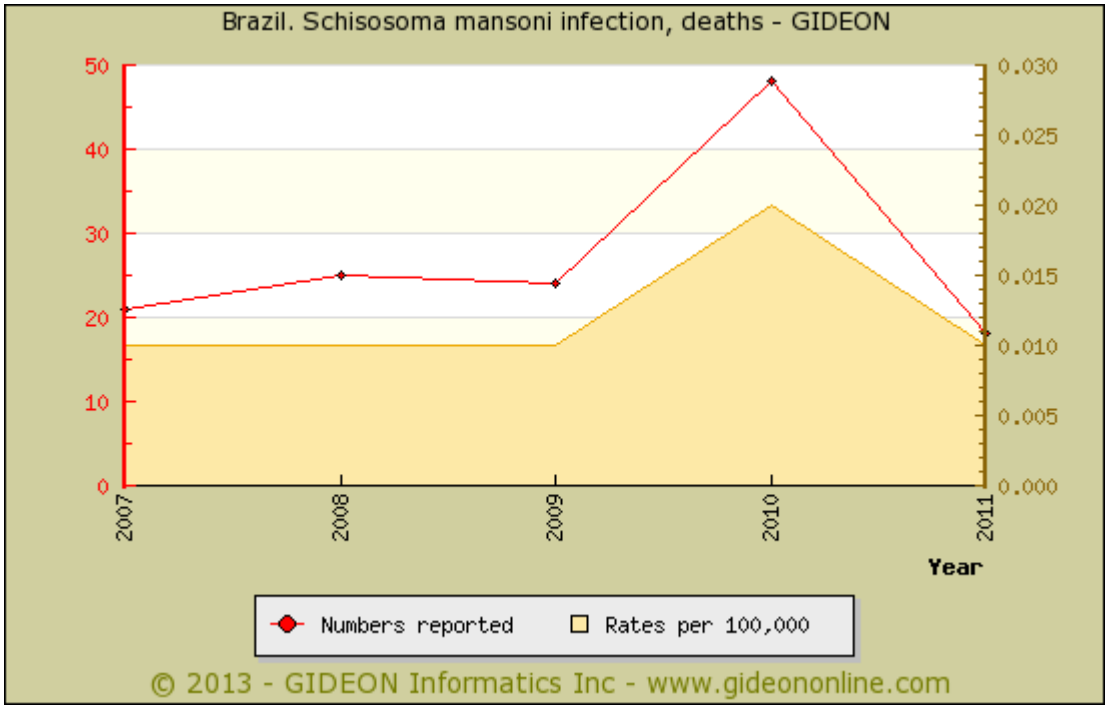

Graph: Brazil. *Schistosoma mansoni* infection, deaths

Notes:
1. 2,248 fatal cases were reported during 1991 to 1994.

Reservoirs:
- 15 species of mammals (most rodents) in this country are naturally infected, including water rats (*Nectomys squamipes*) in Rio de Janeiro. [100]
- Rare instances of infection in cattle have been reported. [101]
- The local snail reservoirs are *Biomphalaria glabrata* [102] , *Bi. tenagophila* [103] and *Bi. straminea* (*Tropicorbis centimetralis*). [104-106]

Notable outbreaks:

2000 - An outbreak (662 cases) was reported in Pernambuco. [107] [108]
2003 (publication year) - An outbreak (17 cases) was reported in metropolitan Belo Horizonte, Minas Gerais. [109]
2006 - An outbreak (32 cases) was reported among tourists in Belo Horizonte. [110]

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Septic arthritis

Agent	BACTERIUM or FUNGUS. Gram positive cocci most common; gram negative bacilli, gonococci, mycobacteria, fungi, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Smear and culture of joint fluid. Cytological and chemical analysis of joint fluid also useful.
Typical Adult Therapy	Antimicrobial agent(s) directed at known or likely pathogen
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever (60% to 80%) associated with swelling, erythema and tenderness (usually single joint, most commonly a knee; elbow or ankle in child); mean fluid leukocyte count in acute bacterial forms = 50,000 / cu mm.
Synonyms	

Clinical

Most cases present with fever, malaise and local findings of warmth, swelling and decreased range of motion. [1] [2]
- Lack of erythema and local warmth are not uncommon.
- The most commonly involved joints are the knee and hip, followed by the shoulder and ankle. [3]
- Non-gonococcal arthritis is mono-articular in 80% to 90% of cases.
- Infection of the costochondral, sternoclavicular and sacroiliac joints is common in intravenous drug users.

Synovial fluid demonstrates low viscosity and turbidity.
- Leucocyte counts usually exceed 50,000 per cu mm.
- Note that leucocytosis, low glucose and high lactate levels are also encountered in some non-infectious forms of arthritis.
- Gram stains are positive in 50% of cases, and cultures in 90%.

Etiological associations:
- Adult below age 30: *Neisseria gonorrhoeae* (often monoarticular involving knee)
- Associated rash: Lyme disease, gonococcemia (often monoarticular, involving knee)
- Child below age 5 years: *Haemophilus influenzae*, *Staphylococcus aureus*, *Streptococcus* spp.
- Chronic arthritis: Tuberculosis, Mycobacteria • nontuberculous, Sporotrichosis and other fungi
- Hematogenous infection: *Staphylococcus aureus*, *Streptococcus pyogenes*
- Injecting drug user: *Pseudomonas aeruginosa* (often sternoclavicular or sacroiliac)
- Traumatic injury to joint: *Staphylococcus aureus*, Enterobacteriaceae, *Pseudomonas aeruginosa*

This disease is endemic or potentially endemic to all countries.

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Septicemia - bacterial

Agent	BACTERIUM. Escherichia coli, Staphylococcus aureus, facultative gram negative bacilli, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Culture of blood and sepsis source.
Typical Adult Therapy	Antimicrobial agent(s) directed at known or likely pathogen
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, rigors, leukocytosis, tachypnea, mental changes; hypotension, acidosis and bleeding diathesis herald septic shock; further signs (eg, urinary infection, phlebitis, etc) may point to the source of infection .
Synonyms	Sepsis, Septicaemia, Septicemia, Septicemie, Septikemie, Setticemia. ICD9: 036.2,036.3,038 ICD10: A40,A41

Clinical

Bacterial septicemia is defined as the presence of signs and symptoms related to bacteremia. [1]
• The clinical spectrum and severity of disease are largely determined by the infecting species, underlying diseases and source of infection.
• Most patients present with fever, tachycardia and leucocytosis, in addition to signs and symptoms referable to a primary infectious focus (eg, urinary tract, abdominal infection, endocarditis, etc).

This disease is endemic or potentially endemic to all countries.

Septicemia - bacterial in Brazil

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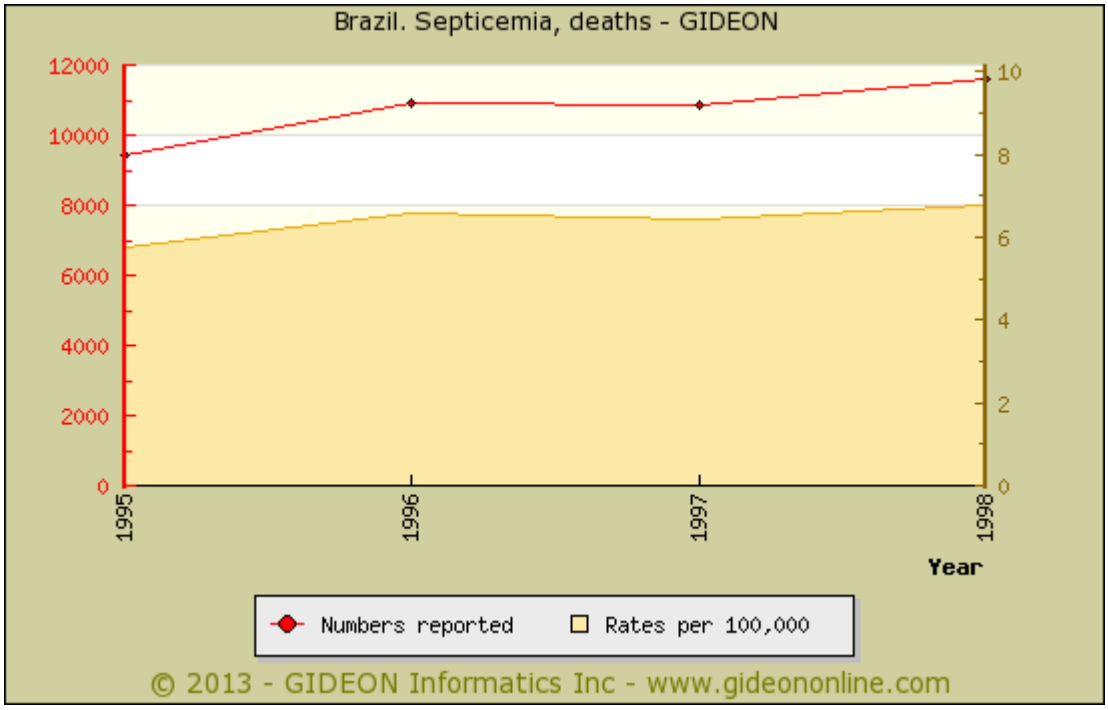

Graph: Brazil. Septicemia, deaths

Bacterial septicemia was the cause of death in 4% of autopsied AIDS patients (Amazonas, 1996 to 2003) [2]

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Shigellosis

Agent	BACTERIUM. Shigella sonnei, Shigella flexneri, Shigella boydii or Shigella dysenteriae A facultative gram-negative bacillus
Reservoir	Human Non-human primate
Vector	None
Vehicle	Fecal-oral Water Dairy products Fomite Fly Vegetables
Incubation Period	48h - 72h (range 7h - 1w)
Diagnostic Tests	Stool culture.
Typical Adult Therapy	Stool precautions. Choice of antimicrobial agent based on regional susceptibility patterns. Continue treatment for five days
Typical Pediatric Therapy	As for adult
Clinical Hints	Watery or bloody diarrhea, tenesmus, abdominal pain and headache; colonic hyperemia and abundant fecal leucocytes are present; usually resolves in 3 days (may persist for up to 14); case fatality rate = 1%.
Synonyms	Bacillaire dysenterie, Bacillary dysentery, Dissenteria batterica, Dysenteria bacillaris, Leptospirenerkrankung, Ruhr, Shigella, Shigellose, Shigelose, Ubertragbare Ruhr. ICD9: 004 ICD10: A03

Clinical

Acute infection:
Approximately 50% of infections are limited to transient fever or self-limited diarrhea.
• 50% of patients progress to bloody diarrhea and dysentery. [1]
• Fever may rise rapidly to 40 C, and febrile seizures are common in children.
• Seizures rarely recur or result in neurological sequelae.
• Dysentery is characterized by passage of 10 to 30 small-volume stools consisting of blood, mucus, and pus.
• Abdominal cramps and tenesmus are noted, and straining may lead to rectal prolapse, notably in young children. [2]
• On endoscopy, the colonic mucosa is hemorrhagic, with mucous discharge and focal ulcerations. Most lesions are in the distal colon.

Complications:
Patients with mild disease generally recover without specific therapy in two to seven days.
• Severe shigellosis can progress to toxic dilatation or perforation of the colon, which may be fatal.
• Mild dehydration is common, and protein-losing enteropathy can occur with severe disease.
• Complications are most commonly described in developing countries and are related both to the relative prevalence of *S. dysenteriae* type 1 and *S. flexneri*, and the poor nutritional state of the local populations.
• *Shigella* bacteremia is not uncommon, and is associated with increased mortality, particularly among infants below one year of age and persons with protein-energy malnutrition.
• Hemolytic-uremic syndrome (HUS) may complicate shigellosis due to *S. dysenteriae* type 1, and usually develops toward the end of the first week of shigellosis. [3-5] The case-fatality rate in these cases is 36%. [6]
• Profound hyponatremia and hypoglycemia may occur.
• Other complications include encephalopathy [7], seizures, altered consciousness, and bizarre posturing, pneumonia [8], meningitis, vaginitis, keratoconjunctivitis [9], pneumonia and "rose spots."
• Reiter's syndrome is seen in patients having histocompatibility antigen HLA-B27. [10] [11]
• Reactive arthritis follows 7% to 10% of *Shigella* infections. [12-15]

This disease is endemic or potentially endemic to all countries.

Shigellosis in Brazil

Shigella flexneri accounts for 52.7% of shigellosis, *S. sonnei* 44.2%, *S. boydii* 2.3%, and *S. dysenteriae* 0.6% (Rio de Janeiro, 1999 to 2004). [16]

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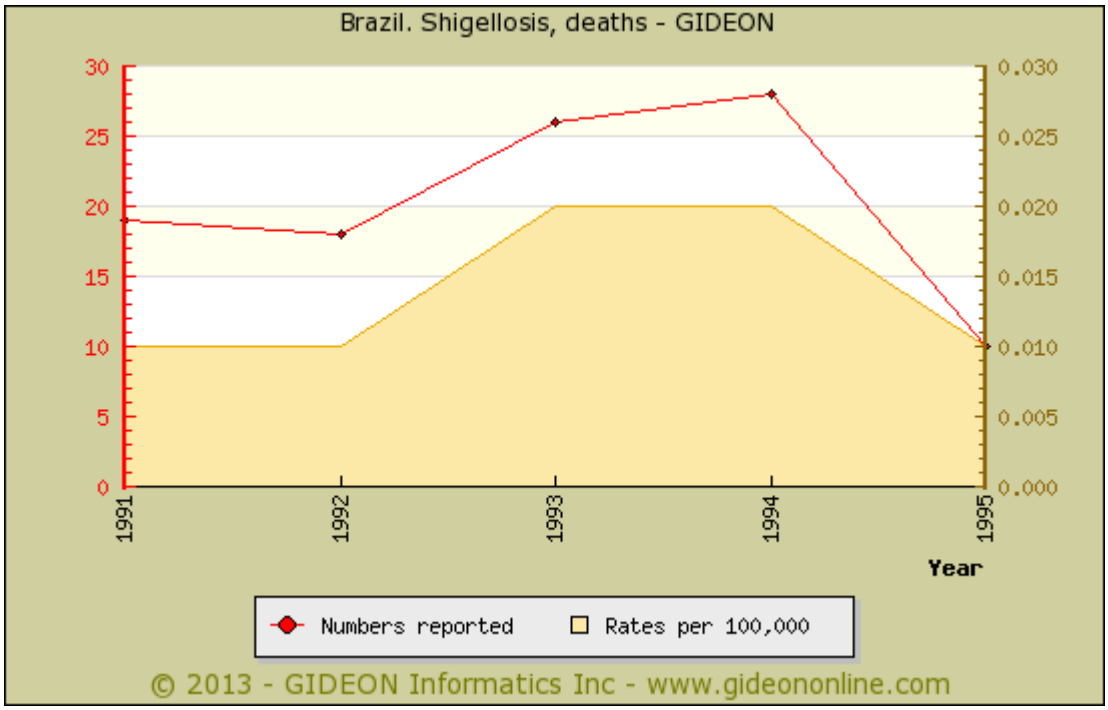

Graph: Brazil. Shigellosis, deaths

Prevalence surveys:

54.3% of diarrhea episodes treated at a tertiary pediatric care facility in Salvador, Bahia (2005 publication) [17]
5% of acute diarrheal disease among urban infants in Sao Paulo (1991 publication) [18]
11.6% of infants hospitalized with acute diarrhea (Natal, 2008 publication) [19]
0.6% of urban children with diarrhea in Salvador (2000 to 2002) [20]
4.1% of children with diarrhea in northeastern Brazil (2008 publication) [21]
5.1% of children below age 6 years with diarrhea, in Porto Velho (Rondonia, 2006 publication) [22]
10.4% of children ages <=5 years in Teresina, Piaui State (2004 to 2007) [23]
8% of acute diarrhea episodes in northeastern Brazil (1983 publication) [24]
0% of oysters from the Cananeia estuary. (Sao Paulo, 1998 to 1999) [25]

Notable outbreaks:

1980 - An outbreak of *Shigella sonnei* and Rotavirus infections was reported in a private school in Rio de Janeiro. [26]

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Sinusitis

Agent	BACTERIUM. Various (Haemophilus influenzae & Streptococcus pneumoniae in most acute cases)
Reservoir	Human
Vector	None
Vehicle	None
Incubation Period	Variable
Diagnostic Tests	Imaging techniques. Culture of sinus drainage.
Typical Adult Therapy	Antimicrobial agent(s) directed at likely pathogens. Drainage as indicated
Typical Pediatric Therapy	As for adult
Clinical Hints	Sinusitis often follows upper respiration infections; headache, fever and local tenderness are common, however the precise presentation varies with patient age and anatomic localization.
Synonyms	Acute sinusitis, Mastoidite, Mastoiditis, Rhinosinusitis, Sinusite. ICD9: 473.9,383.0,461 ICD10: H70,J01

Clinical

Acute community-acquired bacterial sinusitis is usually superimposed on preexisting viral sinusitis.
• In most cases, it is not possible to distinguish between viral and bacterial infections.
• Sneezing, nasal discharge and obstruction, facial pressure and headache are common in both conditions. [1]
• Fever of 38C or more, facial pain, and erythema occur may occasionally herald bacterial infections.
• The nasal discharge may be colored in both viral and bacterial sinusitis.
• Cough and hyposmia may also be present.

Sinusitis following dental infection is associated with molar pain and a foul breath odor.
• Sphenoid sinusitis is associated with severe frontal, temporal, or retroorbital headache that radiates to the occipital region; and hypesthesia or hyperesthesia of the ophthalmic or maxillary dermatomes of the fifth cranial nerve.
• Lethargy and findings suggestive of cavernous sinus or cortical vein thrombosis, orbital cellulitis or orbital abscess may also be present.
• In severe cases of frontal sinusitis, pus may collect under the periosteum of the frontal bone resulting in a 'Pott puffy tumor.'

Rare instances of toxic shock syndrome have followed sinusitis. [2]

This disease is endemic or potentially endemic to all countries.

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Smallpox

Agent	VIRUS - DNA. Poxviridae, Orthopoxvirus: Variola virus
Reservoir	Human
Vector	None
Vehicle	Contact Infected secretions Fomite
Incubation Period	7d - 17d
Diagnostic Tests	Culture and electron microscopy of skin lesions. Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Isolation; supportive. Cidofovir is effective in vitro
Typical Pediatric Therapy	As for adult
Vaccine	Smallpox
Clinical Hints	Fever, myalgia, headache, pustular or hemorrhagic rash; disease resolves in 2 to 3 weeks; case-fatality rate = 25% for severe form (variola major) and 1% for minor form; last naturally-acquired case reported in Somalia in 1977.
Synonyms	Alastrim, Eczema vaccinatum, Kopper, Smallpox, Vailo, Variola, Variola minor, Varioloid. ICD9: 050 ICD10: B03

Clinical

Acute infection: [1] [2]
12 to 14 days after exposure (range 7 to 17 days), the patient experiences a 2 to 3 day prodrome of high fever, malaise, prostration and severe headache and backache.
• This "preeruptive stage" is followed by the appearance of a maculopapular rash (i.e., eruptive stage) that progresses to papules within one to two days.
• Vesicles appear on the fourth or fifth day; pustules by the seventh day; and scab lesions on the fourteenth day.
• The rash first appears on the oral mucosa, face, and forearms; and then spreads to the trunk and legs. [3]
• The palms and soles may also be involved.
• Skin lesions are deeply embedded in the dermis and feel like firm round objects in the skin.
• As the lesions heal, the scabs separate and pitted scarring gradually develops.
• Patients are most infectious during the first week of the rash when the oral mucosa lesions ulcerate and release large amounts of virus into the saliva.
• A patient is no longer infectious after all scabs have separated (3 to 4 weeks after the onset of the rash).
• Rare instances of bone involvement (osteomyelitis variolosa) are described. [4-6]
• During the smallpox era, overall mortality rates were approximately 30%.

Other less common but more severe forms of smallpox include
• a) flat-type smallpox (mortality rate over 96%) characterized by severe toxemia and flat, velvety, confluent lesions that did not progress to the pustular stage or scaring
• b) hemorrhagic smallpox, characterized by severe prodromal symptoms, toxemia, and a hemorrhagic rash.

Hemorrhagic smallpox is uniformly fatal and occur among all ages and in both sexes, but pregnant women appear to be unusually susceptible. [7] [8]
• Illness usually begins with a somewhat shorter incubation period and is characterized by high fever and pain in the head, back, and abdomen.
• Soon thereafter, a dusky erythema develops, followed by petechiae and frank hemorrhages into the skin and mucous membranes.
• Death usually occurs by the fifth or sixth day after onset of rash.

Variola minor is generally less severe, with fewer constitutional symptoms and a more sparse rash.
• A milder form of disease is also seen among those who have residual immunity from previous vaccination.
• In partially immune persons, the rash tends to be atypical and more scant and the evolution of the lesions more rapid.

Disseminated herpes simplex in patients with eczema (Eczema herpeticum) may resemble smallpox. [9]

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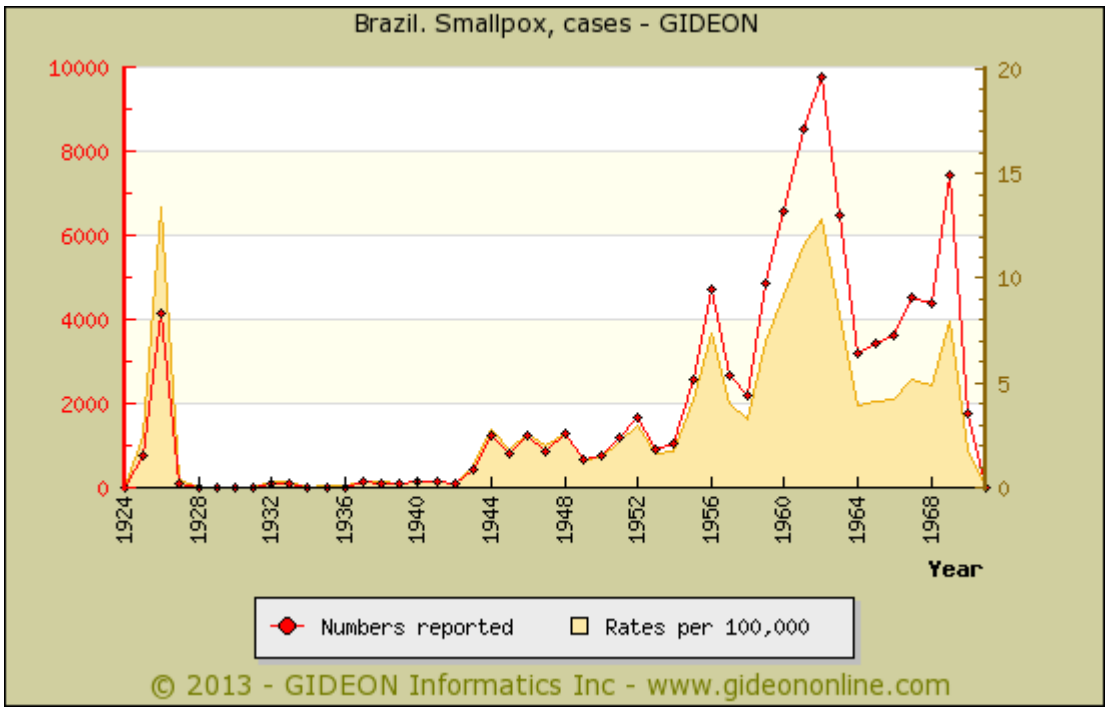
This disease is not currently endemic to any country. Although Smallpox is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Smallpox in Brazil

Smallpox was first recognized in Brazil in 1563. [10]

Outbreaks of smallpox were reported in 1904, 1908, 1911, 1944 to 1945, 1955 to 1956, and 1959 to 1962.

Reviews of smallpox eradication in Brazil - see references [11-13]



Graph: Brazil. Smallpox, cases

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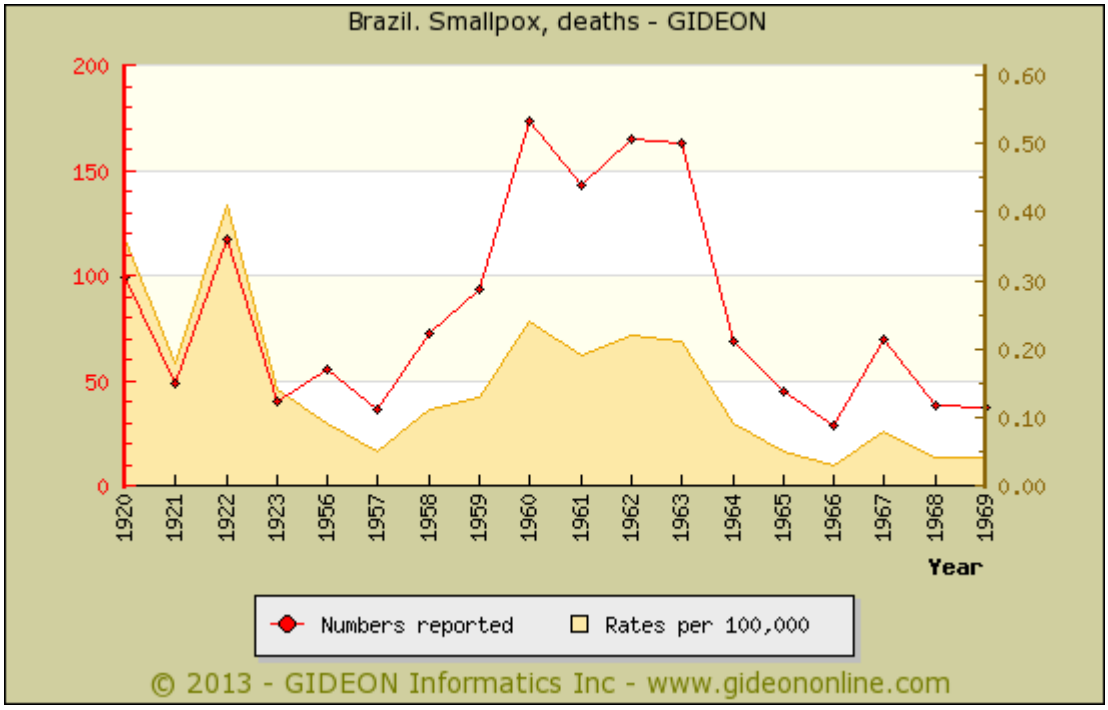

Graph: Brazil. Smallpox, deaths

11,393 patients (533 fatal cases) were recorded by an Infectious Diseases Institute in Sao Paulo during 1898 to 1970. [14]

9,781 cases of variola minor (75 fatal) were reported during 1968 to 1969. [15]

46,144,349 persons were vaccinated during 1962 to 1969.

Indigenous transmission ended in 1971. [16] [17]

Notable outbreaks:

1873 to 1875 - An outbreak of smallpox was reported in Minas Gerais. [18]

1908 - An outbreak (9,000 deaths) was reported. [19]

1911 - An outbreak (250,000 cases) was reported.

1956 - Outbreaks were reported in Braganca Paulista County (484 cases) [20-26] and among school students in Sao Paulo (131 cases). [27]

1969 - Outbreaks (1,492 cases, 12 fatal - in 33 outbreaks) of variola minor were reported - including 547 cases in Utinga, Bahia. [28] [29]

1970 (publication year) - An outbreak was reported in a hospital in Vitoria, Espirito Santo. [30]

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Sparganosis

Agent	PARASITE - Platyhelminthes, Cestoda. Pseudophyllidea, Diphyllobothriidae: Spirometra spp.
Reservoir	Copepod - to bird, amphibian or reptile
Vector	None
Vehicle	Water Undercooked reptile or amphibian meat
Incubation Period	20d - 3y
Diagnostic Tests	Identification of parasite in tissue.
Typical Adult Therapy	Excision
Typical Pediatric Therapy	As for adult
Clinical Hints	Painful or pruritic nodules and eosinophilia; worm present in skin, eye, brain or other foci and may survive for over five years.
Synonyms	

Clinical

The worm usually lodges in subcutaneous tissue or muscle in the chest, abdominal wall, extremities, or scrotum.
• Infection typically presents as a nodular mass, swelling and painful edema.
• Orbital sparganosis is also common, and is manifest by erythema of the lids and conjunctivae, proptosis and the presence of fixed or migrating subconjunctival masses. [1]
• 35 cases of sparganosis of the breast were identified in the literature as of 2010. [2]
• Less common sites have included the urinary tract, pleura, pericardium [3] , brain [4-6] , spinal canal [7] [8] , scrotum [9] [10] , articular bursa [11] , liver [12] and other abdominal viscera.
• Spinal disease may present as a lumbar radiculopathy [13-15] or mimic intramedullary tumor. [16]
• Pulmonary [17] or cerebral sparganosis may mimic tuberculosis [18]
• Genital sparganosis presents as a palpable subcutaneous nodule in the groin, labia, or scrotum and may mimic a tumor in the epididymis and testis or hydrocoele. [19-21]
• The patient may notice "lumps" which appear and then spontaneously disappears, over a period of weeks to months.
• The overlying skin is redness and pruritic, and local bleeding or necrosis may occur.

This disease is endemic or potentially endemic to 49 countries.

Sparganosis in Brazil

Sporadic cases of sparganosis are encountered. [22] [23]

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Sporotrichosis

Agent	FUNGUS. Ascomycota, Euascomycetes, Ophiostomatales: Sporothrix schenckii, S. brasiliensis and S. globosa A dimorphic dematiaceous fungus
Reservoir	Soil Vegetation Wood
Vector	None
Vehicle	Trauma Contact Air (rare)
Incubation Period	1w - 3m
Diagnostic Tests	Fungal culture. Serologic tests available in some centers.
Typical Adult Therapy	Itraconazole 100 to 200 mg PO daily X 3 to 6 months. OR Fluconazole 400 mg PO daily X 6 months. OR Potassium iodide 1 to 5 ml PO TID X 3 to 6 months
Typical Pediatric Therapy	Itraconazole 2 mg/kg PO daily X 3 to 6 months. OR Fluconazole 3 mg/kg PO daily X 6 months.
Clinical Hints	Draining nodules which follow lymphatics; acquired from contact with flowers, thorns, trees or other plant material; eye, brain, testis, bone and other tissues may be involved.
Synonyms	Schenck's disease, Sporothrix brasiliensis, Sporothrix globosa, Sporothrix mexicana, Sporothrix schenckii, Sporotrichose. ICD9: 117.1 ICD10: B42

Clinical

Clinical forms of sporotrichosis:

Cutaneous sporotrichosis begins as a painless erythematous papule which enlarges and suppurates, without systemic symptoms. [1] [2]
- Multiple lesions may spread along lymphatic channels. [3]
- Occasionally only a single lesion appears, and may persist for decades.
- Bilateral infection may occur. [4]
- Hematogenous infection of multiple skin sites has also been described. [5] [6]
- In some cases, ulcers appear on multiple body sites. [7]

Nodular lymphadenitis is also seen in *Nocardia brasiliensis* infection, tularemia, *Mycobacterium marinum* infection, and infections caused by *Leishmania panamensis/guyanensis* [8] [9]
- Lesions of sporotrichosis may rarely mimic those of pyoderma gangrenosum [10] or keratoacanthoma. [11-13]

Pulmonary sporotrichosis characteristically presents as a single upper lobe cavity associated with cough and low-grade fever.
- Multifocal lung lesions have also been reported. [14]
- 86 cases of pulmonary sporotrichosis were reported in the world's literature during 1960 to 2010. [15] Extrapulmonary multifocal disease involved the joints in 45.4%.

Osteoarticular sporotrichosis is characterized by infection of a single bone or large peripheral joint [16] • hip and shoulder involvement is not encountered. [17-20]
- Most patients are afebrile when first seen.
- Occasionally, the infection presents as tenosynovitis, usually of the wrist or ankle.

Other forms include conjunctival infection [21] [22] , hematogenous endophthalmitis, brain abscess, soft tissue mass [23] , meningitis [24] , orchitis, etc. [25]

This disease is endemic or potentially endemic to all countries.

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Sporotrichosis in Brazil

Time and Place:

759 human cases, 64 infected dogs and 1,503 infected cats were reported by a single institution during 1998 to 2004. [26]
- 13 cases were reported in Rio de Janeiro State through active case-finding during 1987 to 1998 - 78.8% following contact with cats.
- 178 cases were reported in Rio de Janeiro State during 1998 to 2001 [27] - 90.7% following contact with cats. [28] [29]
- 94 cases were reported at one hospital in Fiocruz during 2002 - animal contact was four-fold higher among infected patients than among controls. [30]
- 304 cases were reported in Rio Grande do Sul during 1967 to 2002 [31]
- 81 cases of pediatric sporotrichosis were treated by a single institution during 1998 to 2004. [32]
- At least 50 cases were reported from a single institution in Rio de Janeiro during 2005 to 2007. [33]
- Of 421 patients treated in Rio de Janeiro during 1988 to 2003, 67.4% reported contact with cats [34-38]

A series of ten cases of sporotrichosis in Rio Grande do Sul State was associated with armadillo hunting (2010 publication). [39]

A series of 21 HIV-positive patients in Rio de Janeiro were treated for sporotrichosis (7 in disseminated form) during 1999 to 2009. [40]

Cases of infection by *Sporothrix globosa* [41] and *Sporothrix mexicana* have been reported. [42] [43]

Disseminated *Sporothrix brasiliensis* infection was reported in an HIV-positive patient. [44]

Sporothrix has been identified on the nails of domestic cats in this country. [45-50]
- A series of 103 veterinary infections (92 cats and 11 dog) was treated in Rio Grande do Sul State (2011 publication) [51]
- A series of 147 of feline sporotrichosis was collected in Rio de Janeiro during 1998 to 2005) [52]

Notable outbreaks:

2004 (publication year) - An outbreak (347 cases) of sporotrichosis was reported among cats. [53]
2011 (publication year) - An outbreak (3 cases) among members of a family was related to scratches from a domestic cat. [54]

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Spotted fevers - Old World

Agent	BACTERIUM. Rickettsia conorii subsp. Conorii R. aeschlimannii, R. helvetica, R. massiliae, R. monacensis, R. slovaka
Reservoir	Dog Rodent Tick
Vector	Tick (Rhipicephalus sanguineus)
Vehicle	None
Incubation Period	6d - 7d (range 3d - 18d)
Diagnostic Tests	Serology. Demonstration of rickettsiae by immunofluorescence or culture. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg PO BID X 3 to 5d. OR Chloramphenicol 500 mg PO QID X 3 to 5d
Typical Pediatric Therapy	Doxycycline 2 mg/kg PO BID X 3 to 5d (maximum 200 mg/day). OR Chloramphenicol 10 mg/kg PO QID X 3 to 5d
Clinical Hints	Headache, myalgia, maculopapular rash; an eschar may be identifiable; patient may recall tick bite or dog contact during the preceding 1 to 3 weeks; untreated disease resolves within two weeks; case-fatality rates of 2% to 3% are reported.
Synonyms	Boutonneuse fever, Candidatus Rickettsia kellyi, DEBONEL, Febre escaro-nodular, Febre escaronodular, Indian tick typhus, Kenya tick typhus, Marseilles fever, Mediterranean spotted fever, R. aeschlimannii, Rickettsia aeschlimannii, Rickettsia conorii subsp conorii, Rickettsia conorii subsp indica, Rickettsia helvetica, Rickettsia massiliae, Rickettsia monacensis, Rickettsia raoultii, Rickettsia slovaca, Thai spotted fever, TIBOLA, Tick-borne lymphadenopathy. ICD9: 082.1 ICD10: A77.1

Clinical

The clinical features of Mediterranean spotted fever (MSM) are similar to those of Rocky Mountain spotted fever (q.v.); however, an eschar ("tache noire") and diffuse distribution of the rash characterize MSM. [1] [2]
• Hepatomegaly, elevation of serum transaminase levels and splenomegaly are common. [3]
• Complications may include meningitis with CSF pleocytosis (either lymphocytic or polymorphonuclear), encephalitis [4-6], renal failure [7], myocarditis [8], coronary artery ectasia [9], acute pancreatitis [10], bleeding diatheses, splenic rupture [11], hemophagocytic syndrome [12] [13] and retinitis. [14]
• There is evidence that Israeli spotted fever is more virulent than Mediterranean spotted fever. [15]

Spotted fever in India differs from the Mediterranean form in that the rash is often purpuric, and an inoculation eschar at the bite site is rarely found. [16]
• The clinical course is mild to moderately severe.

A syndrome of Dermacentor-borne necrosis with erythema and painful lymphadenopathy (DEBONEL) described in Spain has been ascribed to possible infection by *Rickettsia slovaca*. [17] [18]
• This syndrome appears to be identical to Tick-borne lymphadenopathy (TIBOLA), reported in Hungary. [19]
• Clinical features may include fever, dermal eschar, lymphadenopathy, facial edema, rash, headache, asthenia and alopecia.
• Rarely, tularemia presenting with scalp eschar and cervical lymphadenopathy may suggest infection by *Rickettsia slovaca*, *Rickettsia raoultii* or *Bartonella henselae* [20]

Rickettsia helvetica has been implicated in cases of mild flu-like illness (myalgia, arthralgia, headache, conjunctivitis) without rash, in Denmark, Italy, France and Thailand; and in myocarditis reported from Sweden. [21]

Rickettsia monacensis infection has been associated with headache, joint pain, a nonpruritic, disseminated maculopapular rash of the trunk and lower extremities, including palms and soles.
• An inoculation site eschar is not reported.

This disease is endemic or potentially endemic to 104 countries. Although Spotted fevers - Old World is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

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Spotted fevers - Old World in Brazil

2008 (publication year) - A South African tourist died of spotted fever after arriving to Brazil. [22-27]

2005 - A traveler from Portugal developed Mediterranean spotted fever in Brazil. [28]

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St. Louis encephalitis

Agent	VIRUS - RNA. Flaviviridae, Flavivirus: St. Louis encephalitis virus
Reservoir	Bird Mammal
Vector	Mosquito (Culex pipiens, Cx. tarsalis, Cx. nigripalpus, Cx. restuans, Cx. salinarius, Aedes, Sabethes)
Vehicle	None
Incubation Period	4d - 21d
Diagnostic Tests	Viral culture (blood, brain tissue, CSF). Serology. Nucleic acid amplification. Biosafety level 2.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Headache, meningitis, encephalitis, sore throat, myalgia, vomiting and photophobia; most cases encountered during late summer; infection resolves in 5 to 10 days; case-fatality rate 8% (over 25% above age 65).
Synonyms	American encephalitis, Modoc, Rio Bravo, SLE. ICD9: 062.3 ICD10: A83.3

Clinical

Adults account for 75% of patients with both overt and fatal infections. [1]
• Five to ten percent of patients will suffer from chronic neurological sequelae.

The disease may initially present with constitutional symptoms; aseptic meningitis; and overt and even fatal encephalitis. [2]
• Infection begins with malaise, fever, headache, respiratory symptoms, diarrhea, vomiting and myalgias.
• Symptoms may progress after several days to lethargy, confusion, tremor, clumsiness, and ataxia.
• General motor weakness is the rule, rather than focal neurological signs; however, 25% of patients develop cranial nerve signs.
• Signs of meningeal irritation are more common among children.
• Tremor and cerebellar signs are common.
• Seizures are uncommon, and carry a poor prognosis.
• Pneumonia, thrombophlebitis, pulmonary embolism, stroke, gastrointestinal hemorrhage, and nosocomial infections may intervene.
• The case-fatality rate is 8% (20% above age 60).

The peripheral leucocyte count may be slightly elevated
• Hyponatremia occurs in over 33% of patients.
• The CSF pressure is elevated in 33% of cases
• CSF protein is elevated in 70%.
• Between five to several hundred cells/ cu mm are present.

This disease is endemic or potentially endemic to 21 countries.

St. Louis encephalitis in Brazil

Time and Place:
St. Louis encephalitis is one of ten flavivirus diseases reported in Brazil as of 2000. [3]
- A case of human infection was documented in Belem in 1978. [4]
- A case of human infection was documented in Sao Paulo in 2004. [5] [6]
- In 2006, cases of human infection were confirmed among patients suspected of having dengue. [7]

Seroprevalence surveys:
0.06% of persons in Corte de Pedra, Valencia (Bahia, 1986 publication) [8]
3% of school children in Guanabara State (1975 publication) [9]
43.7% of horses in the Nhecolandia sub-region of South Pantanal (2010 publication) [10]

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5.4% of horses and caimans from the Pantanal (2009) [11]

Eight strains of St. Louis encephalitis virus were isolated from wild rodents, birds, and sentinel mice in Sao Paulo during 1967 to 1969.
- No cases of human disease were reported; however, 5% of the local population were found to be seropositive. [12]

SLE has been reported to cause four epizootics in a forest near Belem. [13]

Seropositive birds have been identified in Sao Paulo State (1978 to 1990) [14]

The presumed vector is *Culex nigripalpus*. [15]

Notable outbreaks:
2006 - An outbreak was reported in Sao Jose do Rio Preto (northwest Sao Paulo state) concurrent to a large dengue outbreak.

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Staphylococcal food poisoning

Agent	BACTERIUM. Staphylococcus aureus exotoxins
Reservoir	Human (nares, hands) Occasionally cattle (udder), dog/cat (nasopharyngeal)
Vector	None
Vehicle	Food (creams, gravies, sauces)
Incubation Period	2h - 4h (range 30 min - 9h)
Diagnostic Tests	Identification of bacterium in food.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	'Explosive' diarrhea and vomiting; usually no fever; no fecal leucocytes; onset 1 to 6 hours after food; resolves within 1 to 2 days; fatality is rare.
Synonyms	Staphylococcus aureus food poisoning. ICD9: 005.0 ICD10: A05.0

Clinical

Usually symptoms start within several hours of ingestion of potentially contaminated foods
• Illness is heralded by nausea, vomiting and intestinal cramping, followed by urgency and profuse watery non-bloody diarrhea.
• Symptoms resolve within 12 to 24 hours.
• Multiple family members or patrons of the same eating establishment may be affected.
• The presence of both explosive diarrhea and vomiting, lack of fever and short incubation period are helpful in distinguishing this entity from other forms of food poisoning.

This disease is endemic or potentially endemic to all countries.

Staphylococcal food poisoning in Brazil

Prevalence surveys:

Staphylococcus aureus was found in 94.1% of raw cow's milk samples in Minas Gerais (2012 population) [1]

Notable outbreaks:

1976 - An outbreak (28 cases) in Rio de Janeiro was associated with contaminated cream cakes served on a commercial airline flight. [2]

1994 (publication year) - An outbreak (12 cases) at a birthday party in south-eastern Brazil was associated with cream-filled cake. [3]

1998 - An outbreak (2,000 cases, approximate) was related to a religious celebration in Minas Gerais. [4]

1998 - An outbreak (180 cases, approximate) was reported in Sao Paulo State. [5]

1999 - Outbreaks (706 cases total = 378 + 328) due to contaminated Minas cheese and raw milk were reported in Minas Gerais.

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Staphylococcal scalded skin syndrome

Agent	BACTERIUM. Staphylococcus aureus phage group 2 A facultative gram-positive coccus
Reservoir	Human
Vector	None
Vehicle	Direct contact; infected secretions
Incubation Period	1d - 4d
Diagnostic Tests	Typical clinical features; Recovery of S. aureus from localized wound or blood ; skin biopsy may be helpful
Typical Adult Therapy	Fluid replacement (as for burn) ; Intravenous Nafcillin or Oxacillin, in addition to application of anti-staphylococcal drug to local source infection; Vancomycin if MRSA
Typical Pediatric Therapy	Fluid replacement (as for thermal burn) ; Intravenous Nafcillin or Oxacillin, in addition to application of anti-staphylococcal drug to local source infection; Vancomycin if MRSA
Clinical Hints	Acute, generalized exfoliative dermatitis which occurs primarily in infants and young children; a pre-existing localized skin infection is present in most - but not all - cases.
Synonyms	Lyell disease, Ritter disease, Ritter von Ritterschein disease, Scalded skin syndrome, SSSS. ICD9: 695.81 ICD10: L00

Clinical

Staphylococcal scalded skin syndrome (SSSS) is characterized by diffuse erythematous cellulitis followed by extensive skin exfoliation. [1] [2]

- Generalized erythema and then bulla formation with separation of the skin at the granular cell layer. [3] [4]
- A warm, 'sandpaper' erythema with accentuation in the flexor creases may mimic scarlet fever; while the presence of flaccid bullae and Nikolsky sign may suggest pemphigus. [5]
- Skin biopsy can be used to differential SSSS from Toxic epidermal necrolysis. [6]
- Facial edema and perioral crusting are often present.

Dehydration may indicate fluid loss (as in thermal burns)

- Complete recovery occurs in most cases, within one to two weeks. [7]
- The case-fatality rate in uncomplicated SSSS is less than 2%.
- Rare instances of recurrence have been reported [8]
- Staphylococcal septicemia complicates SSSS in a minority of cases.

This disease is endemic or potentially endemic to all countries.

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Streptococcus suis infection

Agent	BACTERIUM. Streptococcus suis I and Streptococcus suis II A facultative gram-positive coccus
Reservoir	Pig
Vector	None
Vehicle	Air Secretions Meat Local wounds
Incubation Period	Unknown. Probably hours to few days
Diagnostic Tests	Culture of blood, tissue, body fluids
Typical Adult Therapy	Systemic antibiotic. Usually susceptible in vitro to Penicillin, Amoxicillin, Chloramphenicol and Gentamicin
Typical Pediatric Therapy	Systemic antibiotic
Clinical Hints	Severe multisystem disease, hemorrhagic diatheses, deafness or meningitis appearing hours to a few days after contact with pigs or pig products.
Synonyms	Streptococcus suis. ICD9: 027.8 ICD10: A48.8

Clinical

Demography:
Virtually all patients have been farmers and butchers, of whom 80 percent were men.
• Most had been involved in butchering sick pigs or selling the pork.
• Over 40 percent of the patients were in the age group 50 to 60 years, and none were children. [1]

Signs and symptoms:
• Clinical features of *Streptococcus suis* II infection include high fever, malaise, nausea and vomiting • followed by meningitis, subcutaneous hemorrhage, multi-organ failure (hepatic, renal, pulmonary, cardiac) and coma in severe cases. [2] [3]
• Toxic shock syndrome is common. [4]
• Sensorineural hearing loss is often present. [5] [6]
• Peritonitis [7] , endocarditis [8-10] , mycotic aortic aneurysm [11] , rhabdomyolysis [12] , spondylodiscitis [13] , salcroiliitis [14] , monoarthritis [15-17] , endophthalmitis [18] and cranial nerve palsy [19] have been reported.
• Persons with occupational exposure may exhibit asymptomatic seropositivity toward *S. suis*. [20]
• Relapses of meningitis may occur. [21]

This disease is endemic or potentially endemic to 227 countries.

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Strongyloidiasis

Agent	PARASITE - Nematoda. Phasmidea: Strongyloides stercoralis (Strongyloides fulleborni is occasionally implicated in systemic disease)
Reservoir	Human ? Dog Monkey (for Strongyloides fulleborni)
Vector	None
Vehicle	Skin contact Soil Feces Autoinfection Sexual contact (rare)
Incubation Period	14d - 30d
Diagnostic Tests	Identification of larvae (or ova, for Strongyloides fulleborni) in stool or duodenal aspirate. Serology.
Typical Adult Therapy	Ivermectin 200 micrograms/kg/d PO daily X 2d OR Thiabendazole 25 mg/kg BID (max 3g) X 2d OR Albendazole 400 mg/d X 3d (7 days for hyperinfection syndrome)
Typical Pediatric Therapy	Ivermectin 200 micrograms/kg/d PO daily X 2d OR Thiabendazole 25 mg/kg BID (max 3g) X 2d. OR Albendazole 200 mg/d X 3d (7 days for hyperinfection syndrome)
Clinical Hints	Diarrhea, gluteal or perineal pruritus and rash; eosinophilia often present; widespread dissemination encountered among immune-suppressed patients because of uncontrolled autoinfection (case-fatality rate for this complication = 80%).
Synonyms	Anguilluliasis, Anguillulosis, Cochin China gastroenteritis, Diploscapter, Halicephalobus, Halicephalobus, Larva currens, Leptodera intestinals, Leptodera stercoralis, Micronema, Pseudo-rhabdis stercoralis, Rhabditis stercoralis, Rhabdonema intestinale, Rhabdonema stercoralis, Strongyloides fulleborni, Strongyloides stercoralis, Strongyloidose, Threadworm, Turbatrix. ICD9: 127.2 ICD10: B78

Clinical

Strongyloidiasis may present as long as 75 years following initial acquisition of the parasite. [1]

Gastrointestinal strongyloidiasis:
The symptoms of strongyloidiasis reflect invasion of the skin, larval migration of larvae intestinal penetration.
- Approximately one third of patients are asymptomatic.
- Dermal and pulmonary symptoms resemble those of hookworm [2] , pruritic papular or linear urticarial rash (larva currens [3] [4]) and a Loeffler-like syndrome.
- Intestinal penetration is characterized by abdominal pain, mucous diarrhea and eosinophilia. [5]
- Vomiting, weight loss, protein-losing enteropathy and inappropriate ADH excretion [6] are occasionally encountered.
- Intestinal obstruction has been reported. [7] [8]
- Yellowish mucosal nodules are seen on colonoscopy, predominantly in the ascending colon. [9]
- Findings in colonic infection may mimic those of ulcerative colitis. [10]

Generalized strongyloidiasis:
5 to 22% of patients develop a generalized or localized urticarial rash beginning in the anal region and extending to the buttocks, abdomen, and thighs.
- Extraintestinal infection may involve a wide variety of organs. [11-16]
- Autoinfection is characterized by massive larval invasion of the lungs and other organs.
- Massive systemic strongyloidiasis occurs in patients with lymphoma, leukemia and AIDS; and during high-dose therapy with corticosteroids. [17]
- Findings include generalized abdominal pain, concurrent gram-negative bacillary septicemia (55% of cases) [18] , bilateral diffuse pulmonary infiltrates and ileus.
- Hyperinfection may mimic acute exacerbation of COPD [19]
- Eosinophilia may be present or absent at this stage; and rare instances of eosinophilic meningitis have been reported. [20]
- An outbreak of hyperinfection strongyloidiasis has been reported among immune-suppressed renal transplant recipients. [21]
- *Strongyloides stercoralis* is the only helminth responsible for disseminated infection in immunocompromised patients. [22]

Strongyloides fulleborni infection is usually asymptomatic.

Strongyloides fulleborni kellyi infection [23] is most common among infants, and consist of abdominal distention, mild

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diarrhea and protein-losing enteropathy.
• Respiratory distress may occur, and is associated with a characteristic high-pitched cry.

This disease is endemic or potentially endemic to all countries.

Strongyloidiasis in Brazil

Prevalence rates vary from 15% to 82% in some populations.

Strongyloidiasis is implicated in 5.3% of pediatric diarrhea episodes in northeastern Brazil. [24]

A review of strongyloidiasis in Brazil (2011 publication) - see reference [25]

Prevalence surveys:

13% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [26]
1.3% of children in Uberlandia, Minas Gerais (2008 publication) [27]
5.6% of the Parakana indigenous community in southwestern Para State (1998 publication) [28]
3.8% of Terena Indians (Mato Grosso do Sul) [29]
5.8% in Sao Lourenco da Mata, rural Recife (Pernambuco) (1990 publication) [30]
6.7% in Araquara region, Sao Paulo (2008 publication) [31]
5.8% in rural Recife (1989) [32]
1.0% in Eirunepe, Amazon (2005 publication) [33]
10.81% of the population of Campanis City (1993 publication) [34]
8.1% of persons in Americaninhas, Minas Gerais (2004) [35]
0.4% of persons in Maria Helena, Parana (2004 to 2006) [36]
5.6% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [37]
2.5% of AIDS patients in Sao Paulo (1999 publication) [38]
30.1% of HIV-positive patients before HAART vs. 11% after HAART (Ceara, 2008 publication) [39]
1.7% of adult renal transplant recipients (Porto Alegre, 2001 to 2005) [40]
9.1% of patients with GI malignancy and 2.3% of controls (2007 publication) [41]
0.7% of HIV-positive patients in northeastern Sao Paulo (1998 to 2008) [42]
0.5% to 0.9% of dogs in southeastern Brazil (2006 to 2007 publications) [43] [44]
0.8% of beach sand samples from Santos (*Strongyloides* sp, 2004 to 2005) [45]

Seroprevalence surveys:

24.2% of patients with GI malignancy and 4.5% of controls (2007 publication) [46]
27% of dog keepers and 24.3% of dogs in breeding kennels in southeastern Uberlandia, southeastern Brazil. (Minas Gerais, 2007 publication) [47]

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Subdural empyema

Agent	BACTERIUM. Haemophilus influenzae, oral anaerobes, streptococci, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Imaging techniques (CT scan, etc).
Typical Adult Therapy	Antimicrobial agent(s) directed at known or likely pathogen
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, severe headache, vomiting, and signs of meningeal irritation and increased cerebrospinal fluid pressure; may follow head trauma, meningitis, otitis or sinusitis; case-fatality rate 15% (alert) to 60% (comatose).
Synonyms	

Clinical

Most patients present with headache, meningismus, decreased mental status and hemiparesis. [1]
• 32 cases of suppurative parotitis in neonates were reported during 1970 to 2004. [2]
• In 60 to 90% of cases, sinusitis or otitis is present.
• Extension of the infection into the subdural space is heralded by fever, focal and later generalized headache, vomiting, and meningismus. [3]
• 50% of patients exhibit altered mental function.
• Focal neurological signs appear within 24 to 48 hours, and rapidly progress to hemispheric dysfunction with hemiparesis and hemisensory deficit.
• Seizures, usually focal, occur in 50% of cases, and papilledema in less than 50%.
• Signs of increased intracranial pressure appear, leading to cerebral herniation and death.
• Chronic and even sterile subdural collections are also encountered, often following antibiotic therapy.

This disease is endemic or potentially endemic to all countries.

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Suppurative parotitis

Agent	BACTERIUM. Most commonly Staphylococcus aureus
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Unknown
Diagnostic Tests	Clinical features (local swelling and purulent discharge from salivary ducts). Stain and culture of discharge.
Typical Adult Therapy	Surgical drainage and aggressive parenteral antistaphylococcal therapy
Typical Pediatric Therapy	As for adult
Clinical Hints	Consider when confronted by unexplained fever in the setting of malnutrition, dehydration and obtundation; local swelling and discharge of pus from salivary duct are diagnostic.
Synonyms	Parotitis, bacterial. ICD9: 527.2 ICD10: K11.3

Clinical

Suppurative parotitis is characterized by the sudden onset of firm, erythematous swelling of the pre• and post auricular areas, extending to the angle of the mandible. [1]
• Marked pain and tenderness is accompanied by high fever, chills and marked toxicity.
• Pus may be seen exiting from the parotid duct.
• Progression of the disease can result in massive swelling of the neck, respiratory obstruction, septicemia, fistula formation [2] and osteomyelitis of the adjacent facial bones.
• The condition should be suspected in any patient with unexplained or prolonged fever.

This disease is endemic or potentially endemic to all countries.

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Syphilis

Agent	BACTERIUM. Treponema pallidum subsp. pallidum A microaerophilic gram-negative spirochete
Reservoir	Human
Vector	None
Vehicle	Sexual contact Infected secretions
Incubation Period	2w - 4w (range 10d - >8w)
Diagnostic Tests	Dark field microscopy (chancre). VDRL confirmed by antitreponemal test (FTA, MHTP). Nucleic acid amplification.
Typical Adult Therapy	Primary, secondary or early (< 1 year) latent: Benzathine Penicillin G 2.4 million units IM Other stages: Repeat dosage at one and two weeks Alternatives: Tetracycline, Ceftriaxone
Typical Pediatric Therapy	Primary, secondary or early (< 1 year) latent: Benzathine Penicillin G : Weight <14 kg: 600,000u IM Weight 14 to 28 kg: 1,200,000u IM Other stages: Repeat dosage at one and two weeks
Clinical Hints	Firm, painless chancre (primary syphilis); later fever, papulosquamous rash and multisystem infection (secondary syphilis); late lesions of brain, aorta, bone or other organs (tertiary syphilis).
Synonyms	Canton rash, Chinese ulcer, Christian disease, French disease, German sickness, Harde sjanker, Lues, Neopolitan itch, Polish sickness, Sifilide, Sifilis, Spanish pockes, Syfilis, Treponema pallidum. ICD9: 090,091,092,093,094,095,096,097 ICD10: A50,A51,A52,A53

Clinical

WHO Case definition for surveillance:
The signs and symptoms of syphilis are multiple.
• The primary stage usually, but not necessarily, involves ulceration of the external genital organs and local lymphadenopathy; secondary and tertiary syphilis show mainly dermatological and systemic manifestations. For surveillance purposes, only confirmed cases will be considered.
Confirmed case
• A person with a confirmed positive serology for syphilis (Rapid Plasma Reagin (RPR) or VDRL confirmed by TPHA (*Treponema pallidum* hemagglutination antibodies) or FTA (fluorescent treponemal antibody absorption).
Case classification
• Congenital syphilis: An infant with a positive serology, whether or not the mother had a positive serology during pregnancy.
• Acquired syphilis: All others.
Additional notes:
• The prevalence rate among pregnant women in developing countries varies between 3% and 19%. Maternal syphilis is associated with congenital syphilis (one third of births from such pregnancies), and with spontaneous abortion and stillbirth.
• Because the primary lesion is often painless and secondary syphilis is usually not diagnosed, women are mainly identified through serological screening.

Syphilis is a chronic disease with a waxing and waning course; and is reported from all countries.
• Transmission is mainly by sexual contact.
• Primary, secondary, and early latent syphilis are potentially infectious.

Stages of syphilis:
• Primary syphilis is characterized by a painless chancre at the site of inoculation. [1] Penile swelling without an overt chancre has also been reported. [2]
• The secondary stage is characterized by a generalized (rarely localized [3] non-pruritic polymorphic [4-6] or papulonecrotic [7] rash , lymphadenopathy, and systemic manifestations. [8-13] Moist flat genital or mucosal lesions (condyloma lata) [14] or granulomatous dermatitis [15] may be evident.
• An asymptomatic latent period follows, which for epidemiological purposes is divided into early (<1 year) and late (>1 year) stages.
• The tertiary stage is the most destructive and is marked by cardiovascular [16] and neurological sequelae [17-22] , and gummatous involvement of any organ system. [23-26]
• As of 2009, the world's literature contained 165 reports of cerebral syphilitic gummata • 64% in men and 66% located on the cerebral convexities. [27]

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• Syphilitic uveitis may present in the absence or other clinical manifestations of syphilis. [28] Acute posterior placoid chorioretinitis is also encountered. [29] [30] 143 cases of syphilitic uveitis were reported in the English Language literature during 1984 to 2008. [31]

The clinical features of congenital infection are similar to those of secondary syphilis, and may be associated with deformation of teeth, bones and other structures. [32]

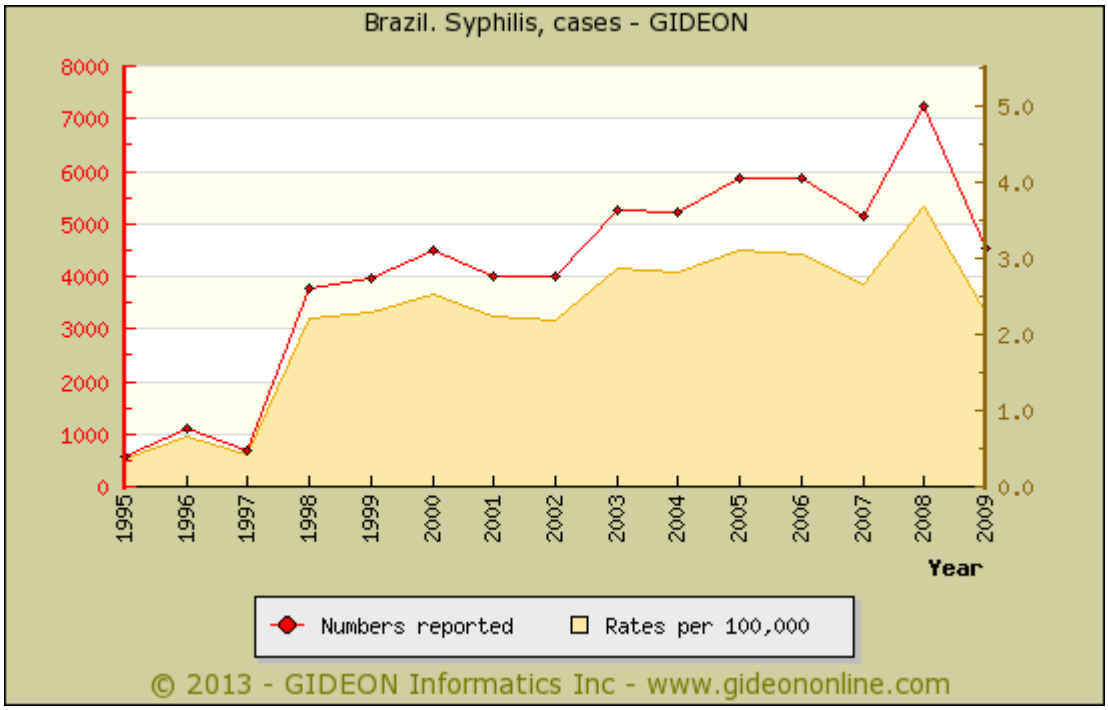
Acquired syphilis in patients with HIV infection is characterized by severe and accelerated infection, often with overt meningitis, hepatitis [33] , lues maligna (a florid papulopustular rash) [34] and other forms of systemic involvement. [35-44]
• The presence of concurrent syphilis does not affect the progression of AIDS. [45]

This disease is endemic or potentially endemic to all countries.

Syphilis in Brazil

Approximately 143,000 cases of venereal syphilis were reported during 1987 to 1996.

439,000 new cases were estimated for 1996.



Graph: Brazil. Syphilis, cases

Notes:
1. Approximately 4,500 cases of congenital syphilis were reported during 1987 to 1996.
2. 11.5% of pregnant women are seropositive (Recife).
Individual years:
2004 - 159 fatal

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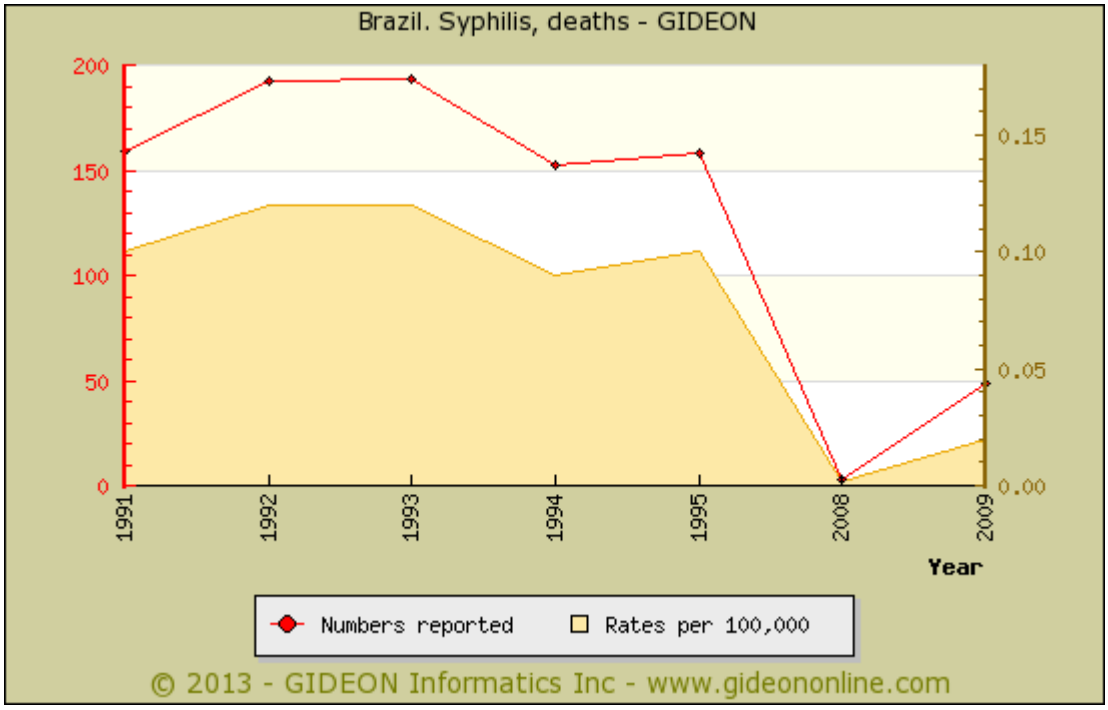
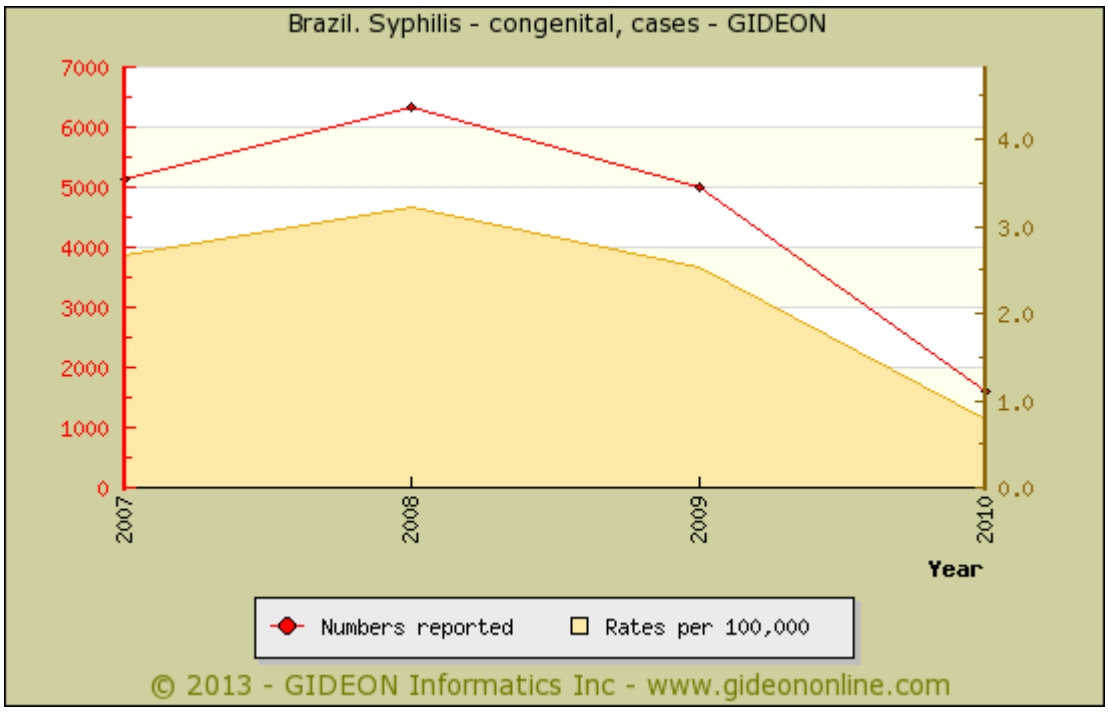


Graph: Brazil. Syphilis, deaths



Graph: Brazil. Syphilis - congenital, cases

Notes:
1. 379 cases of congenital syphilis were identified in a Porto Alegre hospital during 1997 to 2004, accounting for 1.5% of live births. [46]
2. The rate of congenital syphilis in Belo Horizonte, Minas Gerais was 0.9 per 1,000 live births in 2001; 1.6 per 1,000 in 2008. [47]

Prevalence surveys:

0.7% of women with lethal pregnancy outcomes (Para, 2004) [48]

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1.5% of live births in Porto Alegre (congenital syphilis, 1997 to 2004) [49]
13.1% of fetal deaths and 6.5% of neonatal deaths in municipal maternity hospitals during 1996 to 1998, 16.2% of fetal deaths and 7.9% of neonatal deaths during 1999 to 2002 (Rio de Janeiro). [50]

Seroprevalence surveys:

5.7% of homeless persons in Sao Paulo (2002 to 2003) [51]
1% of blood donors (1995) - range 0.5% in the south to 2% in the north
1.1% of blood donors in Sao Paulo (1991) [52]
2.1% of blood donors in Para (ELISA, 2006) [53]
0.4% of sickle cell anemia patients in Bahia (1995 to 2009) [54]
1.12% of patients with mental illness (2009 publication) [55]
18.4% of male prison inmates (Goias, 1989 publication) [56]
34% of injecting drug users (Santos, 1996 publication) [57]
4.0% of female crack cocaine users in Bahia, Salvador (2007 publication) [58]
29% of CSW (Santos, 1990) [59]
18% of male STD patients in Manaus (2008 publication) [60]
8.2% of STD clinic patients in Ceara (1999 to 2008) [61]
5.7% of female prisoners in Sao Paulo (1997 to 1998). [62]
3.4% of incarcerated adolescents (Salvador, 2008 publication) [63]
0.2% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (2007 publication) [64]
1.2% of young women in Vitoria (2006) [65]
1.2% of women ages 18 to 29, in Vitoria, Espirito Santo (2012 publication) [66]
52% of patients attending an STD clinic in Manaus (2007 publication) [67]
20.5% of HIV-positive patients in southern Brazil (1991 to 2008) [68]
3.1% of pregnant HIV-positive women (Rio Grande do Sul, 2012 publication) [69]
9.5% of HIV-positive women in Salvador, Bahia (2012 publication) [70]
1.6% of pregnant women in Parana (1996 to 1998) [71]
1.7% of pregnant women nationwide (1999 to 2000)
1.6% of pregnant women (PAHO statistic) (2004)
0.4% of pregnant women in Espiritu Santo (2007) [72]
0.9% of pregnant women in Sergipe (2009 publication) [73]
1.0% of pregnant women in the western Amazon (2008) [74]
1% of pregnant women (Belem, 2012 publication) [75]
2.2% of pregnant women in the Amazon region (2011 publication) [76]
0.43% of pregnant women in Itajai, Santa Catarina State (2002 to 2007) [77]
0.9% of pregnant women in Sao Paulo (2011 publication) [78]
2.1% of low-income postpartum women and 3.6% of pregnant women treated in Greater Metropolitan Vitoria, Espirito Santo State (1999) [79]
2.6% of women in rural Alagoas (1997) [80]
17.4% of homeless people in Sao Paulo (2006 to 2007) [81]
18.4% of recyclable-waste collectors in Santos (2005) [82]
4.5% of truck drivers crossing the southern Brazilian border at Foz do Iguacu (2003 to 2005) [83]
3.2% of men and 2.6% of women in Alto Solimoes, Amazon border region (2012 publication) [84]
0.53% of armed forces conscripts (2007) [85]

2,439 seropositive blood donors were identified in Sao Paulo during 1999 to 2003. [86]

Syphilis coinfection was detected in 2.7% of HIV/AIDS hospital outpatients; ratio of men to women with coinfection was 4:1, with homosexual men the most effected. (Rio de Janeiro, 2005) [87]

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Taeniasis

Agent	PARASITE - Platyhelminthes, Cestoda. Cyclophyllidea, Taeniidae: Taenia solium & T. saginata (other species occasionally encountered)
Reservoir	Cattle Pig
Vector	None
Vehicle	Meat
Incubation Period	6w - 14w
Diagnostic Tests	Identification of ova or proglottids in feces.
Typical Adult Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 2 g PO once
Typical Pediatric Therapy	Praziquantel 10 mg/kg PO as single dose OR Niclosamide 50 mg/kg PO once
Clinical Hints	Vomiting and weight loss; often symptomatic or first appreciated due to passage of proglottids or 'tape' segments; parasite may survive for over 25 years in the human intestine.
Synonyms	Bandwurmer [Taenia], Drepanidotaenia, Gordiid worm, Hair snake, Mesocestoides, Raillietina, Taenia longihamatus, Taenia saginata, Taenia solium, Taenia taeniaformis, Taeniarhynchiasis, Tapeworm (pork or beef), Tenia. ICD9: 123.0,123.2 ICD10: B68

Clinical

Most cases of *Taenia* infestation are subclinical.

Symptomatic taeniasis may be associated with nausea, vomiting, epigastric fullness, weight loss or diarrhea. [1]
• *Taenia saginata* often becomes apparent when motile proglottids are passed through the anus; however, this is uncommon with *T. solium* infestations.
• Eosinophilia is not a prominent finding.
• Rare complications include appendicitis, cholangitis [2], cholecystitis [3], pancreatitis or intestinal obstruction. [4]
• The major complication of *T. solium* infection, Cysticercosis, is discussed separately in this module.

This disease is endemic or potentially endemic to all countries.

Taeniasis in Brazil

Prevalence surveys:

1% of the population are infested by *Taenia saginata*
0.7% of school children in northwestern Minas Gerais (2002 publication) [5]
0.2% of persons in Americaninhas, Minas Gerais (2004) [6] [7]
0.2% of persons in Maria Helena, Parana (2004 to 2006) [8]
0.7% of HIV-positive patients in northeastern Sao Paulo (*Taenia saginata*, 1998 to 2008) [9]
18.7% of cattle (cysticercosis, Mato Grosso do Sul, 2010 publication) [10]

Seroprevalence surveys:

4.4% of free-roaming pigs in Salvador, 3.2% in Santo Amaro, and 23.5% in Jequie (*T. solium*, Bahia State, 2001 publication) [11]
9.4% of pigs (cysticercosis, Mato Grosso do Sul, 2010 publication) [12]

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Tetanus

Agent	BACTERIUM. Clostridium tetani An anaerobic gram-positive bacillus
Reservoir	Animal feces Soil
Vector	None
Vehicle	Injury
Incubation Period	6d - 8d (range 1d - 90d)
Diagnostic Tests	Isolation of C. tetani from wound is rarely helpful. Serology (specimen taken before administration of antitoxin).
Typical Adult Therapy	Human antitoxin (see Vaccine module). Metronidazole (2 g daily) or Penicillin G (24 million u daily) or Doxycycline (200 mg daily). Diazepam (30 to 240 mg daily). Tracheostomy, hyperalimentation
Typical Pediatric Therapy	Human antitoxin (see Vaccine module). Metronidazole (30 mg/kg daily); or Penicillin G (300,000 units/kilo daily). Diazepam. Tracheostomy, hyperalimentation
Vaccines	DT DTaP DTP Td Tetanus immune globulin Tetanus
Clinical Hints	Trismus, facial spasm, opisthotonus, tachycardia and recurrent tonic spasms of skeletal muscle; sensorium is clear; disease may persist for 4 to 6 weeks; case fatality rate = 10% to 40%.
Synonyms	Lockjaw, Starrkrampf, Stelkramp, Tetano, Tetanos. ICD9: 037,771.3 ICD10: A33,A34,A35

Clinical

Tetanus may present in any of four clinical forms: generalized, localized, cephalic, and neonatal. [1]
• In general, shorter incubation periods are associated with a worse prognosis.
• Certain portals of entry (compound fractures) and underlying conditions (heroin addiction) are also associated with poorer prognoses.
• A series of 11 cases of tetanus related to tungiasis (25% of all tetanus cases) was reported by a single hospital in Brazzaville over an 11-month period (1989 publication). [2]
• An outbreak of 12 cases of tetanus in Argentina was reported among elderly women treated with sheep cell therapy (1996). [3]
• In one case, tetanus was associated with chronic otitis media. [4]
• Tetanus has been reported in a child who bit her own tongue during a convulsion [5] and following a snake bite (2007 publication) [6]
• An attack of tetanus does not result in immunity. Therefore, recurrent tetanus is possible, unless the patient is given a series of toxoid following recovery. [7-14]

Generalized tetanus, the most common form, begins with trismus ("lockjaw") and risus sardonicus (increased tone in the orbicularis oris).
• Abdominal wall rigidity may be present.
• The generalized spasm consists of opisthotonic posturing with flexion of the arms and extension of the legs. [15]
• The patient does not lose consciousness, and experiences severe pain during these spasms.
• Spasms often are triggered by sensory stimuli.
• Respiration may be compromised by upper airway obstruction, or by participation of the diaphragm in the general muscular contraction.
• Autonomic dysfunction, usually occurring after several days of symptoms, is currently the leading cause of death in tetanus.
• Complications of tetanus include rhabdomyolysis and renal failure [16] [17]
• The illness can progress for two weeks, while the severity of illness may be decreased by partial immunity.
• Recovery takes an additional month, but is complete unless complications supervene.
• Lower motor neuron dysfunction may appear after the spasms remit, and persist for several additional weeks.
• A case of *Clostridium tetani* bacteremia has been reported. [18]
• Case-fatality rates of 10% to 50% are reported, but may be as high as 70% in Africa. [19]

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• The differential diagnosis of tetanus includes strychnine poisoning and neuromyotonia (Isaac's syndrome). [20]

Localized tetanus presents as rigidity of the muscles associated with the site of inoculation.
• Initial symptomatology may be limited to back pain [21]
• The illness may be mild and persistent, and tends to resolve spontaneously.
• Weakness and diminished muscle tone are often present in the most involved muscle.
• Localized tetanus is often a prodrome of generalized tetanus.

Cephalic tetanus is a form of localized disease affecting the cranial nerve musculature
• Facial nerve weakness, is often apparent, and extraocular muscle involvement is occasionally noted.

Neonatal tetanus follows infection of the umbilical stump, most commonly as a result of a failure of aseptic technique following delivery of non-immune mothers.
• The condition usually manifests with generalized weakness and failure to nurse; followed by rigidity and spasms.
• The mortality rate exceeds 90%, and psychomotor retardation is common among survivors.
• Poor prognostic factors include age younger than 10 days, symptoms present for fewer than 5 days before presentation to hospital, fever, and the presence of risus sardonicus or fever.
• Apnea is the leading cause of death in the first week of disease, and sepsis in the second week.
• Bacterial infection of the umbilical stump leads to sepsis in almost half of babies with neonatal tetanus.

The WHO Case definition for surveillance of neonatal tetanus is as follows:
• Suspected case: Any neonatal death between 3-28 days of age in which the cause of death is unknown; or any neonate reported as having suffered from neonatal tetanus between 3-28 days of age and not investigated.
• Confirmed case: Any neonate with a normal ability to suck and cry during the first two days of life, and who between 3 and 28 days of age cannot suck normally, and becomes stiff or has convulsions (i.e. jerking of the muscles) or both.
• Hospital-reported cases of neonatal tetanus are considered confirmed.
• The diagnosis is purely clinical and does not depend upon laboratory or bacteriological confirmation.

This disease is endemic or potentially endemic to all countries.

Tetanus in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

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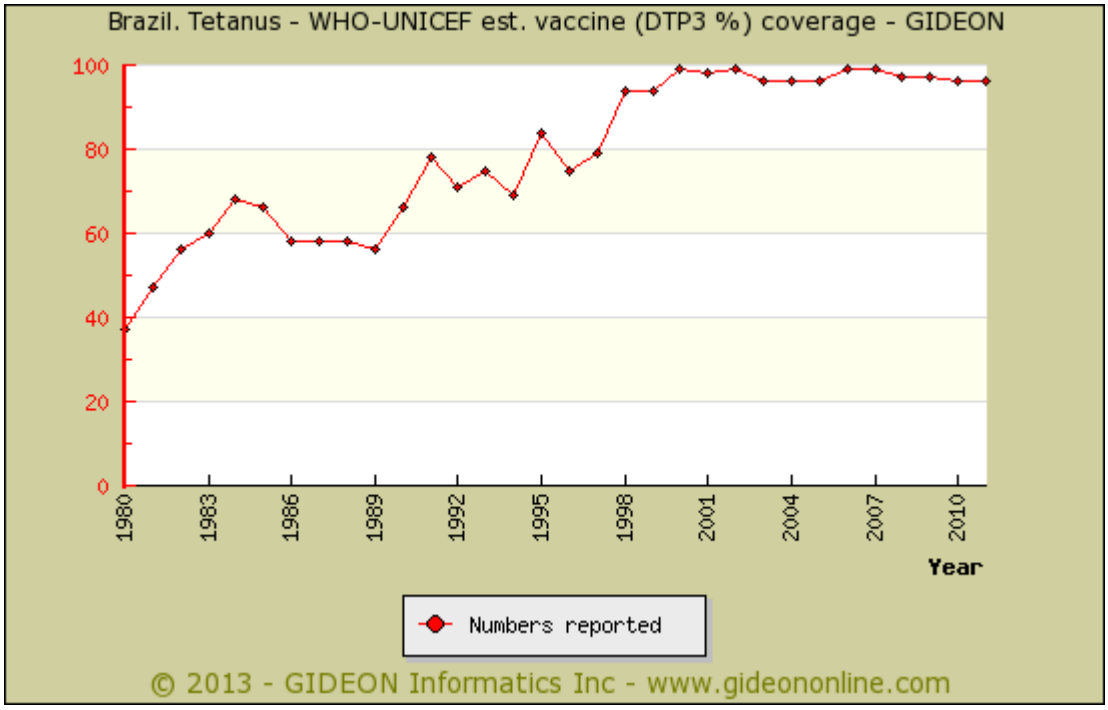


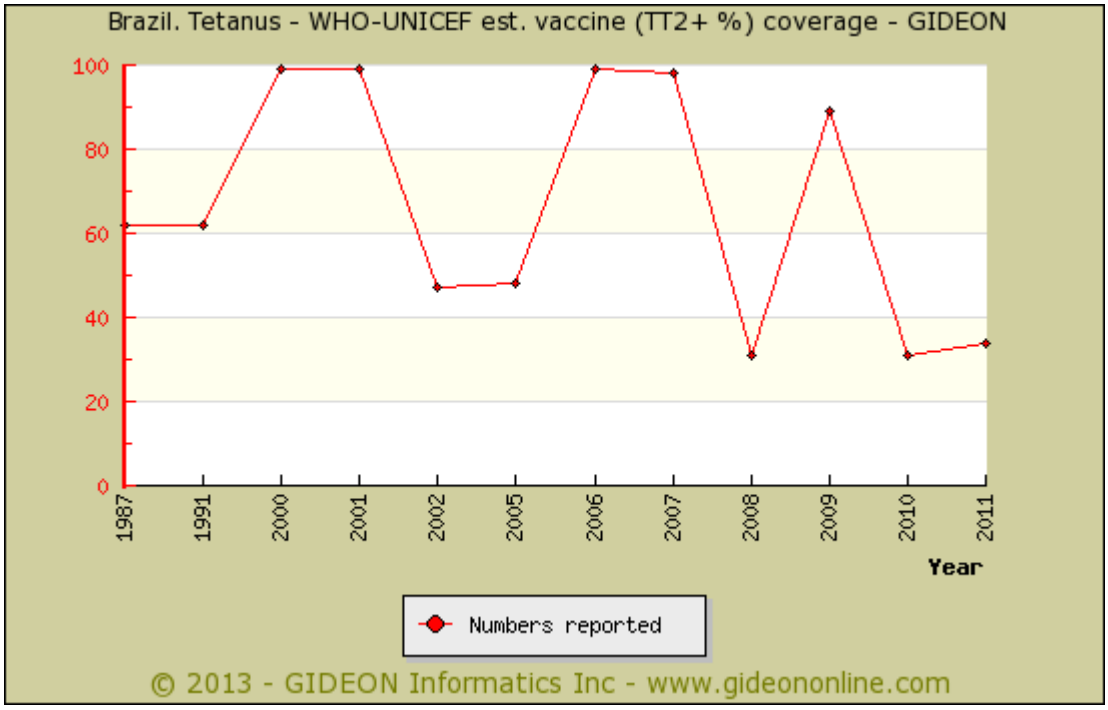
Graph: Brazil. Tetanus - WHO-UNICEF est. vaccine (DTP3 %) coverage

Seroprevalence surveys:

100% of adolescents. 23.1% of adolescents had not received a booster dose of diphtheria or tetanus vaccine in the previous 10 years. (Sao Paulo, 2001) [22]

79% of population (Sao Paulo, 2001 to 2003) [23]

84.7% of health care workers in neonatal units (Sao Paulo, 2002) [24]



Graph: Brazil. Tetanus - WHO-UNICEF est. vaccine (TT2+ %) coverage

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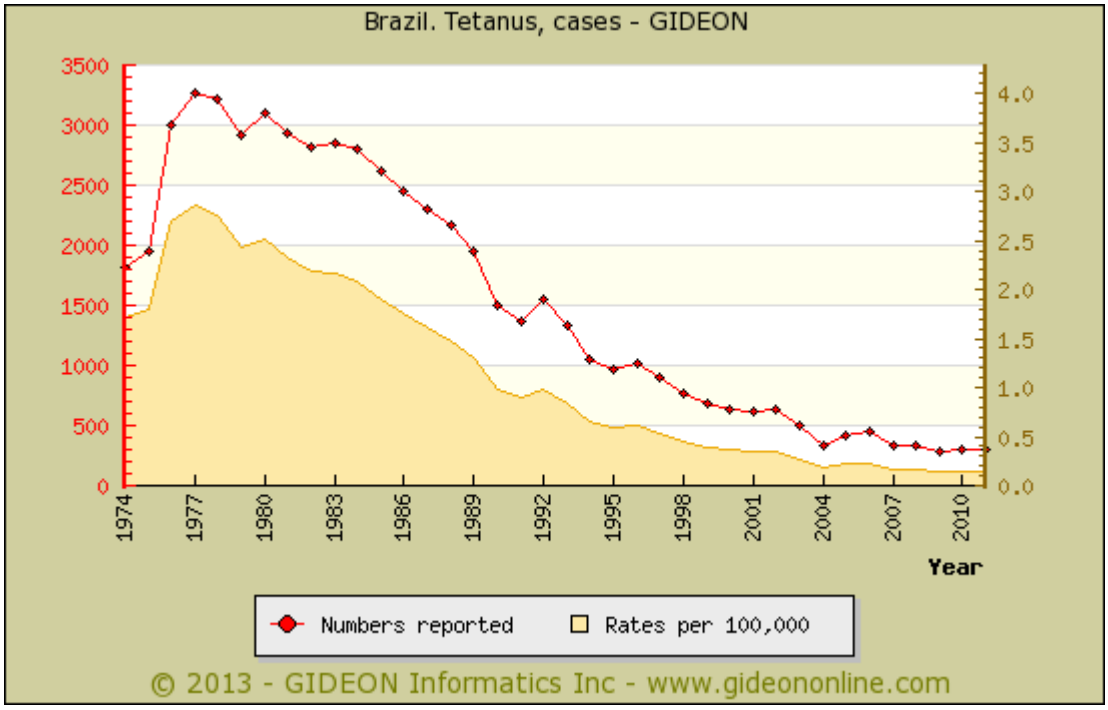
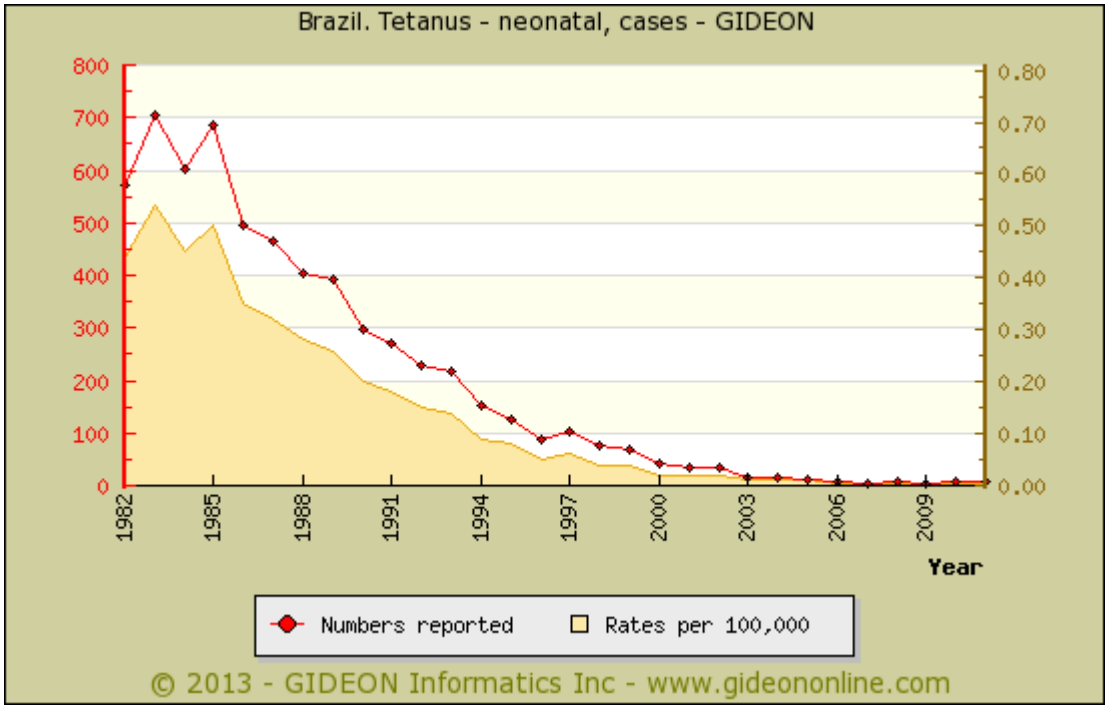


Graph: Brazil. Tetanus, cases

Notes:
1. 27,419 cases (total tetanus) were reported during 1982 to 2000.
2. 131 cases were reported from a referral hospital in Fortaleza during 2002 to 2005 [25]
3. 1,971 cases were reported in Pernambuco during 1981 to 2004 [26]

Tungiasis is implicated in the etiology of 10% of tetanus cases in Sao Paulo. [27]



Graph: Brazil. Tetanus - neonatal, cases

Notes:
1. 5,932 cases of neonatal tetanus were reported during 1982 to 2000.
2. 70 cases (48 fatal) were reported in Minas Gerais during 1989 to 1996. [28]

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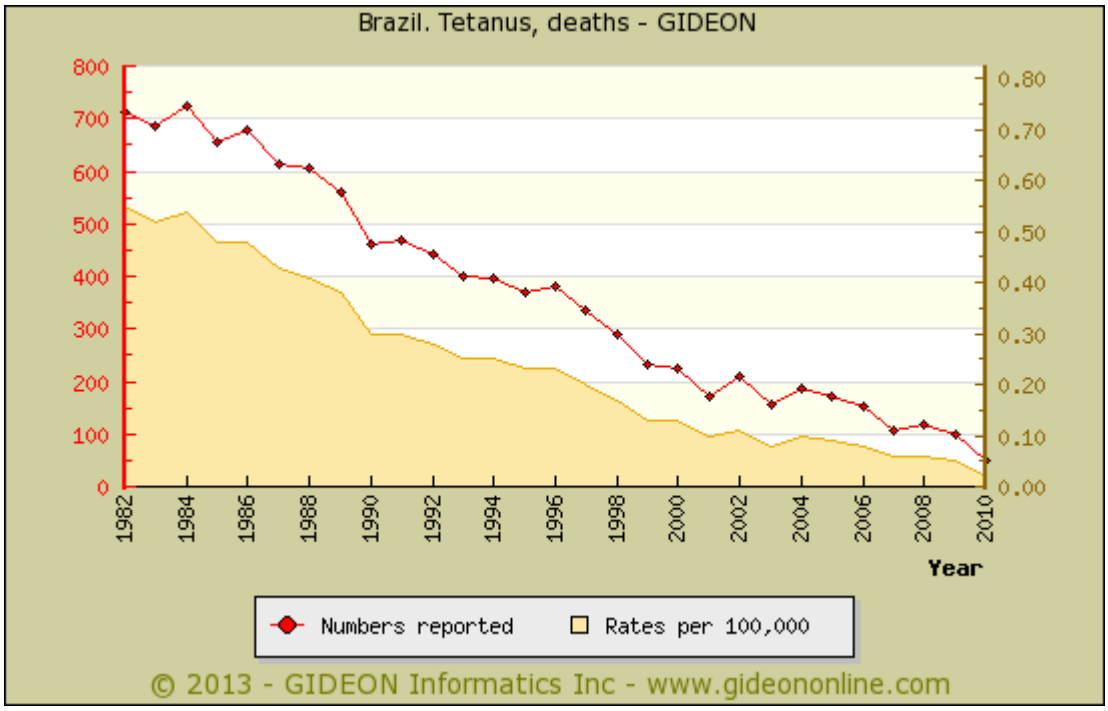
Individual years:
1993 - 31% of total cases for the region.
2004 - 10 fatal.



Graph: Brazil. Tetanus, deaths

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Thelaziasis

Agent	PARASITE - Nematoda. Phasmidea: Thelazia callipaeda [rarely T. californiensis]
Reservoir	Dog Rabbit Deer Cat
Vector	Fly (? Musca and Fannia species)
Vehicle	None
Incubation Period	not known
Diagnostic Tests	Identification of parasite.
Typical Adult Therapy	Extraction of parasite
Typical Pediatric Therapy	As for adult
Clinical Hints	Conjunctivitis and lacrimation associated with the sensation of an ocular foreign body.
Synonyms	Conjunctival spirurosis, Oriental eye worm, Rictularia, Thelazia californiensis, Thelazia callipaeda. ICD9: 372.15 ICD10: B83.8

Clinical

The signs and symptoms of Thelaziasis are related to the presence of a worm in the conjunctival sac, and consist of pain, lacrimation and a foreign body sensation. [1] [2]

This disease is endemic or potentially endemic to all countries.

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Toxic shock syndrome

Agent	BACTERIUM. Staphylococcus aureus, Streptococcus pyogenes, et al - (toxins) Facultative gram-positive cocci
Reservoir	Human
Vector	None
Vehicle	Tampon (occasionally bandage, etc) which induces toxinosis
Incubation Period	Unknown
Diagnostic Tests	Isolation of toxigenic Staphylococcus aureus. Toxin assay available in specialized laboratories.
Typical Adult Therapy	The role of topical (eg, vaginal) and systemic antistaphylococcal antibiotics is unclear
Typical Pediatric Therapy	The role of topical (eg, vaginal) and systemic antistaphylococcal antibiotics is unclear
Clinical Hints	Fever (>38.9), hypotension (<90 mm Hg) and dermal erythema with desquamation; respiratory, cardiac or other disease present; most cases associated with 'super absorbent' tampon use or staphylococcal wound infection; case-fatality rate = 5% to 10%.
Synonyms	Streptococcal toxic shock syndrome, TSS. ICD9: 040.82 ICD10: A48.3

Clinical

CDC (The United States Centers for Disease Control) case definition for surveillance:.
For surveillance purposes, the CDC (The United States Centers for Disease Control) case definition of toxic shock syndrome [1] requires an illness with the following clinical manifestations:
1. fever at least 38.9 C
2. diffuse macular erythema [2]
3. desquamation 1 to 2 weeks after onset of illness (particularly of the palms and soles)
4. hypotension (less than 90 mm Hg for adults, or less than fifth percentile if below age 16 years • or orthostatic hypotension)
5. multisystem involvement, consisting of three or more of the following: acute vomiting or diarrhea; myalgia and elevation of creatine phosphokinase levels; vaginal, oropharyngeal or conjunctival hyperemia; elevation of blood urea nitrogen or creatine to at least twice normal, or sterile pyuria; elevation of serum bilirubin or aminotransferase levels to at least twice normal; platelet count < 100,000/ cu mm; disorientation or alteration in consciousness unrelated to fever and hypotension
6. laboratory examination
• negative cultures of blood, throat or cerebrospinal fluid (however, *S. aureus* may be present in blood)
• negative tests for measles, leptospirosis or rickettsiosis

A probable case requires at least five of the above clinical findings. A confirmed case requires all six clinical findings (unless the patient dies before desquamation can occur).

The case definition for Streptococcal toxic shock syndrome [3] [4] includes isolation of *Streptococcus pyogenes* in addition to:
1. hypotension as above
2. multiorgan involvement characterized by at least two of the following (defined above)
• renal impairment
• coagulopathy
• hepatic dysfunction
• acute respiratory distress syndrome
• a generalized erythematous macular rash which may desquamate [5]
• soft tissue necrosis (fasciitis, myositis, gangrene).

This disease is endemic or potentially endemic to all countries.

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Toxocariasis

Agent	PARASITE - Nematoda. Phasmidea: Toxocara cati and canis
Reservoir	Cat Dog Mouse
Vector	None
Vehicle	Soil ingestion
Incubation Period	1w - 2y
Diagnostic Tests	Identification of larvae in tissue. Serology.
Typical Adult Therapy	Albendazole 400 mg BID X 5d. OR Mebendazole 100 to 200 mg PO bid X 5 days Add corticosteroids if eye, brain, heart or lung involvement is present.
Typical Pediatric Therapy	As for adult
Clinical Hints	Cough, myalgia, seizures, urticaria, hepatomegaly, pulmonary infiltrates or retrobulbar lesion; marked eosinophilia often present; symptoms resolve after several weeks, but eosinophilia may persist for years.
Synonyms	Ascaris suum, Toxocara canis, Toxocara cati, Toxocarose, Visceral larva migrans. ICD9: 128.0 ICD10: B83.0

Clinical

Most infections present in children below the age of 5 years, and are asymptomatic or mild; however rare instances of infection are reported in adults. [1]

Overt disease is characterized by fever, cough, wheezing, eosinophilia, myalgia, tender hepatomegaly and abdominal pain. [2]

• A tender nodular rash may be present on the trunk and legs.
• Chronic urticaria, chronic pruritus, relapsing eosinophilic cellulitis [3] and eczema are also reported. [4]
• Myocarditis [5] [6], pericarditis [7], pulmonary infiltrates, acute respiratory distress syndrome [8], seizures, nephritis, encephalopathy [9], spinal involvement (usually cervical or thoracic) including transverse myelitis [10] [11], encephalomyelitis [12], cerebral vasculitis [13], Bell's palsy [14], eosinophilic meningitis [15-17], eosinophilic pneumonia [18] or pleural effusion [19] [20], eosinophilic ascites [21] [22], eosinophilic abscesses of the liver [23] [24], cystitis [25] and renal dysfunction have been described in heavy infections.
• Eye disease is rare in toxocariasis. [26] Ocular toxocariasis usually presents in children ages 5 to 10 years, and is characterized by formation of a retinal granuloma at or near the macula, resulting in strabismus, iridocyclitis, glaucoma, papillitis or visual loss. [27-32]
• Toxocariasis has been identified as a cause of chronic cough in childhood [33] and of diminished lung function (FEV-1) at any age. [34]
• In some cases, pulmonary and hepatic nodular lesions could be mistaken for malignancy. [35] [36]
• Toxocariasis has been implicated in the etiology of epilepsy [37] and decreased cognitive function among children. [38]

Ascaris suum, a parasite of pigs, has been reported to cause rare cases of myelitis [39], encephalopathy [40], eosinophilic pneumonia [41-43] and focal liver lesions in humans. [44-48]
• *A. suum* has been implicated in cases of eosinophilic colitis [49] and intestinal obstruction. [50]

This disease is endemic or potentially endemic to all countries.

Toxocariasis in Brazil

Hepatic visceral larva migrans granulomas are found in 3.2% of autopsies among children ages 1 to 15 years.
- Toxocara antibodies are detected in 30 to 39% of these children, reflecting a low larval burden in infected children. (Vitoria, 2007 publication) [51]

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Prevalence surveys:

5% of dogs in Sao Paulo State (2002 publication) [52]
14.5% of fecal samples of stray dogs from Itapema City, Santa Catarina (2005 publication) [53]
4.2% of fecal samples from dogs in public squares in Itabuna, Bahia (2008 publication) [54]
9.3% of dog feces from central area of Cassino beach, Rio Grande do Sul (2003 publication) [55]
39.0% of dogs and 29.7% of soil samples in San Remo, Sao Paulo (2005 publication) [56]
8.7% of dogs in Botucatu, Sao Paulo (2008 publication) [57]
8.7% of dogs in Botucatu, Sao Paulo (2010 publication) [58]
8.7% of stray dogs in Fortaleza (2010 publication) [59]
15.5% of dogs in Minas Gerais State (2012 publication) [60]
100% of soil samples from public squares, 18.9% from peridomiciles, 23.1% from schools, and 29.3% of dogs (2012 publication) [61]
68.1% of soil samples from public parks and squares in Guarulhos, Sao Paulo State (2010) [62]
24% of hair samples from dogs (Rio Grande do Sul, 2010 publication) [63]
25.2% of cats of the metropolitan region of Rio de Janeiro (2004 publication) [64]
43.1% of cats in Andradina City, Sao Paulo (2007) [65]
12.3% to 14.0% of soil samples in Campinas (1999)
29.03% of soil samples in the Pontal do Paranapanema region, Sao Paulo State (2008 publication) [66]
79.3% of soil samples in Fernandopolis, Sao Paulo State (2007 to 2008) [67]
96% of soil samples from public parks in Presidente Prudente, Sao Paulo (2012 publication) [68]
44.7% of soil samples from grassy areas and 21.4% from sandy areas (Parana, 2005 to 2007) [69]
10.8% of beach sand samples from Santos (2004 to 2005) [70]

Seroprevalence surveys:

23.9% of persons in Campinas (1999)
52% of persons aged 0 to 76 years (*Toxocara canis*) (Waimiri and Atroari settlements, Balbina, 2006) [71]
26.8% of persons in rural Acre (2004) [72]
46.3% of blood donors in Salvador (2011 publication) [73]
21.5% of persons in rural Sao Paulo (2008 publication) [74]
13.7% of persons in urban Sao Paulo (2009 publication) [75]
11.1% of middle-class and 9.5% of disadvantaged children in Sao Paulo State (2011 publication) [76]
21.8% of lower class, and 3% of upper class children in Distrito Federal
38.8% of school children in the Butanta region of Sao Paulo
12.1% of school children in suburban Recife (2005 publication) [77]
36.8% of children ages 0 to 12 years (Parana, 2005 to 2007) [78]
26.9% of children in San Remo, Sao Paulo (2005 publication) [79]
51.6% of 7-year-old school children in Vitoria, Espirito Santo (2011 publication) [80]
8.7% of children ages 1 to 15 years hospitalized in Uberlandia, Minas Gerais State (2006 publication) [81]
21.5% of children aged 6 months to 5 years in Northwestern Brazil (2007 publication) [82]
28.8% of children (ages 7 months to 12 years) in Maringa (South Brazil, 2004 to 2005) [83]
50.6% of children in Pelotas City, Rio Grande do Sul (2012 publication) [84]
51.6% of children ages 1 to 12 years in Parana State (2005 to 2007) [85]
47% of children ages 4 to 11 years in Salvador, Bahia (2012 publication) [86]
17.8% of children (2012 publication) [87]
82.7% of dogs in Salvador (2011 publication) [88]
50.1% of sheep in Presidente Prudente, southeastern Brazil (2011 publication) [89]

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Toxoplasmosis

Agent	PARASITE - Protozoa. Sporozoa, Coccidea, Eimeriida: Toxoplasma gondii
Reservoir	Rodent Pig Cattle Sheep Chicken Bird Cat Marsupial (kangaroo)
Vector	None
Vehicle	Transplacental Meat ingestion Soil ingestion Water or milk (rare) Fly
Incubation Period	1w - 3w (range 5d - 21d)
Diagnostic Tests	Serology. Cultivation or identification of organisms per specialized laboratories. Nucleic acid amplification.
Typical Adult Therapy	Pyrimethamine 25 mg/d + Sulfonamides 100 mg/kg (max 6g)/d X 4w - give with folinic acid. Alternatives: Clindamycin, Azithromycin, Dapsone. Spiramycin (in pregnancy) 4g/d X 4w
Typical Pediatric Therapy	Pyrimethamine 2 mg/kg/d X 3d, then 1 mg/kg/d + Sulfonamides 100 mg/kg/d X 4w - give with folinic acid. Alternatives: Clindamycin, Azithromycin, Dapsone.
Clinical Hints	Fever, lymphadenopathy and hepatic dysfunction; chorioretinitis; cerebral cysts (patients with AIDS); congenital hydrocephalus, mental retardation or blindness.
Synonyms	Toxoplasma, Toxoplasmose, Toxoplasmosi. ICD9: 130 ICD10: B58

Clinical

Acquired toxoplasmosis:
The clinical features of acquired toxoplasmosis can range from subclinical infection to lymphadenopathy (the most common presentation) to fatal, fulminant disease.
• In healthy adults, infection is usually subclinical, or mimics infectious mononucleosis; however, pharyngitis, posterior and posterior cervical lymphadenopathy are unusual in toxoplasmosis.
• Most patients with acute *Toxoplasma* lymphadenitis experience fatigue, headache, difficulty concentrating and muscle aches. [1]
• In immunocompromised hosts, toxoplasmosis may mimic other opportunistic infections, such as tuberculosis or infection with *P. jiroveci* (formerly *P. carinii*) [2] , or extensive varicella. [3]
• In patients with AIDS, CNS involvement is the most common manifestation, followed by pulmonary disease. [4]

Congenital toxoplasmosis:
The rate and severity of congenital toxoplasmosis are largely related to gestational age at the time of infection. [5] [6]
• Overt clinical signs appear to be more common among American infants vs. European infants with congenital toxoplasmosis. [7] [8]
• The brain and eyes are often affected, presenting as chorioretinitis, hydrocephalus, intracranial calcifications, and seizures. [9]
• 97% of children infected during the first trimester of pregnancy and having normal antenatal ultrasounds are asymptomatic or only slightly affected. [10]
• Rare instances of nephrotic syndrome complicating congenital toxoplasmosis have been reported. [11]

Ocular toxoplasmosis: [12]
Ocular toxoplasmosis occurs from reactivation of cysts in the retina.
• Focal necrotizing retinitis is characteristic lesion, and approximately 35% of all cases of retinochoroiditis can be attributed to toxoplasmosis. [13]
• Risk factors for early (first two years of life) retinochoroiditis include a delay of >8 weeks between maternal seroconversion and the beginning of treatment, female gender, and the presence of cerebral calcifications. [14]
• The incidence and severity of ocular toxoplasmosis varies from country to country. [15]

CNS toxoplasmosis:
The manifestations of CNS toxoplasmosis in the immunocompromised patient range from an insidious process evolving over several weeks to acute onset of a confusional state.
• Signs may be focal or symmetrical.
• *T. gondii* has a predilection to localize in the basal ganglia and brain stem, producing extrapyramidal symptoms resembling those of Parkinson's disease.

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• A normal CT scan does not rule out cerebral toxoplasmosis. MRI is the imaging modality of choice [16]
• Nonfocal evidence of neurological dysfunction may include generalized weakness, headache, confusion, lethargy, alteration of mental status, personality changes, and coma.
• Infection in transplant recipients is often diffuse and disseminated.
• In patients with underlying malignancy (e.g. Hodgkin's disease), the presentation is evenly distributed between focal and nonfocal forms of encephalitis.

Toxoplasmosis and AIDS:
Patients with AIDS tend to present subacutely with nonspecific symptoms such as neuropsychiatric complaints, headache, fever, weight loss, disorientation, confusion, and lethargy evolving over 2 to 8 weeks.
• Later findings include evidence of focal CNS mass lesions, ataxia, aphasia, hemiparesis, visual field loss, vomiting, confusion, dementia, stupor and seizures. [17]
• Toxoplasmosis presenting as subcutaneous mass has been reported among HIV-positive patients. [18] [19]
• Primary cerebral lymphoma in AIDS patients may be mistaken for Toxoplasmosis. [20]

This disease is endemic or potentially endemic to all countries.

Toxoplasmosis in Brazil

A review of the burden of toxoplasmosis in Brazil (2012) - see reference. [21]

Toxoplasmosis is the most common cause of infectious uveitis in Brazil. [22]

88 deaths were ascribed to toxoplasmosis in 1996; 82 in 1997; 94 in 1998.

Contaminated unfiltered drinking water is a common vehicle for toxoplasmosis in Rio de Janeiro [23] and other areas of Brazil. [24]

Three cases of toxoplasmosis in France were acquired through ingestion of raw horse meat imported from Canada and Brazil. [25]

Congenital toxoplasmosis:
The nationwide incidence of congenital toxoplasmosis is 53.5 per 100,000 live births. An estimated 2,649 children are born with congenital toxoplasmosis yearly (2012 publication). [26]
- The incidence of congenital toxoplasmosis in southern Brazil (Rio Grande do Sul) is 80 per 100,000 live births.
- The incidence of congenital toxoplasmosis in Porto Alegre in 6 per 10,000 infants (2007 publication) [27]
- The incidence of congenital toxoplasmosis in Sergipe is 4 per 10,000 infants (2009) [28]
- The incidences of acute toxoplasmosis among pregnant women and congenital toxoplasmosis in Porto Alegre were 4.8/1,000 and 0.9/1,000, respectively (1998 to 2005) [29]
- 17.7% of persons in Erechim (southern Brazil) have ocular toxoplasmosis. [30]
- The incidence of acute toxoplasmosis among pregnant women in Parana is 10.7/1,000 births (2001 to 2003) [31]

261 cases of acute acquired toxoplasmosis were treated at an infectious diseases reference center during 1992 to 2004. [32]

Prevalence surveys:
0.4% of solid organ transplant recipients (2001 to 2006) [33]
3.8% of the general population and 5.8% of seropositive individuals in Barra Mansa, Rio de Janeiro State (ocular toxoplasmosis, 2009 publication) [34]
4.8% of HIV-positive patients in the southern region (history of neurotoxoplasmosis, 2009 to 2010) [35]
10% of autopsied AIDS patients (cause of death, Amazonas, 1996 to 2003) [36]
7.84% of slaughtered goats in Bahia (2009 publication) [37]
34% and 66% of porcine diaphragm and tongue, respectively, in Southern Brazil. (2007 publication) [38]
22.58% of soil samples from school playgrounds in northwestern Sao Paulo (2010 publication) [39]

Seroprevalence surveys:
71% of persons ages 16 to 20 in Rio de Janeiro City (1987)
84% of low income groups in Rio de Janeiro (1997 to 1999)
73.3% of persons in farming areas of rural Rondonia state (2006 publication) [40]
79.45% of persons in farming areas of rural Mato Grosso do Sul (2008 publication)
73.5% of Indians in Lauarete, Sao Gabriel da Cachoeira, Amazonas (2008 publication) [41]
97.4% on farms in southwestern Mato Grosso State (2009 publication) [42]

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65.8% of rural settlers ages 5 to 90 years, in Amazonia (2004) [43]
65.9% of persons in Barra Mansa, Rio de Janeiro State (2009 publication) [44]
64.9% of women at childbearing age in Metropolitan Sao Paulo (2009 publication) [45]
33.8% of female nursing students in Presidente Prudente, Sao Paulo (2010 publication) [46]
56.4% of pregnant women in Bahia (2011 publication) [47]
54.2% of pregnant women in Cascaval, Parana (2008 publication) [48]
69.7% of pregnant women in Fortaleza, Ceara State (2003)
68.8% of pregnant women in Fortaleza (2010 publication) [49]
67.7% of pregnant women in Goias (2008) [50]
66.3% of pregnant women in Natal (2007) [51]
67% of pregnant women in Parana (IgG, 1996 to 1998)
59.0% of pregnant women in Parana (IgG, 2010) [52]
55.3% of pregnant women in Parana (IgG and/or IgM, 2007 to 2008) [53]
60.6% of pregnant women in western Parana (2012 publication) [54]
59.8% of pregnant women in Porto Alegre (2000)
74.7% of pregnant women in Recife. 2.8% had possible active infection (2004 to 2005) [55]
74.5% of pregnant women in Rio Grande do Sul (1997 to 1998) [56]
62.1% of pregnant women in Sao Paulo (2011 publication) [57]
64.4% of pregnant women in Sao Jose do Rio Preto, Sao Paulo (2011 publication) [58]
69.3% of pregnant women in Sergipe (2009 publication) [59]
53.03% of pregnant women in southern Brazil (2003 to 2004) [60]
67% of HIV-negative and 72% of HIV-positive pregnant women in Rio Grande do Sul (2002 to 2005) [61]
80% of HIV-positive patient in the southern region (2009 to 2010) [62]
55.6% of children age <1 year in Maringa municipality, Parana (2003 to 2004)
56% of infants with suspected congenital infection in Goiania (2004 to 2005) [63]
42.2% of school children in Rolandia, Parana (1998)
46.4% of elementary school children in Jataizinho, Parana (2008 publication) [64]
79.0% of male and 63.4% of female blood donors in Recife (2001)
33.3% of liver transplant recipients (1997 to 2007) [65]
78.8% of Enawene-Nawe Indians, 59.6% of Waiapi and 55.6% of Tiriyo (2005 publication) [66]
70.0% of slaughterhouse workers in northern Para (2006 publication) [67]
92% of meat inspectors working in slaughter houses (Belo Horizonte, 1975 publication) [68]

Seroprevalence surveys in animals:

26.7% of stray cats and 40.6% of breeder cats in Sao Paulo; 84.4% of domestic cats in Parana State.
87.3% of cats and 37.5% of pigs in rural western Amazon (2006 publication) [69]
15.7% of cats in Andradina Municipality, Sao Paulo State (2011 publication) [70]
25.75% of cats with *Leishmania chagasi* infection were found to be coinfected by *Toxoplasma gondii* (Sao Paulo State, 2012 publication) [71]
16.3% of cats in Curituba, Parana State (2011 publication) [72]
50.5% of cats with outdoor access, in Sao Luis, Maranhao (2012 publication) [73]
23.80% of dogs with neurological signs, in Curituba, Parana (2011 publication) [74]
14.33% of domiciled cats in Lages, southern Brazil (2010 publication) [75]
84.4% of domestic cats in Santa Isabel do Ivai, Parana State (2004 publication) [76]
22.3% of domestic dogs in Lages and Balneario Camboriu, Santa Catarina State (2009 publication) [77]
69.8% of dogs in Para State (2010 publication) [78]
60.7% of dogs in Lavras, Minas Gerais (2009 publication) [79]
52% of dogs in the southern Amazon region (2012 publication) [80]
18.0% of dogs in Piaui (2011 publication) [81]
22% of dogs with neurological signs, in Botucatu (2012 publication) [82]
20.4% of opossums (*Didelphis marsupialis*)
24.5% of goats slaughtered in the public slaughterhouse of Patos city, Paraiba State, Northeast region (2007 publication) [83]
14.5% of dairy goats in Sao Paulo
30.6% of goats in Serido Oriental microregion, Rio Grande do Norte state (2008 publication) [84]
32.2% of goats (2008 publication) [85]
43.0% of goats in Minas Gerais (ELISA, 2008 publication) [86]
8.54% of swine and 7.0% of ovines slaughtered in Guarapuava (2007 publication) [87]
4.5% of wild boar in Sao Paulo (2009 publication) [88]

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51.5% of sheep and 20.8% of dogs in Parana (2007 publication) [89]
24.2% of sheep in Sao Paulo State (2008 publication) [90]
32.9% of farm sheep in Alagoas (2009 publication) [91]
25.75% of sheep in Curitiba, Parana State (2009 publication) [92]
29.1% of sheep in Northeast Brazil. (Rio Grande do Norte State, 2007 publication) [93]
38.22% of sheep in Federal District (2008 publication) [94]
20.7% of sheep in Mossoro, Rio Grande do Norte (2008 publication) [95]
52% of sheep in the Jaboticabal microregion, Sao Paulo State (2009 publication) [96]
75% of sheep in Uberlandia, Minas Gerais (ELISA, 2010 publication) [97]
10.96% of sheep in slaughterhouses (2010 publication) [98]
18.6% of sheep from the eastern region of Sao Paulo State (2011 publication) [99]
100% of sheep farms in Santa Catarina (2012 publication) [100]
4.35% of goats and 18.75% of sheep in western Maranhao (2011 publication) [101]
39% of goats in Alagoas State (2011 publication) [102]
18.1% of goats in Paraiba State (2012 publication) [103]
39.41% of goats in Curitiba, Parana (ELISA, 2012 publication) [104]
71.0% of cattle and 88.5% of dogs on farms in southwestern Mato Grosso State (2009 publication) [105]
19.3% of slaughtered cattle in Ilheus, 9.8% in Itabuna and 6.8% in Jequie (Bahia State, 2009 publication) [106]
26% of slaughtered cattle in Bahia State (2009 publication) [107]
49.4% of cattle and 11.5% of pigs in Campos dos Goytacazes, northern of Rio de Janeiro State (2011 publication) [108]
1.96% of cattle and 7.64% of pigs in Rio de Janeiro State (2011 publication) [109]
48.3% of pregnant dairy cows (*Bos. taurus*) and 3.7% of cow fetuses in southern Brazil (2012 publication) [110]
23.8% of mules and 43.2% of donkeys in Pernambuco, Rio Grande do Norte, Paraiba, and Sergipe (2012 publication) [111]
12.8% of swine matrices in Nova Mutum and Diamantino, Mato Grosso State (2010 publication) [112]
9.6% of pigs (2000 publication) [113]
13.4% of pig breeding farms in the Toledo microregion of Parana (2010 publication) [114]
18.27% of swine raised for slaughter (Bahia, 2009 publication) [115]
36.2% of swine slaughtered in Patos, Paraiba State (2010 publication) [116]
12.5% of slaughtered pigs in Pernambuco (2012 publication) [117]
80% of chickens (*Gallus domesticus*), 3.0% of (*Bos taurus*), 60.8% of sheep (*Ovis aries*), 81.8% of goats (*Capra hircus*), 43.7% of horses (*Equus caballus*), 59.3% of cats (*Felis catus*), 39.6% of dogs (*Canis familiaris*), 38.2% of black rats (*Rattus rattus*), and 79.7% of cattle egrets (*Bubulcus ibis*) (Fernando de Noronha archipelago, 2007 to 2010) [118]
30.2% of *Cebus* spp. (capuchin monkeys) and 17.6% of *A. caraya* (black and golden howler monkeys) in Parana State (2005 publication) [119]
46% of jaguarundi (*Puma yagouaroundi*); 58% of ocelot (*Leopardus pardalis*); 50% of oncilla (*L. tigrinus*); 54% of margay (*L. wiedii*); 12% of Pampas-cat (*L. colocolo*); 83% of Geofroy's-cat (*L. geoffroyi*); 64% of jaguar (*Panthera onca*) and 48% of puma (*Puma concolor*) (1995 to 2001) [120]
53.3% of free range chickens in Pernambuco, Rio Grande do Norte, Maranhao, Bahia, Ceara, Sergipe, and Alagoas (2008 publication) [121]
40.4% of free-range chickens in Espirito Santo State (2012 publication) [122]
1.02% of sparrows (*Passer domesticus*) in Bahia and Pernambuco (2009 publication) [123]
5% of urban pigeons (*Columba livia*, 2010 publication) [124]
1% of feral pigeons (*Columba livia*) in Jaboticabal (2011 publication) [125]
0% of free-living pigeons in Sao Paulo, Ibiuna and Sorocaba, Sao Paulo State (2010 publication) [126]
75% of capybaras (*Hydrochaeris hydrochaeris*) in Sao Paulo State (IFTA, 2008 publication) [127]
42.3% (MAT) to 69.8% (IFAT) of capybaras
61.5% of capybaras (2010 publication) [128]
19% of Brazilian agouti (*Dasyprocta aguti*, 2011 publication) [129]
10% of free-ranging Brazilian tapirs (*Tapirus terrestris*) in Goias state (one animal seropositive, 2010 publication) [130]
12.9% of nine-banded armadillos (*Dasypus novemcinctus*) (Sao Paulo, 2008 publication) [131]
19.2% of crab-eating foxes (*Cerdocyon thous*) in zoos (2009 publication) [132]
63.4% of wild felids maintained in Brazilian zoos (2010 publication) [133]
75% of wild-maned wolves (*Chrysocyon brachyurus*) in Minas Gerais (2003 to 2008) [134]
66.67% of neotropical felids (*Leopardus geoffroyi*, *Puma yagouaroundi*, *Leopardus wiedii*, *Leopardus tigrinus* and *Leopardus pardalis*, 2010 publication) [135]
0% of vampire bats (*Desmodus rotundus*) in Botucatu region, Sao Paulo (2009 publication)
34.8% of mammals from zoos and breeding facilities in the north and northeast regions (2010 publication) [136]
0.9% of urban rodents in Sao Paulo (2011 publication) [137]
5.5% of opossums (*Didelphis albiventris*) in Botucatu Municipality, Sao Paulo (2008 to 2009) [138]

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1.35% of rabbit meat juices (2011 publication) [139]
86.3% of Amazon River dolphins (*Inia geoffrensis*) from a reserve in Tefe, Amazonas (2011 publication) [140]
39.2% of captive manatees (*Trichechus inunguis*) in northern Brazil (2012 publication) [141]

Acute disseminated toxoplasmosis has been reported in a captive three-toed sloth (*Bradypus tridactylus*).
- Infection has also been documented in nine-banded (*Dasypus novemcinctus*) and six-banded (*Euphractus sexcinctus*) armadillos. [142]
- An outbreak of toxoplasmosis was reported in common wooly monkeys (*Lagothrix lagotricha*) [143]
- Toxoplasmosis has been identified in a Guiana dolphin (*Sotalia guinensis*) from Paranagua Bay, Parana [144]
- Toxoplasmosis has been identified in bats. [145]

Notable outbreaks:

1967 (publication year) - An outbreak of toxoplasmosis was reported in a Paulist seminary in Braganza, Sao Paulo State. [146]

1969 (publication year) - An outbreak of toxoplasmosis was reported in a university in Sao-Jose-dos Campos, Sao Paulo. [147]

1982 (publication year) - An outbreak of toxoplasmosis was reported in a rural area. [148]
1993 - An outbreak (20 cases) in Parana was ascribed to contaminated mutton. [149]
2001 - An outbreak (176 cases) in Parana was ascribed to contaminated water. [150-152]
2002 - An outbreak (426 cases) in Parana was ascribed to contaminated water. [153]
2011 - An outbreak (30 cases) was reported in Mato Grosso. [154]
2012 - An outbreak (34 cases) among employees of an industrial plant in Sao Paulo was associated with ingestion of vegetables. [155]

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Trachoma

Agent	BACTERIUM. Chlamydia trachomatis, type A
Reservoir	Human
Vector	Fly
Vehicle	Infected secretions Fly Fomite
Incubation Period	5d - 12d
Diagnostic Tests	Culture or direct immunofluorescence of secretions. Serology. Nucleic acid amplification.
Typical Adult Therapy	Azithromycin 20 mg/kg as single dose. OR Doxycycline 100 mg/day PO X 14 days. Also administer topical Tetracycline
Typical Pediatric Therapy	Erythromycin 10 mg/kg PO QID X 4w. Also administer topical Tetracycline
Clinical Hints	Keratoconjunctivitis with palpebral scarring and pannus formation; 0.5% of infections result in blindness.
Synonyms	Egyptian ophthalmia, Granular conjunctivitis, Kornerkrankheit, Trachom, Tracoma. ICD9: 076 ICD10: A71

Clinical

Early symptoms include erythema and swelling of both bulbar and palpebral conjunctivae, associated with a watery or purulent discharge. [1] [2]
• Additional findings may include preauricular lymphadenopathy and rhinitis.
• Examination reveals follicular hypertrophy and conjunctival scarring.
• Corneal scars (Herbert's pits), punctate keratitis and pannus formation may also be present. [3]
• As scarring progresses, the eyelashes deviate (entropion) and may produce additional trauma and ulceration of the conjunctivae. [4]
• Reinfection and bacterial superinfection are common [5] and may contribute to the progression of follicular trachoma. [6]

Trachoma may be differentiated from inclusion conjunctivitis by the presence of corneal scarring and a preference of the latter for the upper tarsal conjunctivae

This disease is endemic or potentially endemic to all countries.

Trachoma in Brazil

136,497 cases were reported in 1980.

1,064,218 cases of active trachoma were estimated in 2003.

Prevalence surveys:

11.9% of children in Botucatu, Sao Paulo in 1992 [7] ; 2.9% in 2005 [8]
3.8% of school children ages 6 to 14 in Bauru, Sao Paulo State (2005) [9]
4.5% of students in grades 1 through 4 in Roraima (2011 publication) [10]
3.1% of public-school students in Embu das Artes, Sao Paulo (inflammatory trachoma, 2003 to 2004) [11]
2.2% of pre-school and school children in Sao Paulo (1999) [12]
4.7% of pre-school children in central Sao Paulo (inflammatory trachoma, 2006 publication) [13]
4.9% of male and 4.2% of female school children in Alagoas (2009 publication) [14]
50.9% to 56.4% among native populations in the upper Rio Negro Basin (Amazonas State, 2002 publication) [15]
26.3% of rural villagers in the Araripe plateau region of Ceara State (2010 publication) [16]
7.2% in Bebedouro State (1992 publication) [17]
8.9% in Sao Gabriel da Cachoeira, upper Rio Negro Basin (2008 publication) [18]

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30.3% of Yanomami Indians in Amazonas State (2002 publication) [19]
5.8% of patients presenting with "allergic conjunctivitis" (2010 publication) [20]

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Trichinosis

Agent	PARASITE - Nematoda. Adenophorea: Trichinella spiralis (occasionally T. nativa, T. britovi, T. pseudospiralis, T. nelsoni, et al)
Reservoir	Wild carnivore Omnivore Marine mammal
Vector	None
Vehicle	Meat ingestion
Incubation Period	10d - 20d (range 1w - 10w)
Diagnostic Tests	Identification of larvae in tissue. Serology.
Typical Adult Therapy	Albendazole 400 mg PO BID X 14d. OR Mebendazole 200 to 400 mg PO tid X 3 days, then 400 to 500 mg PO. tid X 10 days. Give with prednisone 50 mg PO daily X 3 to 5 days (then 'taper' dosage)
Typical Pediatric Therapy	Albendazole 7 mg/kg BID X 14 d. OR Mebendazole 200 to 400 mg PO tid X 3 days, then 400 to 500 mg PO. tid X 10 days. Give with prednisone 50 mg PO daily X 3 to 5 days (then 'taper' dosage)
Clinical Hints	Early diarrhea and vomiting; subsequent myalgia, facial edema and eosinophilia; onset 1 to 4 weeks following ingestion of undercooked meat (usually pork); symptoms may persist for two months; case-fatality rate for symptomatic infection = 2%.
Synonyms	Trichinellose, Trichinellosis, Trichinose, Trikinose, Triquiniase, Triqunosis. ICD9: 124 ICD10: B75

Clinical

The great majority of infections are subclinical.
• The development of symptoms depends on the number of larvae ingested.

Signs and symptoms:

During the first week of illness, the patient may diarrhea, abdominal pain and vomiting. [1-3]
• Symptoms associated with larval invasion appear during the second week and include fever, periorbital edema, subconjunctival hemorrhages and chemosis. [4]
• Myositis is also common, and often appears in the extraocular muscles, progressing to involve the masseters, neck muscles, limb and lumbar muscles.
• Additional symptoms may include headache, cough, dyspnea, hoarseness and dysphagia.
• Occasionally, a macular or petechial rash, or retinal or subungual splinter hemorrhages are seen.
• Hepatomegaly is common. [5]
• Laboratory studies may reveal marked eosinophilia, hypoalbuminemia, decreased erythrocyte sedimentation rate, proteinuria or hematuria.
• Rare instances of renal dysfunction [6] , encephalitis [7] and eosinophilic meningitis have been reported. [8]

Clinical course:

• Systemic symptoms usually peak 2 to 3 weeks after infection and then slowly subside; however, weakness may persist for weeks.
• A number of clinical findings may persist for several months: hypocalcemia, hypomagnesemia, fatigue, myalgia (notably in the legs), cardiovascular disorders, neurological, psychiatric, and allergic illnesses. [9]
• Deaths are ascribed to myocarditis [10] [11] , encephalitis or pneumonia.

This disease is endemic or potentially endemic to all countries.

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Trichomoniasis

Agent	PARASITE - Protozoa. Archezoa, Parabasala, Trichomonadea. Flagellate: Trichomonas vaginalis
Reservoir	Human
Vector	None
Vehicle	Sexual contact
Incubation Period	4d - 28d
Diagnostic Tests	Microscopy of vaginal discharge. ELISA, culture, antigen detection tests available. Nucleic acid amplification.
Typical Adult Therapy	Metronidazole or Tinidazole 2g PO as single dose to both sexual partners
Typical Pediatric Therapy	Metronidazole 5 mg/kg PO TID X 7d. OR Tinidazole 50 mg/kg PO X 1 (maximum 2 grams)
Clinical Hints	Vaginal pruritus, erythema and thin or frothy discharge; mild urethritis may be present in male or female.
Synonyms	Pentatrichomonas, Tetratrichomonas, Trichomonaden, Trichomonas, Trichomonas vaginalis, Tricomoniasis, Tritrichomonas. ICD9: 131 ICD10: A59

Clinical

10% to 50% of infections are asymptomatic.
• Symptoms often begin or worsen during the menstrual period.
• Infection is usually characterized by vaginal discharge and vulvovaginal irritation. [1]
• Dysuria may be present, and dyspareunia is common.
• As many as two thirds of infected women complain of a disagreeable odor.
• Abdominal discomfort is present in 5% to 12%.

Examination reveals a copious loose discharge that pools in the posterior vaginal fornix. [2]
• The discharge is yellow or green in 5% to 40%, and bubbles are observed in the discharge in 10% to 33%. [3]
• The material has a pH above 4.5 in 66% to 91% of cases.
• Endocervical disease is not caused by *T. vaginalis*.
• Punctate hemorrhages (colpitis macularis or "strawberry cervix") are seen on colposcopically in 45% of infected women, but in only 2% by visual inspection alone.
• Parasites can be recovered from the urethra and paraurethral glands in more than 95% of the women, and may explain the association of the infection with urinary frequency and dysuria.

Reported complications of trichomonal vaginitis include vulvar ulceration [4] , and vaginitis emphysematosa • the presence gas-filled blebs in the vaginal wall. [5]
• Gestational trichomoniasis may be associated with premature labor and low birth weight, postabortal infection or premature rupture of the membranes.
• Spread of trichomonads beyond the lower urogenital tract is extremely rare.
• Sporadic cases of neonatal pneumonia due to *Trichomonas vaginalis* are reported. [6]

Trichomoniasis has been associated with endometritis, adnexitis, pyosalpinx, infertility, preterm birth, low birth weight, bacterial vaginosis, and increased risk of cervical cancer, HPV, and HIV infection. [7]
• In men, its complications include urethritis, prostatitis, epididymitis, and infertility through interference with sperm function. [8]

Most men carrying trichomonads are asymptomatic; however, the organism is implicated in 5% to 15% of patients with nongonococcal urethritis.
• The discharge from trichomonal urethritis is usually milder than that seen with other infections.
• Epididymitis, superficial penile ulcerations (often beneath the prepuce) and prostatitis are also described.

Tritrichomonas foetus pneumonia [9] and peritonitis have been reported in immunosuppressed patients. [10]
• *Trichomonas tenax* has been reported to cause pneumonia in an immunosuppressed patient. [11]

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This disease is endemic or potentially endemic to all countries.

Trichomoniasis in Brazil

6,139,000 cases were estimated for 1996.

Prevalence surveys:

2.1% of pregnant women (1991 to 1993)
10% of women in rural Alagoas (1997)
1.44% of women using Public Health Services in Mawringa, Parana (2004 to 2007) [12]
4.1% of sexually-active women (ages 12 to 49) in Ceara, Northern Brazil (2007 publication) [13]
2% of women ages 14 to 49 attending a primary health unit (2008 publication) [14]
3.2% of women ages 18 to 40 years, in Sao Paulo (2011 publication) [15]
2.9% of women in Serra Pelada, Para State (2004) [16]
0.64% of female CSW in Sao Paulo (2007 publication) [17]
12.5% of HIV-positive women (2008 publication) [18]
5.25% of women with atypical vaginal squamous cytology, vs. 3.98% of controls (2008 publication) [19]
3.56% of women ages 19 to 44 submitting Papanicolaou smears, in Sergipe (2004 to 2005) [20]

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Trichostrongyliasis

Agent	PARASITE - Nematoda. Phasmidea: Trichostrongylus colubriformis, T. orientalis, T. probolurus
Reservoir	Herbivore
Vector	None
Vehicle	Water Food Vegetation
Incubation Period	21d
Diagnostic Tests	Identification of ova in stool or duodenal aspirate.
Typical Adult Therapy	Albendazole 400 mg PO X 1. OR Pyrantel pamoate 11 mg/kg (max 1g) PO once. OR Mebendazole 100 mg PO BID X 7d
Typical Pediatric Therapy	As for adult
Clinical Hints	Diarrhea, abdominal pain and weight loss; eosinophilia is often present; infestation may persist for years; fatality and sequelae are not reported.
Synonyms	Haemonchus, Marshallagia, Ostertagia, Trichostrongylus. ICD9: 127.6 ICD10: B81.2

Clinical

Most infections are asymptomatic, or characterized by mild nonspecific abdominal symptoms.
- Heavy infections may result in episodic diarrhea, abdominal pain and weight loss. [1]
- Rare instances of cholecystitis are reported.

This disease is endemic or potentially endemic to 38 countries.

Trichostrongyliasis in Brazil

Trichostrongylus colubriformis is present in environmental samples. [2]

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Trichuriasis

Agent	PARASITE - Nematoda. Adenophorea: Trichuris trichiura
Reservoir	Human
Vector	None
Vehicle	Soil ingestion Sexual contact (rare) Fly
Incubation Period	2m - 2y
Diagnostic Tests	Stool microscopy or visualization of adult worms (adults are approximately 3 cm long).
Typical Adult Therapy	Mebendazole 100 mg PO BID X 3d. OR Albendazole 400 mg PO daily X 3 to 7 days OR Ivermectin 200 mg/kg PO daily X 3 days
Typical Pediatric Therapy	Mebendazole 100 mg PO BID X 3d (>age 2). OR Albendazole 400 mg PO X 3 to 7 days OR Ivermectin 200 mg/kg PO daily X 3 days
Clinical Hints	Abdominal pain, bloody diarrhea, rectal prolapse or intestinal obstruction are occasionally encountered; the parasite may survive for as long as five years in the human host.
Synonyms	Trichocephaliasis, Trichuris trichiura, Tricuriasis, Whipworm. ICD9: 127.3 ICD10: B79

Clinical

The vast majority of infections are asymptomatic. [1]
- Symptoms are aggravated by concurrent shigellosis, balantidiasis or amebiasis.
- Heavy infestations are characterized by dysentery or rectal prolapse. [2] [3]
- Infants may develop hypoproteinemia, anemia, mental retardation and digital clubbing. [4-8]
- In some cases, chronic infection may result in edema of the ileocecal valve, suggestive of "malignancy" [9]

This disease is endemic or potentially endemic to all countries.

Trichuriasis in Brazil

Ova of *Trichuris* have been identified in 18th century human remains the area of Rio de Janeiro. [10]

Prevalence surveys:

1.1% of persons in Americaninhas, Minas Gerais (2004) [11]
0.8% of the Parakana indigenous community in southwestern Para State (1992 to 1995) [12]
34.2% of Hupda and 16.3% of other indigenous peoples in Iauarete (2001) [13]
69.9% of persons in Sao Lourenco da Mata, rural Recife (Pernambuco, 1989) [14]
32.9% of homeless persons in Rio de Janeiro (2002 publication) [15]
1.1% of persons in Americaninhas, Minas Gerais (2004) [16]
9.7% of persons in Campos do Jordao (2003 to 2004) [17]
22.5% of persons in Santa Isabel do Rio Negro, Amazon region (2011 publication) [18]
38.6% of school children in Salvador (1997)
12% of children ages 3 to 6 years in day-care centers (Salvador, 2012 publication) [19]
13% of school children in Lages (2004 publication) [20]
36% of school children in Caxias do Sul, RS (2008 publication) [21]
15.6% of primary school children in Aracaju, Sergipe (1999 publication) [22]
78.4% of children in rural Bahia (2006 publication) [23]
4.9% of children presenting to outpatient clinics in Manaus (2009 publication) [24]
18.0% of children ages 1 to 4 1998; 5.0% during 2003 to 2004 (Salvador) [25]
1.0% of Sao Paulo children below age 5 years (1995 to 1996) [26]
0% of children in an urban slum in Sao Paulo (2008 publication) [27]

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12.2% of children in northeastern Brazil. (2010 publication) [28]
0.7% of children ages 0 to 7 years in Uberlandia, Minas Gerais (1994 to 1995) [29]
2.5% of children in Uberlandia, Minas Gerais (2008 publication) [30]
1.1% of children in public daycare centers in Belo Horizonte, Minas Gerais (2008 publication) [31]
11.0% of urban children with diarrhea in Salvador (2000 to 2002) [32]
0.5% of children hospitalized for diarrhea and dehydration (Rio de Janeiro, 2007 publication) [33]
13.1% of HIV-positive patients before HAART vs. 1% after HAART (Ceara, 2008 publication) [34]
13.8% of soil samples in Fernandopolis, Sao Paulo State (*Trichuris* sp., 2007 to 2008) [35]

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Tropical phagedenic ulcer

Agent	BACTERIUM Mixed infection by ? Fusobacterium species and Borrelia
Reservoir	Human
Vector	None
Vehicle	Direct inoculation ? via minor trauma
Incubation Period	Unknown
Diagnostic Tests	Wound smear suggestive of fusobacterial infection.
Typical Adult Therapy	Systemic Penicillin G . Excision/debridement as necessary
Typical Pediatric Therapy	As for adult
Clinical Hints	A deep, painful, foul-smelling ulcer (usually of the leg) with undermined edges; may be complicated by secondary infection.
Synonyms	Acute phagadenic ulcer. ICD9: 682.7 ICD10: A69.8,L97

Clinical

95% of ulcers involve the ankle or lower third of the leg.

- Minor trauma is followed by a tender indurated area which evolves into a round or oval skin ulcer. [1] [2]
- Ulcers favor the lower extremities, and tend to be single, painful and foul-smelling.
- Ulcers spread rapidly, and result in exposure of underlying muscles and tendons.
- Fever and restlessness are common.
- After 4 or more weeks, ulcers may become painless and chronic, and persist for decades.
- Scar carcinomas develop in 2% of cases, and constitute a common form of malignancy in parts of Africa.

This disease is endemic or potentially endemic to 69 countries.

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Tropical pulmonary eosinophilia

Agent	UNKNOWN Possibly related to filarial infection
Reservoir	Unknown
Vector	Unknown
Vehicle	Unknown
Incubation Period	Unknown
Diagnostic Tests	Antifilarial antibodies may be present. Response to therapeutic trial.
Typical Adult Therapy	Diethylcarbamazine 2 mg/kg PO TID X 10d
Typical Pediatric Therapy	As for adult
Clinical Hints	Chronic cough, wheezing, dyspnea, reticular-nodular pulmonary infiltrates and eosinophilia (over 3,000/cu. mm.) acquired in countries known to be endemic for filariasis.
Synonyms	

Clinical

Tropical pulmonary eosinophilia is characterized by recurrent episodes of paroxysmal, dry cough, wheezing, and dyspnea. [1-3]
• Malaise, anorexia, and weight loss are common.
• Symptoms are worse and night.
• Physical examination reveals scattered wheezes and crackles.
• Some patients have fever, hepatomegaly and lymphadenopathy.
• Symptoms fluctuate in severity over many months.

Eosinophilia is present in the majority of patients, often at very high levels (as high as 60,000/cu mm) • however, the level of eosinophilia is not related to the severity of symptoms.
• Chest radiographs reveal scattered reticulonodular opacities [4] which may be mistaken for miliary tuberculosis. [5]
• Serum antibodies to filaria are present.
• A presumptive clinical diagnosis can usually be made through successful response to antifilarial therapy.
• A second course may be necessary in some cases.

This disease is endemic or potentially endemic to 109 countries.

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Trypanosomiasis - African

Agent	PARASITE - Protozoa. Neozoa, Euglenozoa, Kinetoplastidea. Flagellate: Trypanosoma [Trypanozoon] brucei gambiense and T. b. rhodesiense
Reservoir	Human Deer Wild carnivore Cattle
Vector	Fly (Glossina = tsetse fly)
Vehicle	None
Incubation Period	3d - 21d (acute illness)
Diagnostic Tests	Identification of protozoa in CSF, blood, lymph node aspirate. Serology. Nucleic acid amplification.
Typical Adult Therapy	Early: Pentamidine 4 mg/kg IM qod X 10 doses. OR Suramin 1g IV days 1, 3, 7, 14, 21 (after test dose 100 mg) OR Eflornithine (gambiense only) 100 mg q6h IV X 14 d; then 75 mg/kg PO X 21-30 d. Late + CNS disease: Melarsoprol
Typical Pediatric Therapy	Early: Pentamidine 4 mg/kg IM qod X 10 doses. OR Suramin 20 mg/kg IV days 1, 3, 7, 14, 21 (after test dose 20 mg) Late + CNS: Melarsoprol
Clinical Hints	Chancre, myalgia, arthralgia, lymphadenopathy and recurrent fever; later mental changes, sensory disorders and heart failure; disease due to *Trypanosoma brucei rhodesiense* is more rapid and virulent than that due to *T.b. gambiense*.
Synonyms	African sleeping sickness, African trypanosomiasis, Gambian fever, Schlafkrankheit, Trypanosoma brucei, Trypanosoma congolense, Trypanosoma evansi, Trypanosoma lewisi, Trypansomiasis, afrikanische, U.T.I., UTI. ICD9: 086.3,086.4,086.5 ICD10: B56

Clinical

WHO Case definition for surveillance:
• In the early stages, a painful chancre, which originates as a papule and evolves into a nodule may be found at the primary site of tsetse fly bite.
• There may be fever, intense headache, insomnia, painless lymphadenopathy, anemia, local edema and rash. In the later stage, there is cachexia, somnolence and signs of central nervous system involvement.
• The disease may run a protracted course of several years in the case of *Trypanosoma brucei gambiense*. In case of *T. b. rhodesiense*, the disease has a rapid and acute evolution.
• Both diseases are always fatal without treatment.
• The painful chancre is very rare in *T. b. gambiense* infection.
Laboratory criteria for diagnosis
• Presumptive: serological: card agglutination trypanosomiasis test (CATT) for *T. b. gambiense* only or immunofluorescent assay (IFA) for *T. b. rhodesiense* mainly and possibly for *T. b. gambiense*.
• Confirmative: parasitological: detection (microscopy) of trypanosomes in blood, lymph nodes aspirates or CSF.
Case classification
• Suspected: A case that is compatible with the clinical description and/or a history of exposure.
• Probable: A case with a positive serology with or without clinical symptoms in persons without previous history of trypanosomiasis diagnosis or treatment.
• Confirmed: A case with positive parasitology, with or without clinical symptoms.
Notes:
• In the early stage or even early in the late stage of the disease there are often no clinical signs or symptoms which can be associated with the disease.
• Suspicion is then based on local risk of contracting the disease and local disease historical background.
• Confirmed positive healthy carriers are a major public health risk. As a reservoir of parasites, they disseminate the disease, and must be treated as soon as possible.

Acute trypanosomiasis:
The initial sign of African trypanosomiasis in a chancre which develops at the site of inoculation, 1 to 2 weeks following the bite of a tsetse fly.
• The chancre may reach a diameter of several centimeters, and be associated with regional adenopathy, but resolves over several weeks.
• In most cases, the chancre is noted by neither the patient nor the clinician.
• Fever appears weeks to months following inoculation, and is characteristically intermittent.
• Lymphadenopathy is a fairly constant feature of west African trypanosomiasis.

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• The nodes are discrete, movable, rubbery, and nontender.
• Supraclavicular and cervical nodes are often visibly discernible, and enlargement of the nodes of the posterior cervical triangle ("Winterbottom's sign") is common in the west African form.
• Additional findings at this point may include hepatosplenomegaly; edema of the face, hands and feet; pruritis; an irregular circinate, 5 to 10 cm rash on the trunk, shoulders, buttocks and thighs [1] ; headache, asthenia, weight loss, arthralgias, and tachycardia.
• In some cases, trypanosomiasis has been associated with cryoglobulinemic membranoproliferative glomerulonephritis. [2]

***Trypanosoma brucei gambiense* infection:**
In the West African form, the meningoencephalitic stage may develop months or even years after the initial infection. [3]
• Findings include irritability, personality changes, indifference, apathy, daytime somnolence (often with insomnia at night), slurred speech, choreiform movements of the trunk, neck, and extremities, tremors of the tongue and fingers, ataxia, and muscular fasciculations. [4]
• CSF cell counts above 5 per cu mm are considered indicative of brain involvement. [5] [6]
• The final phase of the CNS disease is progression to coma and death [7] ; however, survival without therapy is reported. [8]

***Trypanosoma brucei rhodesiense* infection:**
The East African form tends to follow a more acute course, with an incubation of a few weeks to several weeks.
• Intermittent fever, headache, myalgia, and rash develop early; while lymphadenitis is not a prominent feature.
• Persistent tachycardia is common, and some patients die of arrhythmias, congestive heart failure or pericarditis before the onset of neurological disease.
• If untreated, the East African form is fatal within weeks to months.

This disease is endemic or potentially endemic to 36 countries. Although Trypanosomiasis - African is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Trypanosomiasis - African in Brazil

1997 - The country's first case (imported from Angola) was reported.
2001 - A Brazilian soldier acquired trypanosomiasis (nonfatal) in Angola. [9]
2010 - A Brazilian tourist acquired trypanosomiasis in Zimbabwe. [10]

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Trypanosomiasis - American

Agent	PARASITE - Protozoa. Neozoa, Euglenozoa, Kinetoplastidea. Flagellate: Trypanosoma cruzi
Reservoir	Human Dog Cat Pig Guinea pig Armadillo Rat Fox Opossum Raccoon Bat Mouse Monkey Rabbit
Vector	Bug (Triatome or 'kissing' bug = Panstrongylus, Rhodnius and Triatoma spp.)
Vehicle	Blood, Food (fruit contaminated with insect secretions)
Incubation Period	5d - 14d (acute illness)
Diagnostic Tests	Identification of protozoa in blood or tissue. Serology. Xenodiagnosis. PCR [more sensitive than serology)
Typical Adult Therapy	Nifurtimox 2 mg/kg PO QID X 3m. OR Benznidazole 3 to 5 mg/kg/d X 30 to 120d
Typical Pediatric Therapy	Nifurtimox: Age 1 to 10 years: 5 mg/kg PO QID X 3m Age 11 to 16 years: 3.5 mg/kg PO QID X 3m (age 11 to 16y) OR Benznidazole 3.75 mg/kg PO BID X 2m; or
Clinical Hints	Unilateral periorbital swelling (Romana's sign) with lymphadenopathy, hepatosplenomegaly and encephalitis; later cardiomyopathy, megaesophagus and megacolon; 20% of patients progress to chronic stage; overall case-fatality rate = 10%.
Synonyms	American trypanosomiasis, Chagas' disease, Chagas-Cruz disease, Chagas-Krankheit, Trypanosoma cruzi, Trypanosoma rangeli, Trypansomiasis, amerikanische. ICD9: 086.0,086.1,086.2 ICD10: B57

Clinical

WHO Case definition for surveillance:
Acute stage • Clinical description
• The main clinical signs are mainly fever, malaise, hepatosplenomegaly and lymphadenopathy in the acute phase.
• Many patients present without clinical signs.
• An inflammatory response at the site of infection (chagoma) may last up to 8 weeks.
Laboratory criteria for diagnosis
• Positive parasitology (direct, xenodiagnosis, blood culture) and/or
• Positive serology for *Trypanosoma cruzi* antibodies (IgM) (indirect hemagglutination test (IHA), indirect immunofluorescent antibody test (IFAT), direct agglutination test (DA) and ELISA)
Case classification
• Suspected: Not applicable.
• Probable: (Endemic areas) a case with unexplained fever, hepatosplenomegaly and a chagoma (inflammation at site of infection).
• Confirmed: A clinically compatible case that is laboratory-confirmed.
• Congenital: A newborn with positive parasitology (direct, xenodiagnosis, culture).
• Indeterminate: Positive serology for *T. cruzi* antibodies alone, no other clinical findings related to the disease (e.g. in blood donors).
• Chronic: Positive serology or parasitology with chronic cardiac lesions and/or enlargement of the digestive viscera and/or peripheral neuropathies.

The clinical features of acute and chronic infection are distinct.

Acute Chagas' disease:

• Acute infection can occur at any age, but is usually an illness of children endemic areas. [1]
• The first signs of illness occur one week or more following inoculation, and consist of a "chagoma," indurated area of erythema and swelling with local lymph node involvement.
• The Romana sign consists of painless edema of the eye lids and periocular tissues which follows conjunctival inoculation.
• Later, the patient develops fever, malaise, anorexia, and edema of the face and legs.
• Generalized lymphadenopathy and mild hepatosplenomegaly also may appear.

Overt central nervous system signs are uncommon; however, the occurrence of meningoencephalitis at this stage is associated with a very poor prognosis. [2] [3]
• Severe myocarditis with arrhythmias and hear failure may also develops during the acute disease, and accounts for most deaths at this stage.
• Symptoms resolve gradually over a period of weeks to months in untreated patients; however, periocular swelling,

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lymphadenopathy and splenomegaly may persist.
• Reactivation of neurological disease has been reported among infected HIV-positive patients. [4]

Chronic Chagas' disease:
Chronic Chagas' disease presents years or decades after the initial infection.
• Ten to thirty percent of acute infections will progress to chronic disease.
• Night blindness is a common feature at this stage. [5]
• The heart is most often involved, with signs of arrhythmia, congestive heart failure and thromboembolism. [6]
• These arrhythmias may present as episodes of vertigo, syncope or seizures.
• Cardiomyopathy primarily affects the right ventricle.
• Once congestive heart failure develops, death occurs in a matter of months.
• Cerebral disease may manifest as headache, seizures, focal neurological signs or evidence of ischemia and infarct. [7-9]

Gastrointestinal Chagas' disease may involve the salivary glands, esophagus, lower esophageal sphincter, stomach, small intestine, colon, gallbladder or biliary tree. [10]
The signs and symptoms of megaesophagus similar to those of idiopathic achalasia, consisting of dysphagia, odynophagia, chest pain, cough, and regurgitation.
• Aspiration may occur during sleep, and recurrent pneumonia is common.
• An increased incidence of cancer of the esophagus is reported in patients with chagasic esophageal disease.
• Megacolon is characterized by chronic constipation and abdominal pain, occasionally complicated by volvulus or perforation. [11]

A review of Chagas disease among HIV-positive patients • see reference [12]

This disease is endemic or potentially endemic to 22 countries.

Trypanosomiasis - American in Brazil

Time and Place:
Trypanosoma cruzi has been identified in pre-Colombian human remains in Peruacu Valley, Minas Gerais. [13]
- Chagas disease was first described in 1909, during the construction of a railroad system in Brazil. [14]
- During the 1970's, the disease was endemic to 44.5% of the country, and most common in Goias, Minas Gerais and Rio Grande do Sol - 49 million persons lived in at-risk areas.
- The disease was first reported in the Western Brazilian Amazon in 1979. [15]
- As of 1994, the endemic states include Alagoas, Bahia, Goias, Maranhao, Mato Grosso, Minas Gerais, Paraiba, Piaui, Rio Grande do Norte and Rio Grande do Sul.
- As of 2000, only Bahia and Sao Paulo remain endemic.

The first autochthonous human cases of Chagas disease in Amazonian Brazil were reported in Belem (Para State) in 1969.
- A total of 205 cases were recorded in the area to 2000.
- 233 cases of acute Chagas disease were registered in Para, Amapa and Maranhao during 1988 to 2005 [16]
- Although Sao Paulo State had been free of the disease since the 1970's, a single autochthonous case was reported in 1995.

During 1980 to 1985, 6,340,000 persons were infested.
- 40,002 deaths from Chagas disease were registered in Sao Paulo during 1985 to 2006.

The nationwide incidence for congenital infection is 195.3 per 100,000 live births.

2,493 municipalities were infested by *Triatoma infestans* in 1970; 711 in 1983; 83 in 1993.

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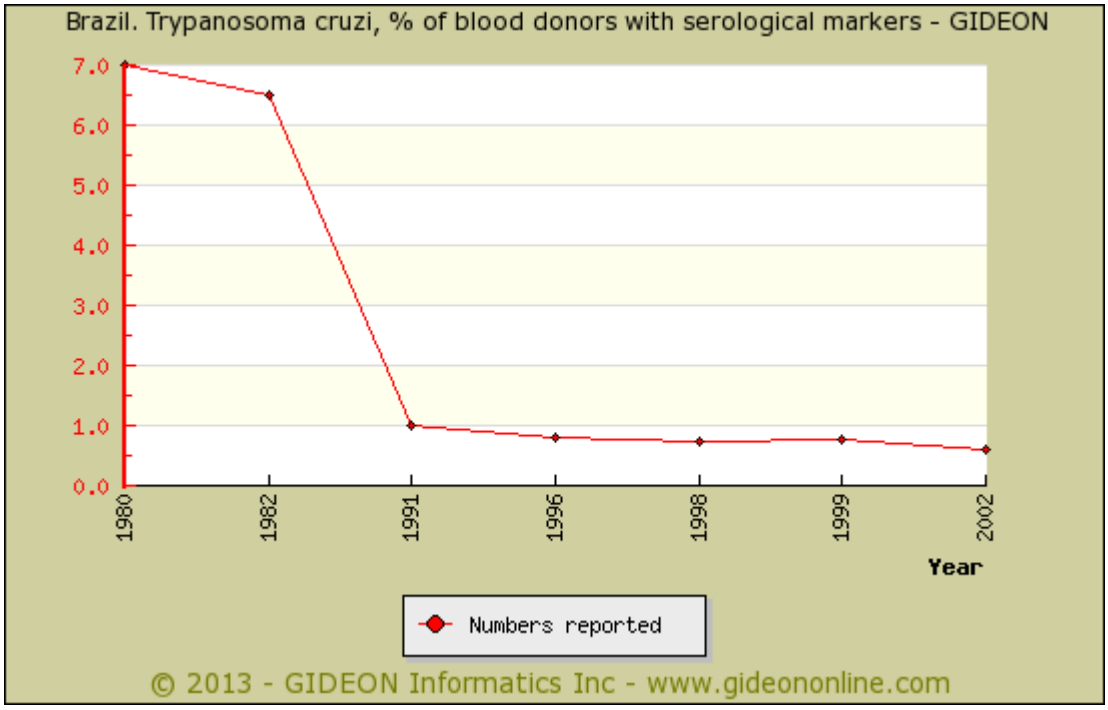

Graph: Brazil. *Trypanosoma cruzi*, % of blood donors with serological markers

Notes:
1. 1.9% of blood donors in Igatu were found to be seropositive (1996 to 1997).
2. 0.25% of first-time blood donors in Sao Paulo were found to be seropositive (1996 to 2001).
3. 2.3% of blood donors in Goiana were found to be seropositive (1988 to 1989). [17]
4. 0.47% of blood donors in Pelotas City were found to be seropositive (2004 to 2005) [18]
5. 0.114% of blood donors were found to have confirmed seropositivity (three blood centers, 2007 to 2008) [19]

Prevalence surveys:

40.7% of autopsies performed in Uberaba, Minas Gerais (1970 to 2004) [20]

39.6% of free-ranging tamarin monkeys (*Sanguinus bicolor*) from Amazon Rainforest are infected by *Trypanosoma rangeli*, and 0% by *T. cruzi* (2008 publication) [21]

93% of *Didelphis* sp. In Santa Catarina, during the course of a local outbreak among humans (2008 publication) [22]

3.2% of *Panstrongylus geniculatus*, 0.6% of *Rhodnius neglectus* and 0.1% of *Triatoma sordida* in Mato Grosso do Sul (2008 publication) [23]

8.3% of captured bugs in central-western municipalities of Minas Gerais (2003 to 2007) [24]

2.7% triatomes in Triangulo Mineiro and Alto Parana?ba (southeastern Brazil, 2002 to December 2004) [25]

10.9% of *Triatoma braziliensis* from rock piles in Ceara (month of July, 2010 publication) [26]

18% of *Triatoma sordida* in the rural Quilombola community of Furnas do Dionizio, State of Mato Grosso do Sul (2011 publication) [27]

69% of *Triatoma pseudomaculata* and 25.0% of *Rhodnius nasutus* in periurban Ceara State (2011 publication) [28]

50% of triatomes in Monte Negro, Rondonia (2012 publication) [29]

56.5% of *Panstrongylus megistus* in Agua Boa village, (Itabaianinha, Sergipe, 2009 to 2010) [30]

Seroprevalence surveys:

0.1% of children ages <=5 years (all rural areas except for Rio de Janeiro State, 2011 publication) [31]

4.2% of children in endemic states in 1980 and 0.15% in 1994.

0.019% of school-age children in Espirito Santo State (a non-endemic area) (1999 to 2000).

20.5% of persons in rural southwestern Bahia (1986 publication) [32]

6.5% of persons in the west mesoregion and 3.3% in Caico (Rio Grande do Norte State, 2012 publication) [33]

2.5% of sickle cell anemia patients in Bahia (1995 to 2009) [34]

5% of children ages 0 to 7 years in 1980; 0.28% in 1999.

0.9% of pregnant women in Parana (1996 to 1998)

1.2% of periurban residents and 21.9% of domestic dogs in Ceara State (2012 publication) [35]

3% of inhabitants of 15 villages in Paco do Lumiar County (2007 publication) [36]

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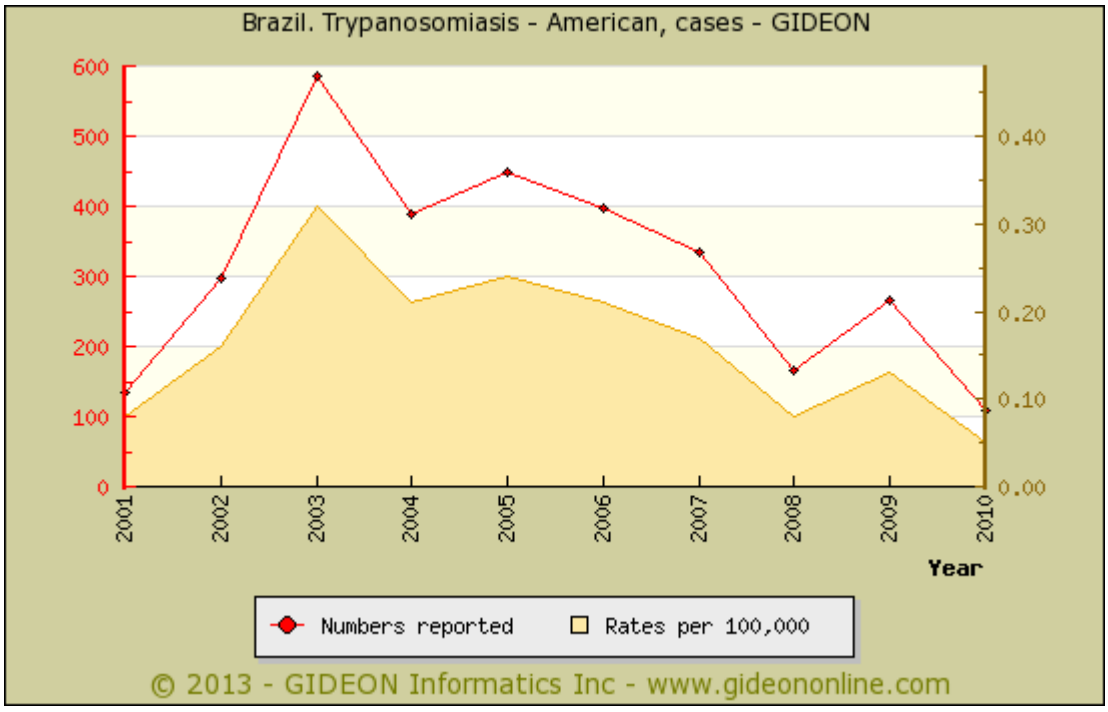
1.2% of persons in Monte Negro, Rondonia (2012 publication) [37]
1.2% of persons in Amazonas State (2012 publication) [38]
25.5% of persons in Agua Comprida, Minas Gerais (2010 publication) [39]
14% of various bat species (Molossidae, Noctilionidae, Phyllostomidae and Vespertilionidae) from various regions (2008 publication) [40]
45% of baboons with lymphocytic myocarditis (2009 publication) [41]
53.5% of free-ranging coatis (*Nasua nasua*) from Pantanal region, Mato Grosso do Sul (2010 publication) [42]
0.4% of dogs in rural Botucatu, Sao Paulo State (2011 publication) [43]

It is estimated that each year 75,000 Brazilians develop cardiac arrhythmias, 45,000 megaesophagus and 30,000 megacolon.



Graph: Brazil. Trypanosomiasis - American, cases

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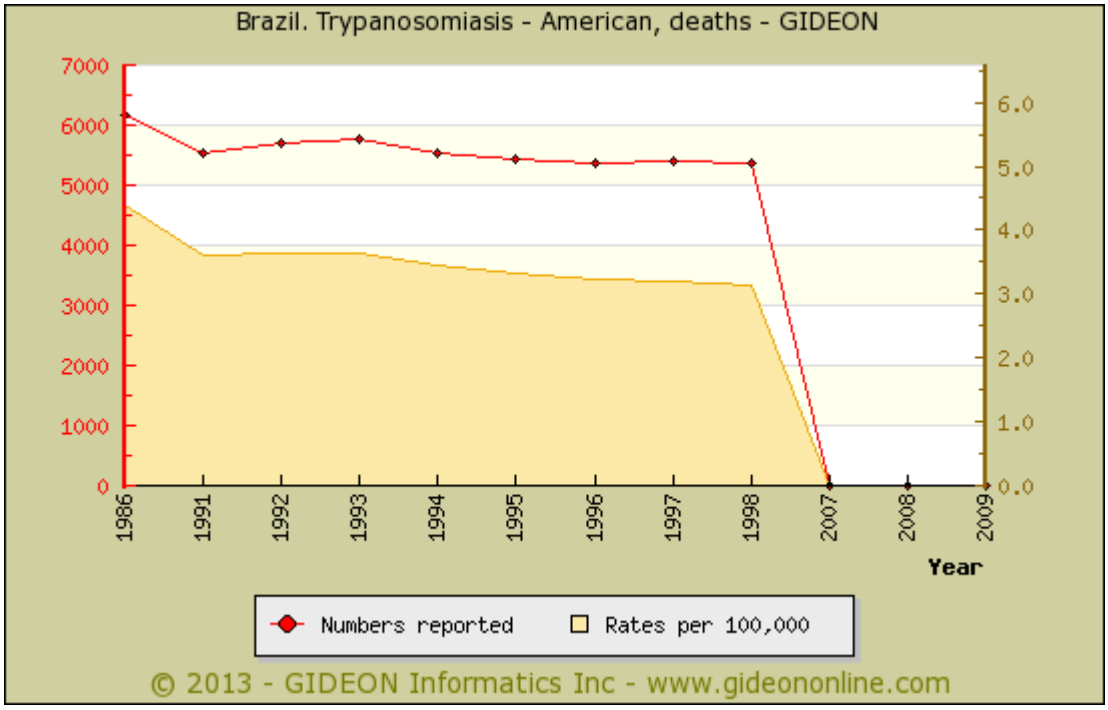

Graph: Brazil. Trypanosomiasis - American, deaths

Notes:
1. Chagas' disease is the most common cause for death from parasitic disease in Brazil.
2. Chagas' disease accounted for 24.1% of sudden and unexpected death in Minas Gerais (Mineiro triangle) in 1980; 6.8% in 1990.
3. 53,930 deaths were attributed to Chagas' disease during 1999 to 2007 - 3.37 per 100,000 per year. [44]

Reservoirs:
- Local reservoir species include 9 marsupials, 4 edentates, 10 rodents; 14 bats; 3 carnivores), 3 primates and one artiodactyle (domestic pig).
- The principal reservoir is *Akodon arviculoides* (18.4% infection rate).
- *Didephis albiventris* and *Trichomys apereoides* are important reservoirs in rural Piaui. [45]
- The coati (*Nasua nasua*) serves as a reservoir host for *Trypanosoma cruzi* in the Pantanal region [46] [47]
- An infected anteater (*Tamandua tetradactyla*) was identified in the Amazon basin. [48]

Vectors:
- At least 10 of the 16 known sylvatic triatome species are active in transmission in the Amazon basin: *Eratyrus mucronatus*, *Microtriatoma trinidadensis*, *Panstrongylus geniculatus*, *P. lignarius*, *P. rufotuberculatus*, *Rhodnius brethesi*, *R. neglectus*, *R. paraensis*, *R. pictipes*, *Cavernicolai pilosa*, *R. prolixus* and *R. robustus*.
- Principal vectors include *Panstrongylus megistus* and *Triatoma infestans*. *Panstrongylus geniculatus* is also involved in zoonotic transmission.
- *Rhodnius pictipes* and *Rhodnius robustus* have been identified as vectors in the Western Brazilian Amazon. [49]
- *Triatoma brasiliensis* is the principal vector in the northeast, where *Tr. pseudomaculata* is also active.
- The principal vectors in Sao Paulo State are *Panstrongylus megistus*, *Triatoma sordida* and *Triatoma tibiamaculata* (1990 to 1999).
- 0.19% of domiciliary units in Berilo were found to be infested with *T. pseudomaculata*; however, none were infected with *T. cruzi* (2007 publication) [50]
- 3.2% of *Panstrongylus geniculatus*, 0.6% of *Rhodnius neglectus* and 0.1% of *Triatoma sordida* in Mato Grosso do Sul are infected (2008 publication) [51]
- 69% of *Triatoma pseudomaculata* and 25.0% of *Rhodnius nasutus* in periurban Ceara State are infected (2011 publication) [52]
- *Panstrongylus megistus* is the predominant vector in Minas Gerais - 8.3% of captured bugs in central-western municipalities were positive (2003 to 2007) [53]
- The principal vector in Uberaba, Minas Gerais State is *Triatoma sordida*. [54] *Panstrongylus megistus* also serves as a vector in Minas Gerais. [55]
- *Rhodnius pictipes* has been implicated in transmission in Amapa State. [56]
- *Triatoma costalimai* is implicated in transmission in Goias State. [57]

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- Infected *Triatoma vitticeps* have been identified in Espiritu Santo State. [58] ; *Triatoma rubrovaria* in Rio Grande do Sul. [59]
- Infected *Triatoma vitticeps* have been identified in Sao Fidelis, Rio de Janeiro (2008 publication) [60]
- Infected *Triatoma sherlocki* have been be identified in Bahia (2009 publication) [61]
- *Rhodnius nasutus* [62] , *Triatoma brasiliensis* and *Triatoma pseudomaculata* are active in Ceara State. [63]
- Infected triatomes (*Triatoma sordida*, *Panstrongylus diasi*, *Panstrongylus megistus*, *Panstrongylus geniculatus* and *Rhodnius neglectus*) have been identified in Triangulo Mineiro and Alto Parana?ba (southeastern Brazil, 2002 to December 2004) [64]

Other infecting species:

- Human infection by *Trypanosoma rangeli* has also been described. [65]
- *Trypanosoma rangeli* has been identified in triatomes (*Rhodnius nasutus*) in Ceara State. [66]

Notable outbreaks:

1968 (publication year) - An outbreak of food-borne trypanosomiasis was reported. [67]

1979 (publication year) - An outbreak was reported in the Sao Francisco Valley region of Bahia. [68]

1986 (publication year) - An outbreak (20 cases) was reported in Riacho de Santana, southwestern Bahia. [69]

1986 - An outbreak (26 cases) in Paraiba state was ascribed to food-borne infection. [70]

1996 - An outbreak (17 cases) was reported in Amapa. [71]

2000 - An outbreak (11 cases) was reported in Belem - oral acquisition was suspected. [72]

2001 (publication year) - An outbreak (13 cases) was reported among members of a family in Abaetetuba, Brazilian Amazon. [73]

2005 - An outbreak (25 cases, 6 fatal) in Santa Catarina was caused by ingestion of sugar cane juice ('garapa'), presumably contaminated with vector insects. [74-77]

2006 - An outbreak (20 cases) in Para was caused by ingestion of Bacaba wine, contaminated with insect vectors. [78] A separate publication described 11 cases in the area associated with Acai (*Euterpe oleracea*) palm fruit (? same outbreak) [79] [80]

2007 - Outbreaks (116 cases in 15 outbreaks) of ingestion-related trypanosomiasis were reported in Para, Amazonas and Amapa. [81-85]

2008 (publication year) - An outbreak (7 cases) in Bahia was ascribed to drinking water contaminated by Triatome feces. [86] [87]

2010 - An outbreak (12 cases) in Amazonas was related to consumption of acai (a fruit). [88] [89]

2012 - An outbreak (24 cases) of trypanosomiasis in Para State was related to consumption of acai. [90-92]

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Tuberculosis

Agent	BACTERIUM. Actinomycetes, Mycobacterium tuberculosis An aerobic acid-fast bacillus
Reservoir	Human Cattle
Vector	None
Vehicle	Air Dairy products
Incubation Period	4w - 12w (primary infection)
Diagnostic Tests	Microscopy. Culture. Nucleic acid amplification. Inform laboratory when this diagnosis is suspected.
Typical Adult Therapy	Respiratory isolation. Typical pulmonary infection is treated with 6 months of Isoniazid, Rifampin & Pyrazinamide
Typical Pediatric Therapy	As for adult
Vaccine	BCG
Clinical Hints	Cough, "night sweats" and weight loss; often presents as prolonged fever (FUO) or infection of bone, meninges, kidneys or other organs; most infections represent reactivation of old foci in lungs, brain, bone, kidneys etc.
Synonyms	Consumption, Mycobacterium africanum, Mycobacterium bovis, Mycobacterium caprae, Mycobacterium orygis, Mycobacterium tuberculosis, Oryx bacillus, Phthisis, TB, TB meningitis, Tuberculose, Tuberculose miliar, Tuberculosi, Tuberculous meningitis, Tuberkulose, White plague. ICD9: 010,012,013,014,015,016,017,018 ICD10: A15,A16,A17,A18,A19

Clinical

WHO Case definition for surveillance:
Pulmonary tuberculosis, sputum smear positive (PTB+)
• Tuberculosis in a patient with at least two initial sputum smear examinations (direct smear microscopy) positive for Acid-Fast Bacilli (AFB), or
• Tuberculosis in a patient with one sputum examination positive for acid fast bacilli and radiographic abnormalities consistent with active pulmonary tuberculosis as determined by the treating medical officer, or
• Tuberculosis in a patient with one sputum specimen positive for acid-fast bacilli and at least one sputum that is culture positive for acid-fast bacilli.
Pulmonary tuberculosis, sputum smear negative (PTB-)
Tuberculosis in a patient with symptoms suggestive of tuberculosis and having one of the following:
• Three sputum specimens negative for acid-fast bacilli
• Radiographic abnormalities consistent with pulmonary tuberculosis and a lack of clinical response to one week of a broad-spectrum antibiotic
• Decision by a physician to treat with a full curative course of antituberculous chemotherapy
Pulmonary tuberculosis, sputum smear negative, culture positive
• Tuberculosis in a patient with symptoms suggestive of tuberculosis and having sputum smear negative for acid-fast bacilli and at least one sputum that is culture positive for *M. tuberculosis* complex
Extra-pulmonary tuberculosis
• Tuberculosis of organs other than lungs: pleura, lymph nodes, abdomen, genito-urinary tract, skin, joints and bones, tuberculous meningitis, etc.
• Diagnosis should be based on one culture positive specimen from an extra-pulmonary site, or histological or strong clinical evidence consistent with active extra-pulmonary tuberculosis, followed by a decision by a medical officer to treat with a full course of anti-tuberculous therapy
• Any patient diagnosed with both pulmonary and extra-pulmonary tuberculosis should be classified as a case of pulmonary tuberculosis

The clinical features of tuberculosis are protean, and largely determined by the site of infection and clinical substrate.
• Most infections represent reactivation of a dormant focus in a lung, with resultant chronic fever, weight loss, nocturnal diaphoresis, productive cough and typical roentgenographic findings. [1]
• Reactivation of an extrapulmonary focus (kidney, bone, central nervous system [2] [3], skin [4], gastrointestinal [5-10] and hepatobiliary system [11], eyes [12] [13], skeletal muscle [14-18], reproductive tract [19], breast [20] [21], etc) will result in signs referable to the infected organ.
• The extent and severity of disease are influenced by patient age, nutrition, immune function [22] [23], and many other

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factors which are beyond the scope of this module.
• Nocardiosis may mimic tuberculosis, particularly in the setting of HIV infection. [24]
• The appearance of a miliary infiltrates in tropical pulmonary eosinophilia [25] or *Chlamydophila pneumoniae* infection may suggest a diagnosis of tuberculosis. [26]
• Spinal histoplasmosis may mimic tuberculosis spondylodiscitis [27] ; and gastrointestinal histoplasmosis may mimic abdominal tuberculosis. [28]
• The clinical features of melioidosis are similar to those of tuberculosis: prolonged fever, weight loss, latency with reactivation, upper-lobe infiltrates, etc. [29-31]
• Tularemia [32] and leprosy may manifest as lymphadenopathy mimicking tuberculosis [33]

This disease is endemic or potentially endemic to all countries.

Tuberculosis in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Highest rates for pulmonary tuberculosis are reported from the North, followed by the Northeast.
- Highest rates for all forms of tuberculosis are reported from Rio de Janeiro State.

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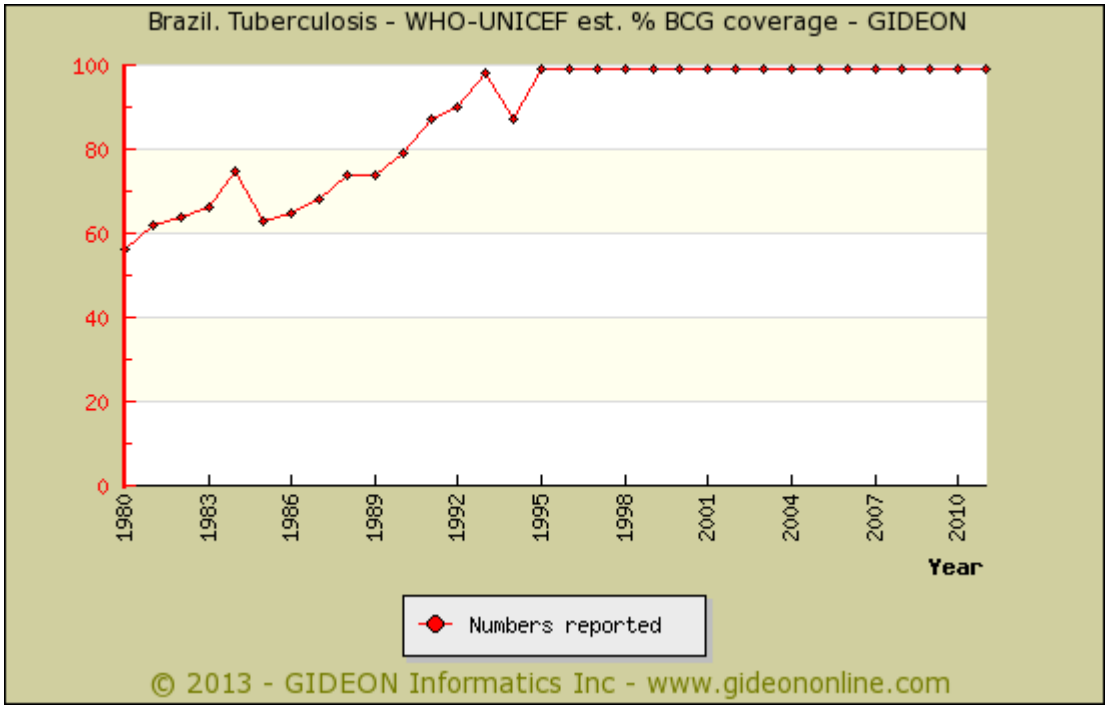
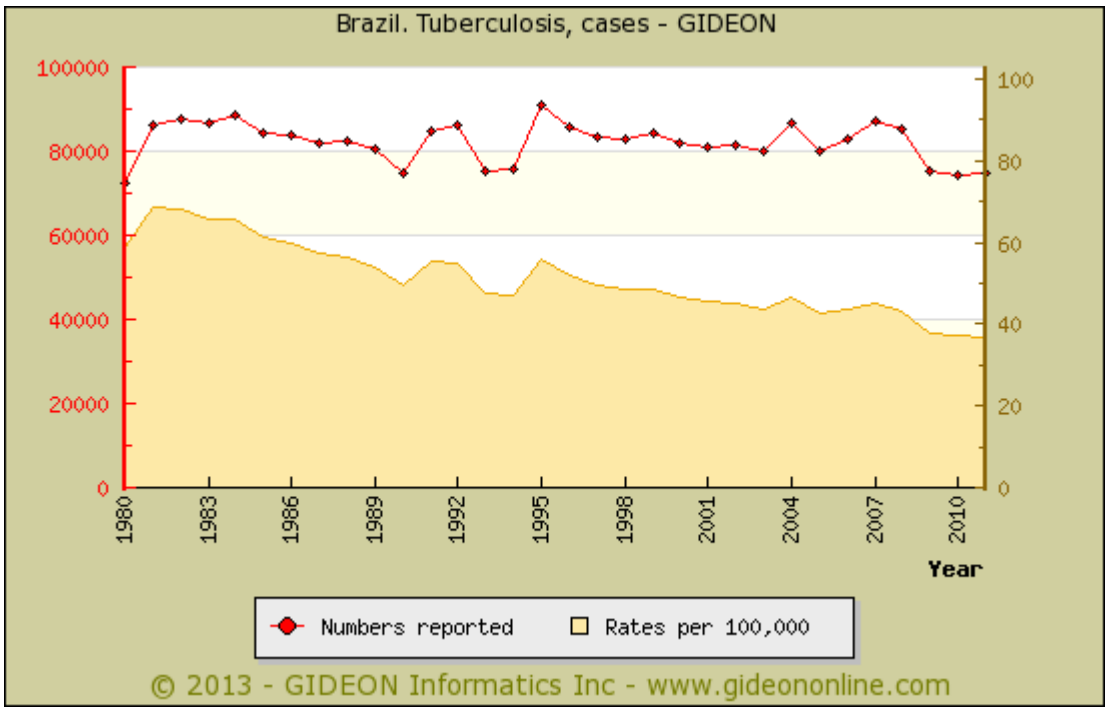


Graph: Brazil. Tuberculosis - WHO-UNICEF est. % BCG coverage

Notes:
1. 4,811,053 doses of BCG were administered in 1995, 3,896,441 in 1996 and 4,744,850 in 1997.



Graph: Brazil. Tuberculosis, cases

Notes:
1. 1,691,128 cases were reported during 1980 to 2000 - including 339112 in Sao Paulo and 221417 in Rio de Janeiro
2. 1997 - The true number was estimated at 122,000 (75 per 100,000 - 112.1 per 100,000 in Rio de Janeiro)

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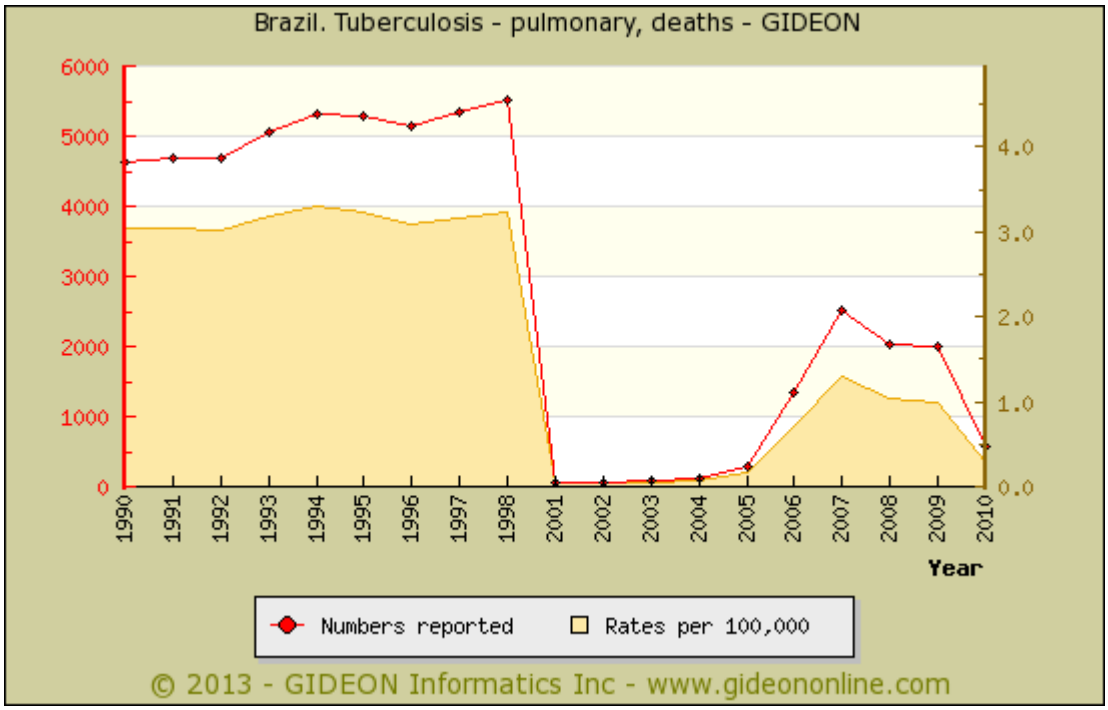
Disease rates per 100,000 in the Amazon region: 51.3 in 2001; 50.5 in 2002; 49.6 in 2003; 49.5 in 2004; 47.4 in 2005; 45.1 in 2006. [34]

Prevalence surveys:

2.0% of solid organ transplant recipients (2001 to 2006) [35]
41.7% of infectious granulomatous laryngitis cases (2007 publication) [36]
15% of water buffaloes (*Bubalus bubalis*) in Rio de Janeiro (2012 publication) [37]



Graph: Brazil. Tuberculosis - pulmonary, deaths

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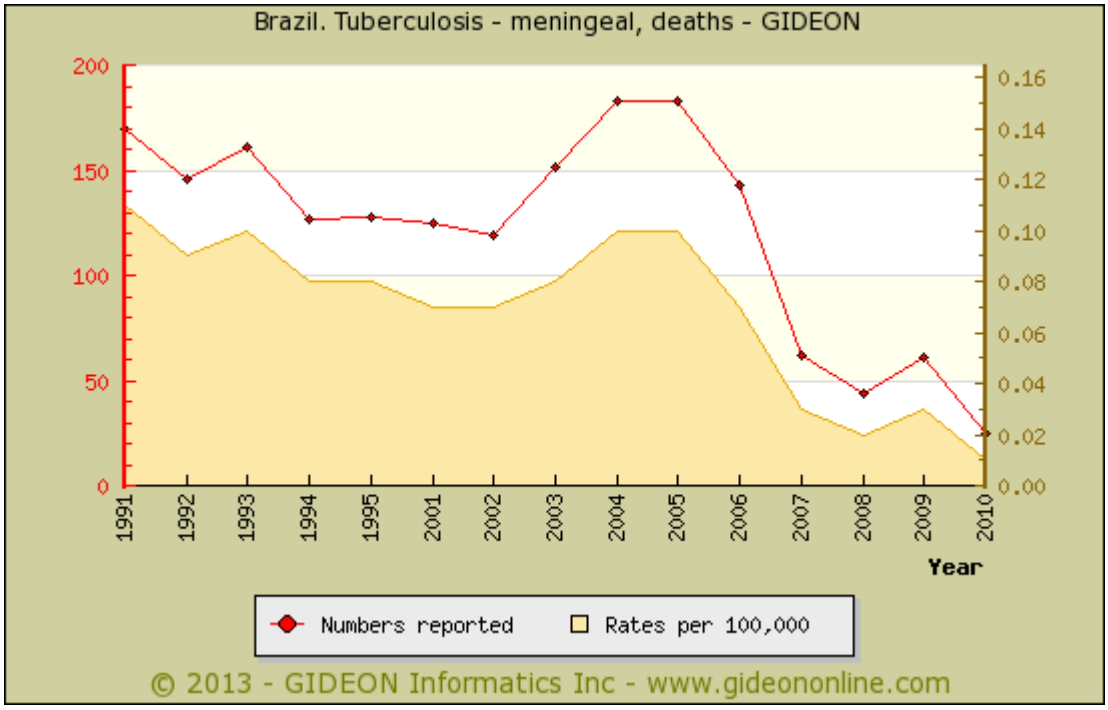
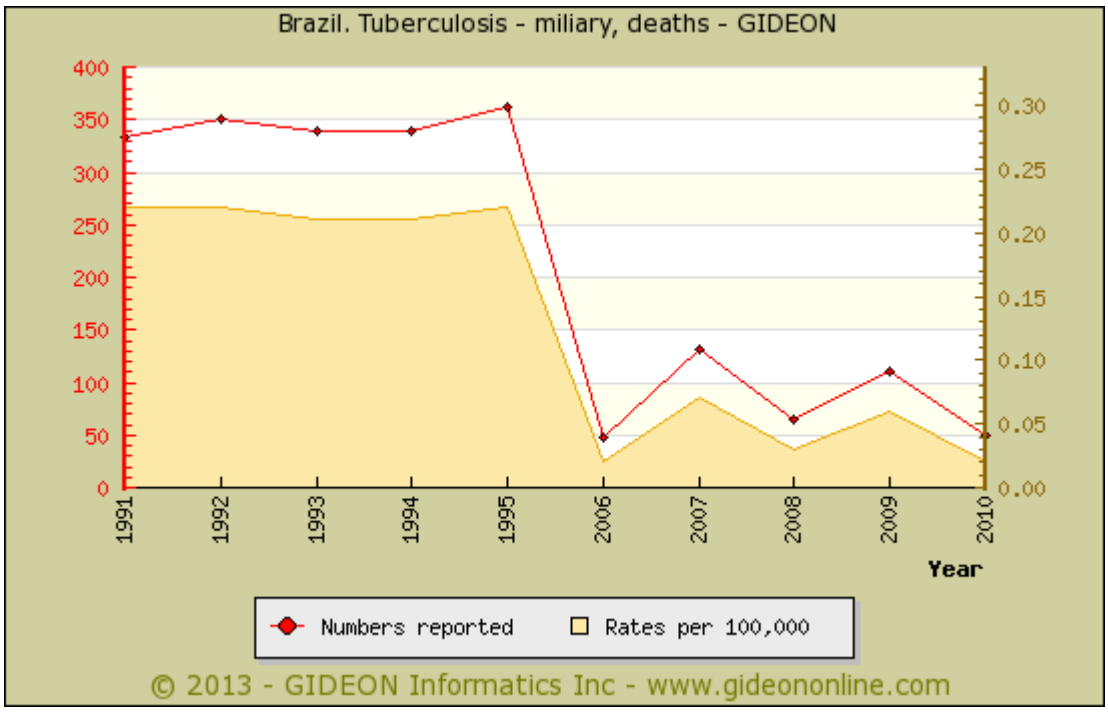


Graph: Brazil. Tuberculosis - meningeal, deaths



Graph: Brazil. Tuberculosis - miliary, deaths

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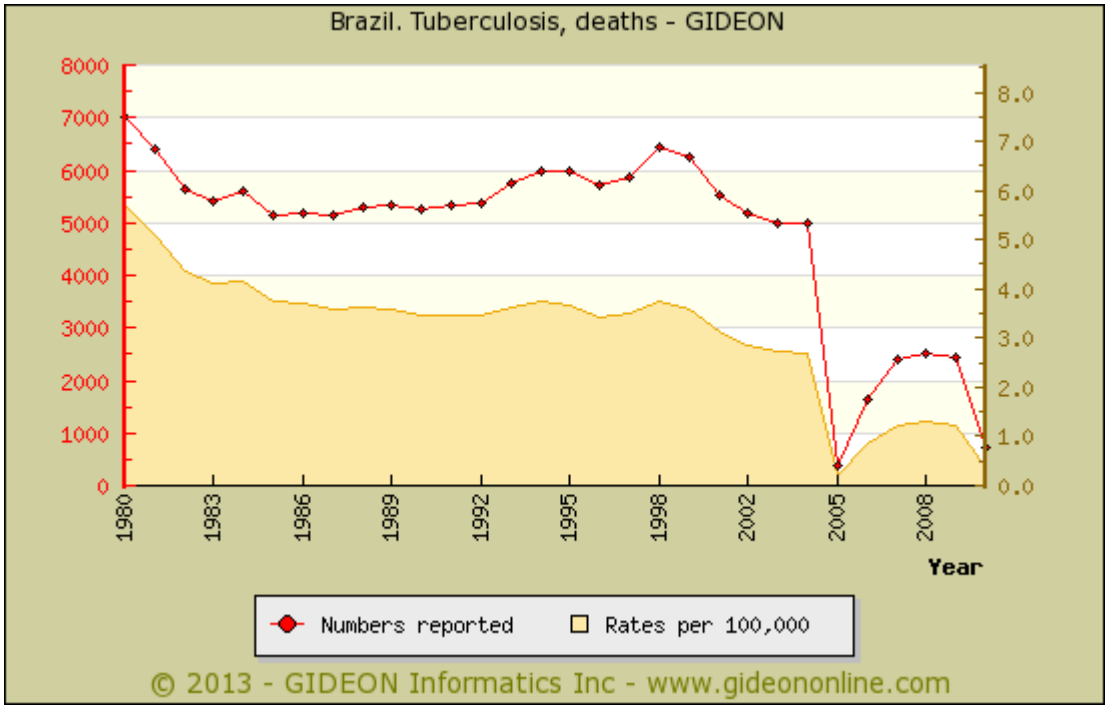
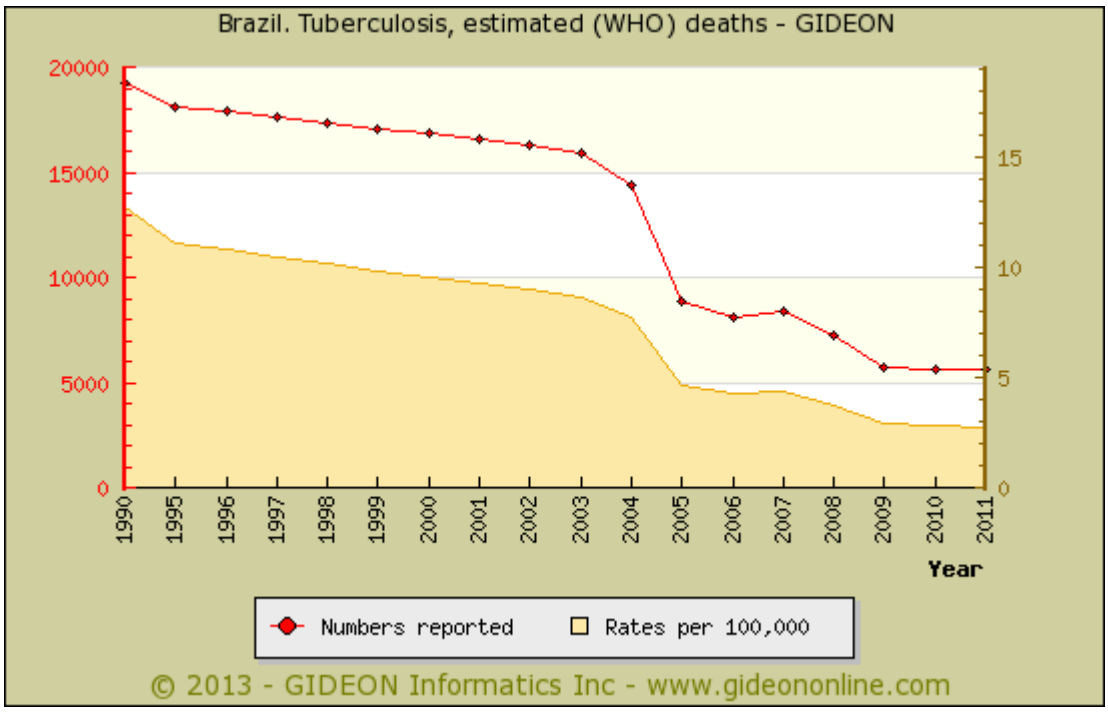


Graph: Brazil. Tuberculosis, deaths



Graph: Brazil. Tuberculosis, estimated (WHO) deaths

The rate of tuberculosis among prison inmates in Campinas, Sao Paulo was 1,397.62 per 100,000 in 1994; 559.04 per 100,000 in 1999. [38]

Tuberculosis and HIV infection:

- - Tuberculosis was the cause of death in 28% of autopsied AIDS patients (Amazonas, 1996 to 2003) [39]
- 17% of AIDS patients have tuberculosis (30.6% in Ceara State, 1986 to 1992). [40]
- Five percent of tuberculosis patients were HIV-positive in 1997; 35.6% in 1998.

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- 27.6% of patients with tuberculosis in Goias State were found to be HIV-positive - 67% of tuberculosis patients who were coinfected by HCV (2011 publication) [41]
- 49.9% of prison inmates with tuberculosis in Campina, Sao Paulo are HIV-positive (1993 to 2000).
- The rate of tuberculosis among Surui Indians in Rondonia is 2,518.9 per 100,000 per year (1991 to 2002). [42] [43]

A national survey (1995 to 1996) demonstrated INH resistance in 6.3% and multidrug resistance in 1.3%.

Notable outbreaks:

2002 - An outbreak (5 cases) of tuberculosis was reported at a psychiatric hospital in Goias. [44]
2004 (publication year) - An outbreak was reported in an inpatient institution in Sao Paulo. [45]
2012 (publication year) - An outbreak of *Mycobacterium bovis* infection was reported among coatis (*Nasua nasua*. [46]

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Tungiasis

Agent	PARASITE - Insecta Siphonaptera (Flea), Tungidae: Tunga penetrans and T. trimamillata ("sand fleas")
Reservoir	Pig Dog ? Various other mammals
Vector	None
Vehicle	Contact
Incubation Period	8d - 12d
Diagnostic Tests	Identification of parasite.
Typical Adult Therapy	Extraction of parasite Ivermectin has been advocated in some publications.
Typical Pediatric Therapy	As for adult
Clinical Hints	Painful papule or nodule, usually on the feet - may be multiple; begins 1 to 2 weeks after walking on dry soil; secondary infections and tetanus are described.
Synonyms	Bicho de pe, Chica, Chigger, Chigoe flea, Jigger, Nigua, Puce-chique, Tu, Tunga penetrans, Tunga trimamillata, Tungosis. ICD9: 134.1 ICD10: B88.1

Clinical

Virtually all infestations are limited to the foot, notably the interdigital and periungual regions. [1] [2]
• Ectopic infections are occasionally noted on the hands, elbows, thighs or gluteal region • and even the eyelids. [3]
• Irritation begins 8 to 12 days following infection, and is manifested as a small 'pit' which evolves into a circular ulcer associated with pain, edema, erythema and pruritis.
• On dermoscopy, circumferential rings may be evident surrounding a central black lesion • the 'radial crown' sign. [4]
• Secondary bacterial infection, thrombophlebitis or even tetanus may follow.
• Most infestations are characterized by 2 to 3 fleas, although hundreds may occasionally be present. [5] [6]
• Severe disease may be characterized by deep ulcerations, necrosis leading to denudation of underlying bone, and auto-amputation of digits.
• Ectopic infection (hands, elbows, knees, neck [7] , anus and genitals) is encountered, often in small children.
• Studies in an endemic region of Brazil revealed 17 lesions (maximum 98) per patient, and almost all had nail deformation and edema.
• Nail loss (46%), pain and fissures (70%), digit deformation (25%), abscesses (42%), and walking difficulty (59%) were common. (Brazil, 2007 publication) [8]

A series of 11 cases of tetanus related to tungiasis (25% of all tetanus cases) was reported by a single hospital in Brazzaville over an 11-month period (1989 publication). [9]
• Tungiasis is implicated in the etiology of 10% of tetanus cases in Sao Paulo, Brazil (2001 publication). [10]

This disease is endemic or potentially endemic to 89 countries.

Tungiasis in Brazil

There is evidence that tungiasis existed in pre-Hispanic America, for at least 14 centuries. [11]

Prevalence surveys:
34% of persons in the Fortaleza 'favela' , including 65.4% of male children ages 5 to 9 years. Many patients suffer from >50 lesions (2005 publication) [12]
3.2% of European travelers exiting northern Brazil (2007 publication) [13]
62.6% of humans and 35.6% of dogs in Araruama township (Rio de Janeiro State) are infested (2003 publication) [14]
1.6% of school children in Criciuma, Santa Catarina State (2009 publication) [15]
3.2% of dogs in Minas Gerais State (2012 publication) [16]
100% of free-ranging jaguar (*Panthera onca*) in the Pantanal region of Mato Grosso do Sul (2011 publication) [17]

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9.3% of soil samples in northeastern urban slums, 32% in a Yanomami village in the north (2010 publication) [18]

Overt outbreaks of tungiasis are reported. [19]

Infestation rates peak during the dry season (Fortaleza, 2001). [20] [21]

Tungiasis is implicated in the etiology of 10% of tetanus cases in Sao Paulo (2001 publication). [22]

Tungiasis is often acquired by foreign travelers in Brazil. [23-30]

Infection is common among rats, cats, jaguars [31] and dogs in endemic regions. [32]
- *Tunga penetrans* infection has been identified in a giant anteater (*Myrmecophaga tridactyla*) in Minas Gerais. [33]

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Typhoid and enteric fever

Agent	BACTERIUM. Salmonella serotype Typhi (other Salmonella species cause 'paratyphoid' fever) A facultative gram-negative bacillus
Reservoir	Human
Vector	None
Vehicle	Fecal-oral Food, Fly Water
Incubation Period	15d - 21d (range 5d - 34d)
Diagnostic Tests	Culture (blood, urine, sputum culture). Stool usually negative unless late untreated infection). Serology.
Typical Adult Therapy	Ceftriaxone 2 g IV q12h to q 24h X 5 to 7d. OR Ciprofloxacin 750 mg PO (400 mg IV) Q12h X 2w. OR Azithromycin 1 gram PO on day 1; then 500 mg days 2 to 7. Add corticosteroids if evidence of shock or decreased mental status.
Typical Pediatric Therapy	Ceftriaxone 50 to 80 mg/kg IV daily X 5 to 7d. OR Azithromycin 15 mg/kg PO on day 1; then 7.5 mg/kg on days 2 to 7.
Vaccines	Typhoid - injectable Typhoid - oral
Clinical Hints	Transient diarrhea followed by fever, splenomegaly, obtundation, rose spots (during second week of illness); leukopenia and relative bradycardia often observed; case fatality rate = 0.8% (treated) to 15% (untreated).
Synonyms	Abdominal typhus, Abdominaltyphus, Buiktyphus, Enteric fever, Febbre tifoide, Febbre tifoidea, Fiebre tifoidea, Paratifoidea, Paratyfus, Paratyphoid, Salmonella serotype Typhi, Tyfoid, Typhoid, Typhoide. ICD9: 002 ICD10: A01

Clinical

Enteric fever is a defined syndrome of systemic illness associated with *Salmonella* infection.
• Enteric fever caused by *S. typhi* is referred to as "typhoid fever," and that caused by *S. paratyphi*, is referred to as "paratyphoid fever."
• Symptoms are often nonspecific and insidious in onset. [1] [2]
• The differential diagnosis of fever, abdominal pain with hepatosplenomegaly also includes malaria, amebic liver abscess, brucellosis [3] , visceral leishmaniasis, and dengue fever.
• The clinical features of scrub typhus [4] and melioidosis may also mimic those of enteric fever. [5]

Acute illness:

Following an incubation period of 5 to 21 days, an initial enterocolitis may develops without associated fever.
• Constipation is present in 10 to 40% of patients; abdominal pain 20 to 40%; hepatosplenomegaly in 50%.
• Such symptoms as chills, diaphoresis, headache, anorexia, cough, sore throat, vertigo and myalgia often precede the onset of fever.
• Psychosis or confusion ("muttering delirium") occur in 5 to 10%, encephalopathy in 21% [6] , and seizures and coma in less than 1%.
• Patients appear acutely ill.
• Cervical lymphadenopathy develops in some patients, and pulmonary disease is rare at this stage.
• 3% have signs and symptoms of cholecystitis, and jaundice is reported in as many as 12% of cases. [7]
• Instances of "typhoid hepatitis" appear to represent super-infection by hepatitis virus, rather than a complication of typhoid fever. [8]

Course of illness and complications:

Symptoms resolve by the fourth week of infection without antimicrobial therapy.
• Weight loss, and debilitation may persist for months, and 10% of patients will experience a relapse.
• Relapse is more common among antibiotic-treated than non-treated patients.
• Intestinal perforation is characterized by recurrent fever, abdominal pain, intestinal hemorrhage and tachycardia occurring in the 3rd to 4th week of illness. 65.7% of perforations are solitary and involved the anti-mesenteric border of the terminal ileum [9-12] There is a male predominance among patients with typhoidal perforation. [13] During a typhoid fever outbreak in

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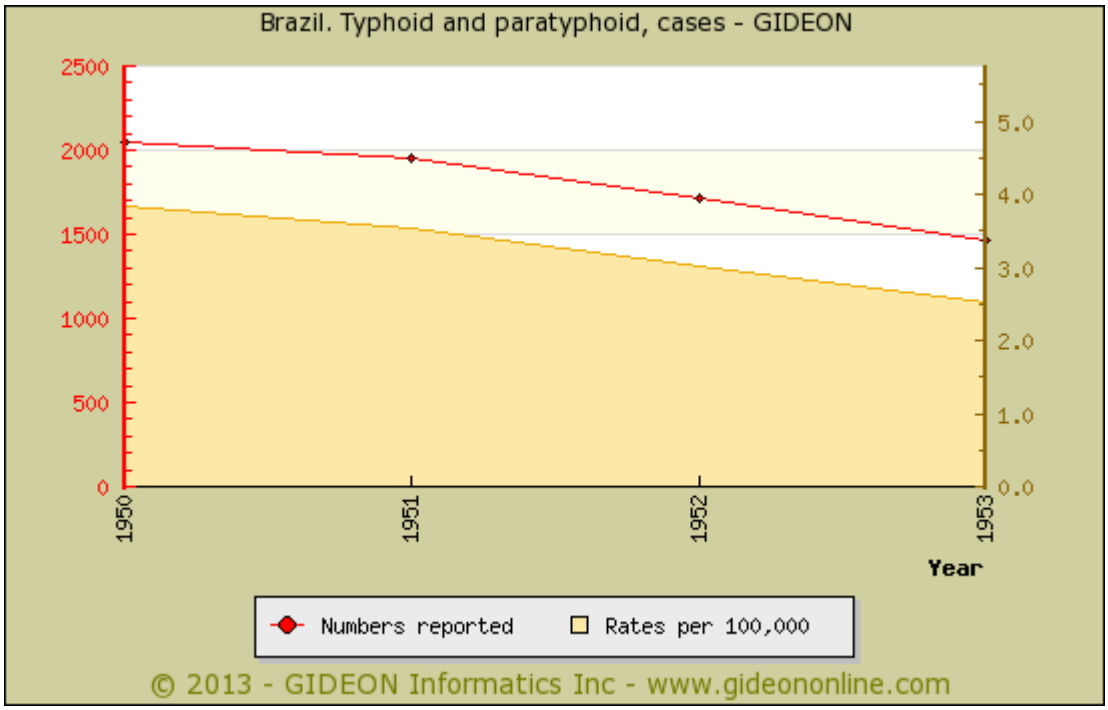
Uganda, 43% of patients presented with intestinal perforation. [14]
• 70% of pregnancies will end in miscarriage when complicated by untreated typhoid.
• Rare instances of acalculous cholecystitis [15-18] , gall-bladder perforation [19] , pancreatitis, intestinal intussusception [20] , rhabdomyositis, renal failure [21] , genital ulceration [22] , spondylitis/spondylodiscitis [23] , transverse myelitis [24] , cranial nerve palsy [25] , cerebral venous sinus thrombosis [26] , endophthalmitis [27] and ectopic abscesses [28] [29] have been reported in typhoid patients.
• The case-fatality rate is 10% to 15%

Carrier state:
The carrier state is defined as persistent shedding of *Salmonella typhi* in stool and/or urine for >=12 months. [30]
- Approximately 5% of people who contract typhoid continue to carry the disease after they recover.
• Long-term carriage is associated with an increased incidence of cancers of the gallbladder [31] [32] , pancreas, colo-rectum and lung. [33] [34]

Laboratory findings include leukopenia (albeit an initial leucocytosis is common), thrombocytopenia, coagulopathy and hepatic dysfunction.
• The most sensitive laboratory test for enteric fever is blood culture.
• Serum transaminase elevations appear to reflect myopathy rather than hepatic disease in most cases. [35]

This disease is endemic or potentially endemic to all countries.

Typhoid and enteric fever in Brazil



Graph: Brazil. Typhoid and paratyphoid, cases

Notes:
1. During 1945 to 1949, the mean annual incidence was 2,658 cases [36]

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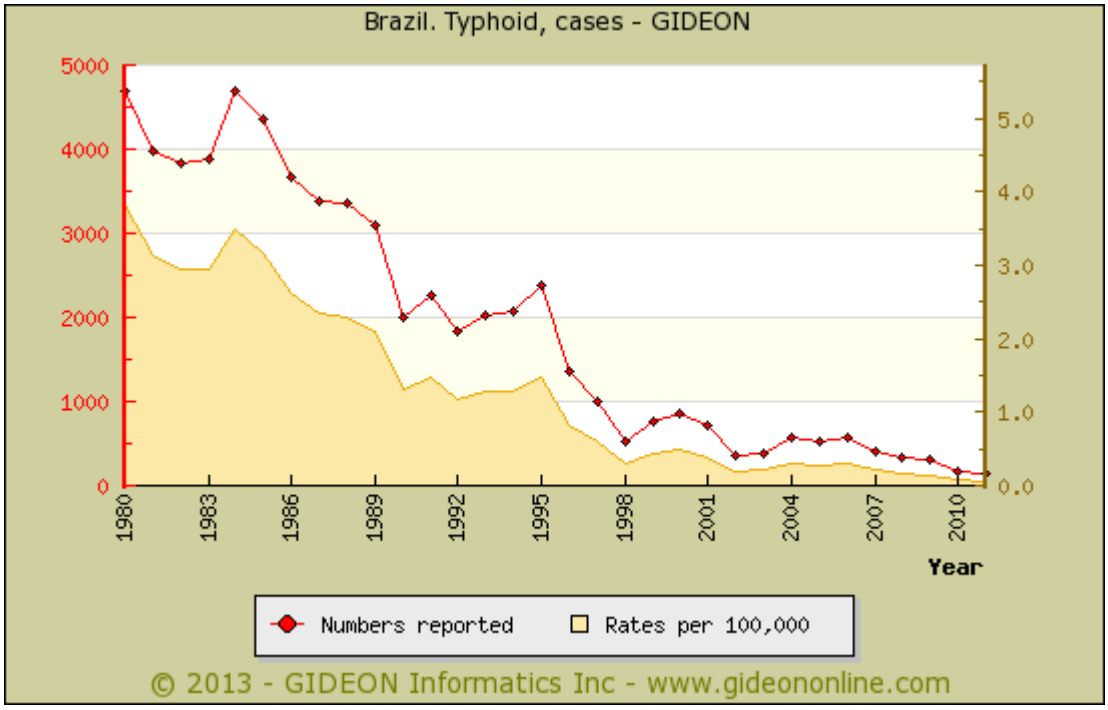
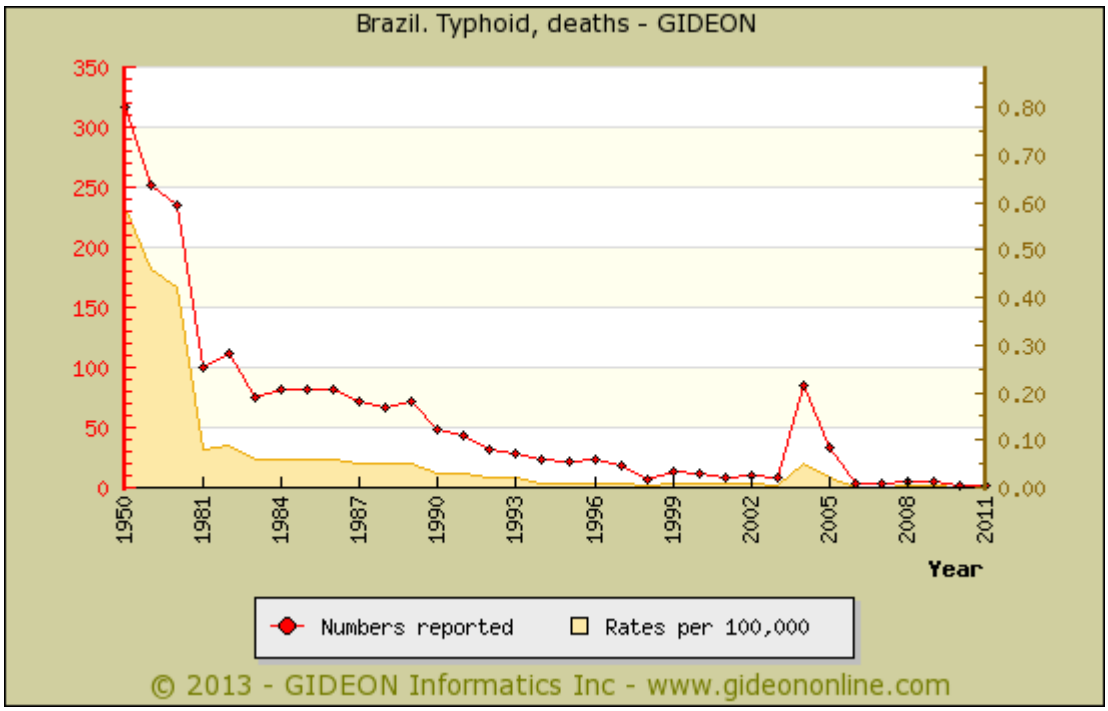


Graph: Brazil. Typhoid, cases

Notes:
1. 55,939 cases of typhoid were reported during 1980 to 2000 - 33.3% of these from Bahia; and only 1.7% from Rio de Janeiro and 2.6% from Sao Paulo.



Graph: Brazil. Typhoid, deaths

Notable outbreaks:

1972 - An outbreak (500 cases) was reported in Sao Paulo. [37]
2004 - An outbreak of typhoid was reported in Santos, Sao Paulo. [38]

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Typhus - endemic

Agent	BACTERIUM. Rickettsia typhi
Reservoir	Rat
Vector	Flea (Xenopsylla or Nosopsyllus spp.)
Vehicle	None
Incubation Period	10d - 12d (range 4d - 18d)
Diagnostic Tests	Serology. Identification of rickettsiae in smear or culture of skin lesions. Nucleic acid amplification.
Typical Adult Therapy	Doxycycline 100 mg BID X 3 to 5d
Typical Pediatric Therapy	Doxycycline 2 mg/kg BID X 3 to 5d (maximum 200 mg/day); or Chloramphenicol 12.5 mg/kg QID X 3 to 5d
Clinical Hints	Fever, headache and myalgia; truncal maculopapular rash (present in 60%) appears on days 3 to 5 and persists for 4 to 8 days; fever resolves after 12 to 16 days; case fatality rate (untreated) = 2%.
Synonyms	Endemic typhus, Murine typhus, Rickettsia typhi, Ship typhus, Tifo murino, Tifus pulgas, Vlektyphus. ICD9: 081.0 ICD10: A75.2

Clinical

The features of endemic typhus are similar to those of epidemic typhus, but less severe. [1]
- Headache and myalgia predominate.
- The rash is nonspecific and may be lacking in 50% of patients. [2]
- Major complications are rare.
- The severity of infection has been associated with old age, delayed diagnosis, hepatic and renal dysfunction, central nervous system abnormalities, and pulmonary compromise.
- Ocular complications include uveitis, retinal hemorrhage, choroidal dots, papilledema and optic neuritis [3] [4]
- Rare instances of splenic infarction [5] and hemophagocytic syndrome have been reported. [6]
- As many as 4% of hospitalized patients die.

This disease is endemic or potentially endemic to all countries.

Typhus - endemic in Brazil

Seroprevalence surveys:

1.1% of healthy persons in Minas Gerais (2005 publication) [7]

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Urinary tract infection

Agent	BACTERIUM OR FUNGUS. Escherichia coli, other facultative gram negative bacilli, enterococci, et al
Reservoir	Human
Vector	None
Vehicle	Endogenous
Incubation Period	Variable
Diagnostic Tests	Urine culture and leucocyte count.
Typical Adult Therapy	Antimicrobial agent(s) directed at known or likely pathogen
Typical Pediatric Therapy	As for adult
Clinical Hints	Fever, dysuria, frequency, flank pain and vomiting; infection in children or men and infection which relapses in women may warrant radiological studies to rule out underlying obstruction or calculus.
Synonyms	Cistite, Cistitis, Cystite, Cystitis, Pielite, Pielitis, Pielonefrite, Pielonefritis, Prostatite, Pyelitis, Pyelonephrite, Pyelonephritis, Trigonitis, Tubulointerstitial nephritis, Urethritis, Uretrite, Zystitis. ICD9: 791.9,136.9,599.0,590,601.0 ICD10: N10,N30,N41

Clinical

Young children often exhibit nonspecific signs such as fever, poor feeding and vomiting.
• Abdominal pain may be present.
• After early childhood, dysuria, urgency, and frequency are generally present in UTI.
• Adult women with cystitis have frequent and urgency, often with lower abdominal or lower back pain.
• The urine may be foul smelling or turbid and is often bloody.
• Onset of symptoms is usually abrupt.
• Some infections progress to upper tract involvement, with fever, rigors, nausea, vomiting, abdominal and flank pain.
• Classical signs of 'upper' vs. 'lower' UTI are often misleading and do not necessarily point to the location of infection.

In the elderly, UTIs are often asymptomatic or manifest by nonspecific signs.
• Frequency, urgency, nocturia, and incontinence in this age group may also mimic other disorders in this age group.
• Infection associated with neurogenic bladders and indwelling catheters may not necessarily present with localizing symptoms.

Acute uncomplicated cystitis is most common in young women but may also be seen in men, children or the elderly. [1]
• Typical symptoms include dysuria, frequency, urgency, and suprapubic or pelvic pain. [2]
• Suprapubic tenderness is present in 10 to 20 percent, and gross hematuria in 20 to 30 percent.
• Approximately ten percent of patients with symptoms of acute cystitis will be found to have occult infection of the upper urinary tract.
• Bacterial vaginosis may predispose to urinary tract infection [3]

Acute pyelonephritis presents with flank, low back, or abdominal pain, in addition to fever, rigors, sweats, headache, nausea, vomiting, malaise, and prostration. [4]
• Antecedent or concomitant symptoms of cystitis may or may not be present.
• Fever and flank pain are relatively specific indicators of renal infection.
• A minority of patients with pyelonephritis develop septicemia, or necrotizing renal or perinephric abscesses.
• The latter are often associated with urinary tract obstruction or diabetes [see Perinephric abscess].

All urinary infections in males should be considered complicated until proven otherwise, and prompt a careful search for anatomical or functional abnormality of the urinary tract.

Comprehensive reviews of prostatitis. [5] [6]

This disease is endemic or potentially endemic to all countries.

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Vaccinia and cowpox

Agent	VIRUS - DNA. Poxviridae, Orthopoxvirus. Cowpox virus
Reservoir	Cattle Cat Rodent
Vector	None
Vehicle	Cattle Cat
Incubation Period	2d - 4d
Diagnostic Tests	Viral isolation from skin exudate or biopsy. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Secretion precautions; supportive
Typical Pediatric Therapy	As for adult
Vaccine	Vaccinia immune globulin
Clinical Hints	Vesicles or pustules (usually on hand) progressing to crusts; painful regional lymphadenopathy; follows contact with infected animals or smallpox vaccination (largely abandoned); see Buffalopox (India note).
Synonyms	Aracatuba, Buffalopox, Camelpox, Cantagalo, Cowpox, Passatempo, Vaccinia, Vaiolo. ICD9: 051.0 ICD10: B08.0

Clinical

Cowpox is characterized by single or multiple vesicles of the hands or face, which evolve to pustules that may persist for two or more months. [1]
• The surrounding tissues are swollen and painful, and tender regional adenopathy is present.
• Most lesions occur on the thumbs, forefinger and first interdigital cleft.
• Secondary lesions may appear on the hands, forearms or face through self-inoculation.
• Facial cellulitis with necrotizing lymphadenitis has been reported. [2]
• Vaccinia infections caused by unintentional transfer from vaccination sites usually involve the face, nose, mouth, lips, genitalia, anus, or eye. [3]
• Poxviruses are known to remain infectious in the scabs of patients for months to years. [4]
• Infectious virus is present at the site of primary vaccination for at least 21 days. [5]

The rash evolves as follows:
• One to six days following inoculation), an inflamed macule appears at the site of contact.
• On days 7 to 12, the lesion becomes papular, then vesicular.
• On days 13 to 20, the vesicle becomes hemorrhagic and then pustular, and tends to ulcerate, with surrounding edema and induration. Secondary contiguous lesions may appear.
• After 3 to 6 weeks, the vesicopustule progresses to a hard, black eschar • often surrounded by edema, induration and erythema.
• At weeks 6 to 12, the eschar sloughs, and the lesion heals with scarring.
• Additional findings include lethargy, vomiting, sore throat, conjunctivitis, periorbital edema and keratitis during the early phase of infection.
• A generalized rash does not occur.

One case of post-cowpox encephalitis has been reported.
• During 2002 to 2006, cases of vulvar vaccinia in the United States were acquired through sexual exposure to recently-vaccinated military personnel. [6]

Previous smallpox vaccination may attenuate the severity of infection.

The clinical features of buffalopox include fever, lymphadenopathy and pox lesions on the hands (acquired from contact with the udders of cattle [7]).

Camelpox virus infection in humans is characterized by papules, vesicles, ulceration and finally scabs over fingers and hands (eg, areas in contact with infected camels) [8]

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This disease is endemic or potentially endemic to 14 countries. Although Vaccinia and cowpox is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

Vaccinia and cowpox in Brazil

Viral variants:

The emergent strain of 'cowpox' appears to be caused by a distinct virus, "**Cantagalo virus**." [9-13]

- A second emergent variant, **Aracatuba virus**, has been associated with human and bovine infection in Sao Paulo State. [14] [15]
- A related agent, **Passatempo virus**, has been identified in skin lesions of persons involved in milking cows in Brazil. [16]
- Some experts suggest that reported outbreaks represent cases of pseudocowpox.

Rodent urine and feces may play a role in the transmission of these viruses. [17]

- There is evidence that peridomestic rodents may provide a link between wildlife and bovine outbreaks. [18]
- Seropositivity toward Orthopoxviruses has been documented in a variety of monkey species (*Cebus apella*, *Alouatta caraya*, *Nasua nasua* and *Dasyprocta* spp. [19]

52.0% of persons working in areas of bovine outbreaks will become infected. [20]

Seroprevalence surveys:

27.89% of persons in rural Acre (Orthopoxvirus, 2010 publication) [21]
24.4% of monkeys in the Brazilian Amazon (Orthopoxviruses, 2001 to 2002) [22]
31.2% of milk buffaloes in Minas Gerais (Orthopoxvirus, 2010). [23]

Notable outbreaks:

1955 to 1956 - An outbreak (16 cases, 0 fatal) followed vaccination were reported in Sao Paolo.
1956 - An outbreak (3 cases, 0 fatal) occurred on a dermatology ward in Sao Paolo.
1997 to 1998 - An outbreak (18 cases) of 'cowpox' was reported in Mato Grosso do Sul.
1999 - An outbreak (3 humans and 10 cows) of 'cowpox' was reported in Rio de Janeiro State. [24]
2001 - An outbreak of vaccinia (1,020 cattle and numerous human cases) was reported in Minas Gerais.
2002 - An outbreak (37 humans and 1,500 cattle) of 'cowpox' was reported in Lagoinhan, Sao Paulo State. [25]
2002 - Accidental infection of a laboratory worker was reported. [26]
2001 to 2003 - 74 cases of Cantagalo virus infection were documented in Sao Paulo and Goias States. [27]
2007 - An outbreak (33 cases) of suspected vaccinia was reported in West Figueiropolis and Araputanga (Mato Grosso). [28]
2008 (publication year) - An outbreak of cowpox was reported among humans and cattle in Southwest region of Sao Paulo State. [29]
2008 - An outbreak (15 cases) of Cantagalo virus infection was reported in Tocantins. [30]
2009 - Outbreaks (2 outbreaks) of bovine vaccinia were reported in the midwestern region of Sao Paulo State. [31]
2011 - An outbreak (3 human and 91 bovine cases) of group 2 vaccinia virus infection was reported in Minas Gerais. [32]
2013 (publication year) - An outbreak of vesicular disease due to Orf virus and a parapoxvirus was reported among dairy cows in midwestern Brazil. [33]

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Varicella

Agent	VIRUS - DNA. Herpesviridae, Alphaherpesvirinae: Human Herpesvirus 3 (Varicella-zoster virus)
Reservoir	Human
Vector	None
Vehicle	Air Direct contact
Incubation Period	2w - 3w
Diagnostic Tests	Viral culture (vesicles). Serology. Nucleic acid amplification.
Typical Adult Therapy	Respiratory isolation. Severe/complicated cases: Acyclovir 10 to 12 mg/kg IV q8h X 7d Adolescent / young adult: 800 mg PO X 5 per day X 7 d. Alternatives: Valacyclovir 1 g PO TID; or Famciclovir 500 mg PO TID
Typical Pediatric Therapy	Respiratory isolation. Acyclovir [severe/complicated cases] 150 mg/sq m IV q8h X 7d
Vaccines	Varicella Varicella-Zoster immune globulin
Clinical Hints	Cough and fever followed by a pruritic papulovesicular rash after 1 to 2 days; pneumonia is often encountered; case fatality rate = 4.3 per 100,000 cases (7% in immune-suppressed patients).
Synonyms	Chickenpox, Lechina, Skoldkopper, Vannkopper, Varicela, Varizellen, Vattenkoppor, Waterpokken, Windpocken. ICD9: 052 ICD10: B01

Clinical

Acute infection:
The predominant features of varicella are fever, cough, malaise, lymphadenopathy and a generalized pruritic vesicular rash typically consisting of 250 to 500 lesions.
• The rash generally begins on the scalp and proceeds to the trunk and extremities, with most lesions on the trunk.
• Skin lesions are initially maculopapular, progressing to vesicles on an erythematous base. [1]
• Atypical varicella, including lesions on palms and soles, may mimic monkeypox in endemic areas. [2]

Complications:
Complications include hepatitis [3] [4] , encephalitis (notably involving the cerebellum) [5-7] , myelitis [8] , arthritis [9] , secondary bacterial infections, Reye's syndrome, disorders of the facial [10-12] and other cranial nerves [13] , meningitis [14] , cerebral venous thrombosis [15] , transverse myelitis [16] , acute urinary retention [17] , pancreatitis [18] , appendicitis [19] , pneumonia [20-22] , empyema [23] , acute respiratory distress syndrome (ARDS) [24-27] , spontaneous pneumothorax [28] , myocarditis [29] , atrioventricular block [30] , hemorrhagic pericarditis [31] [32] , optic neuritis [33-35] , uveitis [36] , acute retinal necrosis [37] [38] , necrotizing scleritis [39] [40] , deep venous thrombosis or thromboembolism [41] , purpura fulminans [42] [43] , idiopathic thrombocytopenic purpura [44] and hemophagocytic lymphohistiocytosis. [45]
• Pyomyositis [46] , osteomyelitis [47] , necrotizing fasciitis or Fournier's gangrene may occasionally complicate varicella [48]
• Post varicella cerebral infarction has been described in young, previously healthy children within a few months of VZV infection and is characterized by middle cerebral artery territory infarction and proximal MCA disease. [49] A similar condition has been reported in immunocompromised patients following herpes zoster involving the ophthalmic branch of the trigeminal nerve as well as in the context of primary varicella complicated by granulomatous angiitis [50] Extra-cranial vascular thrombosis of large or small vessels has also been reported [51]
• VZ virus infection may be associated with facial nerve palsy [52] or Ramsay-Hunt syndrome (Bell palsy unilateral or bilateral, vesicular eruptions on the ears, ear pain, dizziness, preauricular swelling, tingling, tearing, loss of taste sensation, and nystagmus) [53]
• Immunocompromised individuals, neonates, infants, adolescents and adults are at risk of severe illness and complications. [54-56]
• VZ virus infection can be a presenting symptom of hyperparathyroidism and occurs twice as often in persons with hypercalcemia than age-matched controls. [57]
• Use of nonsteroidal anti-inflammatory drugs during primary varicella, has been implicated as a risk factor for subsequent occurrence of streptococcal necrotizing fasciitis.

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Anterior uveitis • differential diagnosis:
Anterior uveitis due to Rubella virus is characterized by younger age at onset and a chronic course, typically associated with cataract at presentation. [58]
• Rubella virus has been implicated in the etiology of Fuchs heterochromic iridocyclitis. [59]
• Anterior uveitis due to Herpes simplex and Varicella-Zoster viruses is more common in adults, and often follows an acute course.
• Herpes simplex anterior uveitis presents with conjunctival redness, corneal edema, a history of keratitis, and the presence of posterior synechiae. Anterior chamber inflammation is common with Herpes simplex virus, while vitritis is more common with Rubella and Varicella-Zoster virus.
• Rubella, Herpes simplex and Varicella-zoster viruses are associated with intraocular pressure of more than 30 mmHg and development of glaucoma (18%-30%; P = 0.686).
• Focal chorioretinal scars were present in 22% of Rubella cases, 0% of HSV and in 11% of VZV uveitis cases.

Perinatal infection: [60]
Newborn infants whose mothers had onset of varicella within 5 days before delivery or within the 48 hours after delivery are at risk for neonatal varicella. [61-66]
• Neonatal varicella carries a case-fatality rate as high as 30%.
• Maternal infection [67] during the first 20 weeks of pregnancy carries a risk (0.4% to 2.0%) of congenital varicella, characterized by low birth weight, hypoplasia of extremities, dermal scarring, focal muscular atrophy, encephalitis, cortical atrophy, chorioretinitis and microcephaly. [68-70]

This disease is endemic or potentially endemic to all countries.

Varicella in Brazil

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

Prevalence surveys:
0.5% of CSF specimens from cases of suspected viral CNS infection (Sao Paulo, 2006 publication) [71]

Seroprevalence surveys:
94.2% of young adults: 88.7% in Fortaleza, 99.5% in Curitiba; 89.4% in tropical regions and 97.35 in temperate regions (2003 publication) [72]
90% of children in Sao Paulo by age 10 years (1992 to 1994) [73]
45.5% of urban children (2011 publication) [74]
80.8% of Kuikuro and Kaiabi non-vaccinated indigenous communities (2001, Xingu National Park) [75]
99.1% of health care workers in neonatal units (Sao Paulo, 2002) [76]

During 1985 to 2004, chickenpox was identified as the underlying cause in 1,037 deaths and an associated cause in 150 deaths in Sao Paulo. [77]

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Venezuelan equine encephalitis

Agent	VIRUS - RNA. Togaviridae, Alphavirus: Venezuelan equine encephalitis virus
Reservoir	Rodent Horse
Vector	Mosquito - Culex [Melanoconion] spp, Aedes taeniorhynchus, Psorophora confinnis, Anopheles aquasalis)
Vehicle	None
Incubation Period	2d - 5d (range 1d - 6d)
Diagnostic Tests	Viral culture (throat, blood, brain tissue). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Vaccine	Western equine encephalitis
Clinical Hints	Fever, myalgia, arthralgia, vomiting, conjunctivitis and encephalitis; encephalitis more common and more severe among children; case-fatality rate = 20%.
Synonyms	Everglades, Peste loca, Rio Negro, Tonate. ICD9: 066.2 ICD10: A92.2

Clinical

Clinical features of eastern encephalitis (EEE) [1] , western equine encephalitis (WEE) and Venezuelan equine encephalitis (VEE) are similar, and will be discussed as a group. [2] [3]

EEE and WEE infections begin with headache, high fever, chills and vomiting.
• Vertigo, sore throat and respiratory symptoms are common in WEE infection.
• CNS involvement is heralded by confusion and somnolence which may progress to coma.
• Focal or generalized seizures are most common in younger patients.
• Physical examination reveals nuchal rigidity, depressed or hyperactive reflexes, tremors, or spastic paralysis.

The peripheral blood reveals a leukocytosis, more prominent in EEE than in WEE.
• Cerebrospinal fluid protein levels are elevated.
• WEE tends to produce a lymphocytic pleocytosis of 50 to 500/mm3; and EEE of 600 to 2000/mm3 .

Sequelae are most severe following EEE infection, and include mental retardation, behavioral changes, seizure disorders and paralysis.
• Sequelae occur in 30% of infants recovering from WEE, and 70% of infants recovering from EEE.

WEE is characterized by a case-to-infection ratio of approximately 1/1,000 for adults, 1/60 for children and 1/1 for infants. [4]
• Respiratory symptoms and overt pneumonia are common. [5]
• Younger patients tend to suffer more severe disease, with rapid onset of symptoms, presence of seizures and permanent neurological residua. [6]
• Parkinsonism may occur in adults following WEE.

VEE is often characterized by a mild flu-like illness in endemic countries.
• Overt infection is heralded by fever, chills, myalgia, headache with or without photophobia, hyperesthesia, and vomiting.
• Sore throat is noted in some cases.
• 4% of children and less than 1% of adults progress to overt encephalitis, a few days to a week following the prodrome.
• Encephalitis is characterized by nuchal rigidity, ataxia, convulsions, coma, and paralysis.
• Lymphopenia, is common, often associated with neutropenia and mild thrombocytopenia.
• Hepatic dysfunction is common, and CSF examination reveals a few hundred lymphocytes.
• The case-fatality rate is less than 1%, but increases to 20% in cases of encephalitis.

Rio Negro virus has been associated with acute febrile illness among humans in Argentina. [7]

This disease is endemic or potentially endemic to 21 countries.

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Vibrio parahaemolyticus infection

Agent	BACTERIUM Vibrio parahaemolyticus A facultative gram-negative bacillus
Reservoir	Marine water Seafood Fish
Vector	None
Vehicle	Seafood
Incubation Period	10h - 20h (range 2h - 4d)
Diagnostic Tests	Stool culture - alert laboratory when this organism is suspected.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Vomiting and explosive diarrhea, 4 to 24 hours following ingestion of seafood (often steamed crabs); diarrhea may persist for 7 to 10 days; case fatality rate = 0.1%.
Synonyms	Vibrio parahaemolyticus. ICD9: 005.4 ICD10: A05.3

Clinical

Symptoms usually begin within 10 to 20 hours after ingestion of seafood, and persist for 2 to 10 days.
- Illness is characterized by vomiting (50%), abdominal pain and watery or explosive diarrhea.
- Fever is noted in 25% of patients.
- Dysentery has been described in some cases. [1]

Rare instances of bacteremia and extra-intestinal infection are reported. [2-4]

This disease is endemic or potentially endemic to all countries.

Vibrio parahaemolyticus infection in Brazil

Prevalence surveys:

11.6% of natural and precooked mussels. (Rio de Janeiro, 2007 publication) [5]
100% of oysters in the Cananeia estuary (Sao Paulo, 1998 to 1999) [6]
100% of raw oysters collected in restaurants, supermarkets, groceries and beach huts in Sao Paulo State (2006 to 2007) [7]
5% of fish and 19% of shellfish recovered from coastal waters (1980 publication) [8]
99.2% of oyster samples collected in an estuary in the southern coast of Sao Paulo State (2009 publication) [9]

Vibrio parahaemolyticus, *Vibrio carchariae*, *Vibrio alginolyticus* and *Vibrio vulnificus* have been isolated from oysters (*Crassostrea rhizophorae*) served raw in restaurants. (Rio de Janeiro, 2007 publication) [10]
- During 2008 to 2009, *Vibrio parahaemolyticus* was identified among oysters in Fortaleza. [11]

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West Nile fever

Agent	VIRUS - RNA. Flaviridae, Flavivirus: West Nile virus
Reservoir	Bird Horse Bat ? Tick
Vector	Mosquito (Culex univittatus. Cu. pipiens, Cu. vishnui, Cu. neavei, Coquillettidia, Aedes and Anopheles spp.)
Vehicle	Blood transmission [rare]
Incubation Period	3d - 6d (range 1d - 7d)
Diagnostic Tests	Viral culture (blood, CSF). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Clinical Hints	Myalgia, arthralgia, lymphadenopathy, headache, conjunctivitis and a macular rash; sporadic instances of encephalitis, meningitis and myocarditis are reported; illness resolves within one week in most cases.
Synonyms	Bagaza, Fiebre del Oeste del Nilo, Lourdige, Near Eastern equine encephalitis, Ntaya, Usutu, WNF. ICD9: 066.4 ICD10: A92.3

Clinical

Acute infection:
West Nile fever in humans usually is a minor influenza-like illness, characterized by an abrupt onset of moderate to high fever lasting 3 to 5 days.
• The fever is occasionally biphasic, and may be accompanied by rigors.
• Additional findings include frontal headache, sore throat, backache, myalgia, arthralgia, fatigue, conjunctivitis and retrobulbar pain. [1]
• A maculopapular or roseolar rash [2] [3] appears in approximately 50% of cases, spreading from the trunk to the extremities and head.
• Lymphadenopathy, anorexia, nausea, abdominal pain, diarrhea, and respiratory symptoms are also encountered.

Neuroinvasive disease:
Occasionally (<15% of cases), acute aseptic meningitis or encephalitis occurs, associated with neck stiffness, vomiting, confusion, disturbed consciousness, somnolence, tremor of extremities, abnormal reflexes, convulsions, pareses, and coma. [4] [5]
• Such patients may then develop anterior myelitis and acute asymmetric flaccid paralysis, reminiscent of poliomyelitis or Guillain-Barre syndrome. [6-9] Rare instances of progressive brachial diplegia are reported. [10]
• Focal encephalitis with seizures may mimic herpes simplex encephalitis. [11] [12]
• A case of stuttering (? lingual myoclonus) during the course of West Nile fever has been reported. [13]
• Risk factors for neuroinvasive disease include age >45 years, male sex, hypertension and diabetes mellitus. [14]
• Multifocal chorioretinitis is common among patients with neuroinvasive disease. [15-20]
• Most fatal cases occur in patients older than 50 years. [21]

Hepatosplenomegaly, hepatitis, pancreatitis [22] , myocarditis [23] and hemorrhagic fever have been reported. [24]

Prolonged convalescence (up to one year) may follow recovery from encephalitis; and myalgia, confusion and lightheadedness may persist beyond this period. [25-29]
• Recovery is complete (less rapid in adults than in children, often accompanied by long-term myalgias and weakness), and permanent sequelae have not been reported.
• Prolonged depression persists in as many as 31% of patients following recovery. [30] [31]
• Residual kidney disease is common following West Nile fever. [32]

Laboratory findings:
Laboratory findings consist of a slightly increased sedimentation rate and mild leukocytosis.
• Profound and prolonged lymphocytopenia is reported in some cases. [33]
• Cerebrospinal fluid in patients with central nervous system involvement is clear, with moderate pleocytosis and elevated

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protein.
• A distinctive CSF plasmacytosis may be present.
• The virus can be recovered from the blood for up to 10 days in immunocompetent febrile patients, and as late as 22 to 28 days after infection in immunocompromised patients.
• Peak viremia occurs 4 to 8 days postinfection.

Usutu virus infection was reported in two immune-compromised humans in 2009. [34-36]
• Clinical features included fever of 39.5 °C, headache and neurological findings. One patient developed fulminant hepatitis.

Ntaya virus, a related flavivirus, has been associated with febrile illness and neurological findings. [37]

This disease is endemic or potentially endemic to 86 countries. Although West Nile fever is not endemic to Brazil, imported, expatriate or other presentations of the disease have been associated with this country.

West Nile fever in Brazil

Prevalence surveys:

0% of patients with viral meningitis/encephalitis in Rio de Janeiro (2010 publication) [38]
0% of mosquitoes in the Pantanal (2011 publication) [39]

Seroprevalence surveys:

3% of horses and 0% caimans from the Pantanal. (2009) [40] [41]
8% of horses and 3.2% of chickens from the Pantanal (2012 publication) [42]

Potential vectors for West Nile fever in Brazil include Culicidae [43] and *Coquillettidia venezuelensis*. [44]

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Western equine encephalitis

Agent	VIRUS - RNA. Togaviridae, Alphavirus: Western equine encephalitis virus
Reservoir	Bird Horse Amphibian Reptile
Vector	Mosquito (Culex tarsalis)
Vehicle	None
Incubation Period	5d - 15d
Diagnostic Tests	Viral culture (CSF, blood, brain tissue). Serology. Nucleic acid amplification. Biosafety level 2.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Vaccine	Western equine encephalitis
Clinical Hints	Headache, back pain, vomiting, meningitis and encephalitis; disease most severe in children; most often encountered in late summer and autumn in temperate regions; resolves after 5 to 10 days; case fatality rate = 5%.
Synonyms	WEE. ICD9: 062.1 ICD10: A83.1

Clinical

Clinical features of eastern encephalitis (EEE) [1] , western equine encephalitis (WEE) and Venezuelan equine encephalitis (VEE) are similar, and will be discussed as a group. [2] [3]

EEE and WEE infections begin with headache, high fever, chills and vomiting.
• Vertigo, sore throat and respiratory symptoms are common in WEE infection.
• CNS involvement is heralded by confusion and somnolence which may progress to coma.
• Focal or generalized seizures are most common in younger patients.
• Physical examination reveals nuchal rigidity, depressed or hyperactive reflexes, tremors, or spastic paralysis.

The peripheral blood reveals a leukocytosis, more prominent in EEE than in WEE.
• Cerebrospinal fluid protein levels are elevated.
• WEE tends to produce a lymphocytic pleocytosis of 50 to 500/mm3; and EEE of 600 to 2000/mm3 .

Sequelae are most severe following EEE infection, and include mental retardation, behavioral changes, seizure disorders and paralysis.
• Sequelae occur in 30% of infants recovering from WEE, and 70% of infants recovering from EEE.
• Parkinsonism may occur in adults following WEE.

VEE is often characterized by a mild flu-like illness in endemic countries.
• Overt infection is heralded by fever, chills, myalgia, headache with or without photophobia, hyperesthesia, and vomiting.
• Sore throat is noted in some cases.
• 4% of children and less than 1% of adults progress to overt encephalitis, a few days to a week following the prodrome.
• Encephalitis is characterized by nuchal rigidity, ataxia, convulsions, coma, and paralysis.
• Lymphopenia, is common, often associated with neutropenia and mild thrombocytopenia.
• Hepatic dysfunction is common, and CSF examination reveals a few hundred lymphocytes.
• The case-fatality rate is less than 1%, but increases to 20% in cases of encephalitis.

WEE is characterized by a case-to-infection ratio of approximately 1/1,000 for adults, 1/60 for children and 1/1 for infants.
• Respiratory symptoms and overt pneumonia are common. [4]
• Younger patients tend to suffer more severe disease, with rapid onset of symptoms, presence of seizures and permanent neurological residua. [5]

This disease is endemic or potentially endemic to 7 countries.

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Western equine encephalitis in Brazil

Seroprevalence surveys:

0% of persons living in the Ribeira Valley, Sao Paulo (2000 publication) [6]
36.4% of horses in the Nhecolandia sub-region of South Pantanal (2010 publication) [7]
1.2% of horses in the Brazilian Pantanal (1993 publication) [8]

Seropositive birds have been identified in Sao Paulo State (1978 to 1990) [9]

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Whipple's disease

Agent	BACTERIUM. Actinomycetes, Tropheryma whipplei A gram positive bacillus
Reservoir	Unknown
Vector	None
Vehicle	None
Incubation Period	Unknown
Diagnostic Tests	Identification of inclusions in lamina propria (other tissues). Tissue culture. Nucleic acid amplification.
Typical Adult Therapy	Ceftriaxone 2.0 g IV daily X 14 days. OR Penicillin G 6 to 324 million units daily + + streptomycin 1 g daily X 14d. Then: Sulfamethoxazole/trimethoprim 800/160 mg PO BID X 1 year. OR Doxycycline 100 mg PO BID X 1 year
Typical Pediatric Therapy	This disease is not described in children
Clinical Hints	A chronic multisystem disorder characterized by weight loss, diarrhea, abdominal and joint pain; dermal hyperpigmentation, fever and lymphadenopathy often present; PAS-positive macrophages present in intestinal biopsy material.
Synonyms	Intestinal lipodystrophy, Lipophagic granulomatosis, Mesenteric chyladenectasis, Steatorrhea arthropericarditica, Tropheryma whipplei. ICD9: 040.2 ICD10: K90.8

Clinical

The typical patient with Whipple's disease has a history of recurrent arthralgia or arthritis involving multiple joints for several years. [1]

- Joint complaints precede systemic and gastrointestinal disease in approximately one-third of patients [2] , and may persist for years in the absence of diarrhea. [3]
- Infection of prosthetic joints has been reported. [4]
- Diarrhea, low-grade fever and weight loss are characteristic, and hyperpigmentation is present in 50% of patients.
- Generalized lymphadenopathy is common.
- A syndrome of dementia and obesity or ataxia linked associated with *T. whipplei* infection has been recently described. [5]

As many as one third of the patients develop cardiac involvement characterized by the presence of systolic murmurs, a pericardial friction rub, congestive heart failure, and nonspecific electrocardiographic changes. [6]

- The most common pathological changes are endocarditis [7-9] with negative blood cultures, presenting with thickened and deformed mitral or aortic valves. [10]
- In one series, Whipple's disease was identified in 6.3% of patients with culture-negative endocarditis (2011 publication) [11]
- 30 to 40% of patients develop pleuritic chest pain, chronic nonproductive cough, and dyspnea.
- The chest X-ray may show a pleural effusion or pulmonary infiltrates.
- Isolated *T. whipplei* endocarditis may occur without other systemic features of Whipple's disease. [12] [13]

Relapse of Whipple's disease has been reported following therapy. [14]

- Recurrence of symptoms following therapy may represent an immune reconstitution syndrome. [15]

Tropheryma whipplei was isolated from 6.4% of blood specimens from febrile patients with cough (Senegal, 2008 to 2009) [16]

Other features of Whipple's disease include personality changes or dementia [17-19] , hypersomnia [20] , amnesic syndrome [21] , peripheral or cranial nerve neuropathy [22] , encephalitis [23] [24] , cerebral pseudotumor [25] [26] , chronic headache [27] , endocarditis [28-31] , pericarditis [32] , pneumonia [33] [34] , pulmonary hypertension [35] [36] , subcutaneous nodules, anemia, myoclonus, ataxia [37] , chorioretinitis [38] , vitritis [39] , uveitis [40] [41] , Parinaud syndrome [42] . salcroiliitis [43] and spondylitis [44] , hypoalbuminemia and hypokalemia. [45]

The features of Whipple's disease may resemble those of lymphoma, celiac disease, Crohn's vasculitis, sepsis, an inflammatory process, liposarcoma, rheumatoid arthritis, seizure disorder, cerebrovascular accident, xanthoma, or central

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nervous system neoplasm. [46]

This disease is endemic or potentially endemic to all countries.

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Yaws

Agent	BACTERIUM. Treponema pallidum subsp. pertenue: microaerophilic gram-negative spirochete
Reservoir	Human ? Non-human primate
Vector	None
Vehicle	Contact ? Insect bite ? Fomite
Incubation Period	3w - 5w (range 10d - 12w)
Diagnostic Tests	VDRL and antitreponemal tests (FTA, MHTP) positive as in syphilis.
Typical Adult Therapy	Benzathine Penicillin G 1.2 million units IM as single dose. A single oral dose of Azithromycin is also effective.
Typical Pediatric Therapy	Benzathine Penicillin G : Weight <14kg: 300,000u IM Weight 14 to 28kg: 600,000u IM Weight >28kg - 1.2 million u IM A single oral dose (30 mg/kg) of Azithromycin is also effective.
Clinical Hints	Dermal papillomata, periostitis and soft tissue suppuration; regional lymphadenopathy common; relapses often seen during initial 5 years of illness; gummas and hyperkeratotic plaques in later stages.
Synonyms	Anakhre, Bouba, Breda's disease, Charlouis' Disease, Frambesia, Gangosa, Goundou, Granuloma tropicum, Gundo, Henpue, Henpuye, Ogo Mutilans, Parangi, Patek, Pian, Treponema pallidum subsp pertenue. ICD9: 102 ICD10: A66

Clinical

Yaws has three clinical stages. [1] [2]
• Stage 1 is characterized by the a variety of flat and/or raised skin lesions.
• Stage 2 (Gangosa Syndrome, Ogo, or Rhinopharyngitis Mutilans) may involve the bones, joints, and/or skin.
• Stage 3 (Goundou Syndrome, Henpue, Henpuye, Gundo, or Anakhre) may also involve the bones, joints, and/or skin.

After an incubation period of approximately 3 weeks, a primary painless 2 to 5 cm pruritic papule ("mother yaw") appears at the site of inoculation. [3]
• The lesions may ulcerate, but generally heal completely after 3 to 6 months.
• Secondary lesions appear in crops from weeks to months later, measure 1 to 5 cm and tend to ulcerate or take the shape of raspberries (frambesoids), round or discoidal papillomas. [4]
• Osteoperiostitis may be evident at this stage [5] ; however systemic symptoms are usually not present
• The secondary stage may persist for up to 6 months, and relapse over periods as long as 10 years. [6]
• The third stage is characterized by destructive necrotic and gummatous lesions of skin, bone, nasopharynx and contiguous structures.

This disease is endemic or potentially endemic to 67 countries.

Yaws in Brazil

Time and Place:
Brazil instituted a Yaws-elimination program during 1956 to 1961. [7]
Most cases of yaws have been reported in the following regions: [8]
- North: inhabited areas of the Amazon
- Northeast: mountain ranges of Meruoca, Uruburetama, Baturite, Araripe and Serra Grande (Ceara); and Zona da Mata (Paraiba, Pernambuco and Alagoas)
- East: northeastern Minas Gerais, southern Bahia and northern Espirito Santo; and Baixada Fluminense, Rio de Janeiro
- Midwest: Goaiz and Mato Grosso, and territory of Guapore.

350,000 cases were estimated in 1953 (0.6% of the population) - with highest rates in the north, north-east and east. [9]
- 530,000 cases were estimated in 1956. [10]

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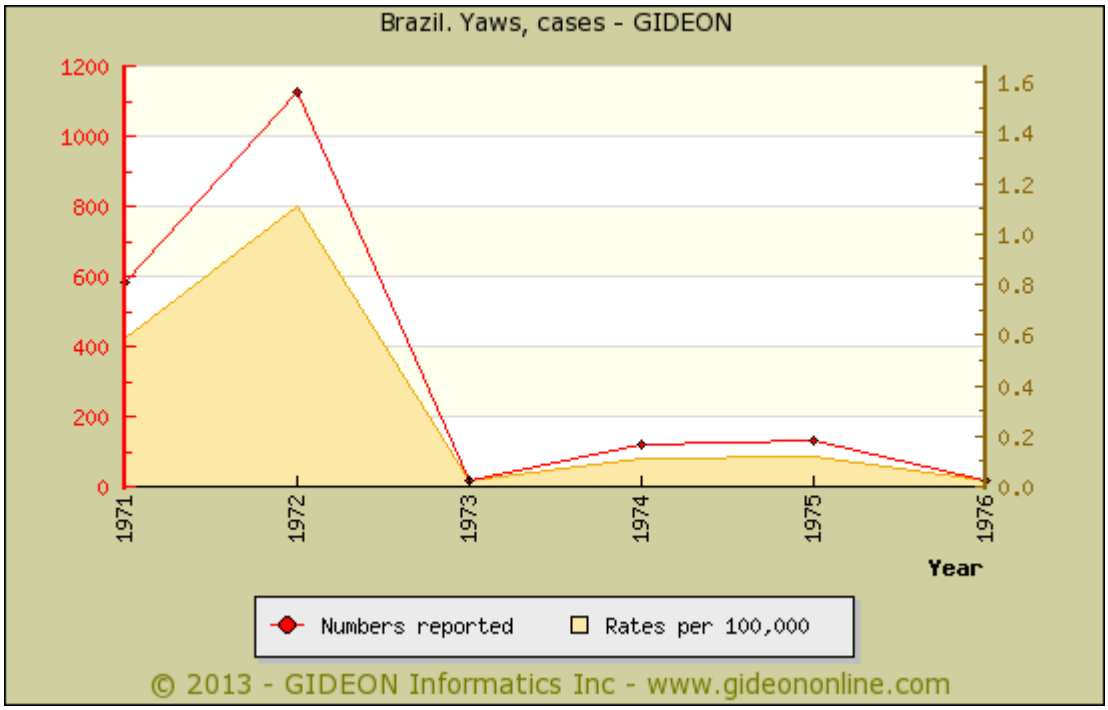

Graph: Brazil. Yaws, cases

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Yellow fever

Agent	VIRUS - RNA. Flaviridae, Flavivirus: Yellow fever virus
Reservoir	Human Mosquito Monkey ? Marsupial
Vector	Mosquito - Stegomyia (Aedes), Haemagogus, Sabethes
Vehicle	None
Incubation Period	3d - 6d (range 2.5d - 14d)
Diagnostic Tests	Viral culture (blood, liver). Serology. Nucleic acid amplification. Biosafety level 3.
Typical Adult Therapy	Supportive
Typical Pediatric Therapy	As for adult
Vaccine	Yellow fever
Clinical Hints	Headache, backache, vomiting, myalgias, jaundice, hemorrhagic diathesis, relative bradycardia and leukopenia; illness is often biphasic; 10% to 60% die within 7 days of onset.
Synonyms	Bulan fever, Febbre gialla, Febre amarela, Fever of Fernando Po, Fever of the blight of Benin, Fiebre amarilla, Fievre jaune, Gelbfieber, Gele koorts, Gul feber, Gula febern, Inflammatory fever, Kendal's disease, Magdalena fever, Maladie de Siam, Pest of Havana, Stranger's fever. ICD9: 060 ICD10: A95

Clinical

WHO Case definition for surveillance: [1]
Clinical description
• Characterized by acute onset of fever followed by jaundice within 2 weeks of onset of first symptoms.
• Hemorrhagic manifestations and signs of renal failure may occur.
Laboratory criteria for diagnosis
• Isolation of yellow fever virus, or
• Presence of yellow fever specific IgM or a four-fold or greater rise in serum IgG levels in paired sera (acute and convalescent) or
• Positive post-mortem liver histopathology or detection of yellow fever antigen in tissues by immunohistochemistry or
• Detection of yellow fever virus genomic sequences in blood or organs by PCR
Case classification
• Suspected: A case that is compatible with the clinical description.
• Probable: A suspected case with presence of yellow fever IgM antibody (in the absence of vaccination within 30 days); or positive postmortem liver histopathology; or an epidemiological link to a confirmed case or outbreak.
• Confirmed: A probable case; and a fourfold or greater increased in antibody titers; or presence of yellow fever neutralization antibody; or detection of yellow fever virus, viral genome or antigen in blood or tissues.

The clinical presentation of yellow fever can range from a self-limited flu-like illness to overwhelming hemorrhagic fever, with a case-fatality rate of 50%. [2]
• As many as 50% of infections may be clinically inapparent.

Infection is heralded by abrupt onset of fever, headache, and myalgias associated with conjunctival injection, facial flushing, relative bradycardia (Faget's sign) and leukopenia. [3]
• Although most cases do not progress beyond this stage, a remission of fever for a few hours to several days may be followed by high fever, headache, lumbosacral pain, nausea, vomiting, abdominal pain, and somnolence.
• At this stage, the patient exhibits icteric hepatitis and a hemorrhagic diathesis with prominent bleeding from the gastrointestinal tract, epistaxis, bleeding gums, and petechial and purpuric hemorrhages.
• Weakness, prostration, protracted vomiting and albuminuria are prominent.
• Deepening jaundice and elevations in serum transaminase levels continue for several days, accompanied by azotemia and progressive oliguria.
• Direct bilirubin levels rise to 5 to 10 mg/dl, while alkaline phosphatase levels are only slightly raised.
• Eventually, hypotension, shock, and metabolic acidosis develop, compounded by myocardial dysfunction and arrhythmias.
• Additional findings may include acute tubular necrosis, confusion, seizures, and coma.
• CSF examination reveals an elevated protein level without pleocytosis.
• Death usually occurs within 7 to 10 days after onset.

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This disease is endemic or potentially endemic to 47 countries.

Yellow fever in Brazil

Brazil is designated a COUNTRY WITH RISK OF YELLOW FEVER VIRUS TRANSMISSION.

Time and Place:
Brazil is divided into three areas regarding yellow fever: [4]
- the endemic area, with 12 states, mostly in the Northern (Amazon) and West-Central regions;
- the transition or epizootic or buffer zone between the endemic and non-endemic areas
- the non-endemic area, where there is no YF transmission

As of 1997 risk existed in almost two-thirds of the country.
Sylvatic yellow fever outbreaks follow a seven year cycle in the central-western region and a 14 year cycle for the northern region. [5]

Yellow fever is currently or recently reported in:
Amapa State:
- Boca de Acre Municipio
- Itacoatira Municipio
- Macapa Municipio
- Nova Airao Municipio

Amazonas State:
- Careiro Municipio
- Manacapuru
- Pres. Figueiredo Municipio

Bahia State
Coribe
Espiritu Santo State
Goias State:
- Alta Paraiso Municipio
- Amarlina
- Avelinopolis
- Bonopolis de Goias
- Cavalcante Municipio
- Corumbaia Municipio
- Doverlandia Municipio
- Glores de Goias
- Goias Velho Municipio
- Itaguaru
- Jaragua
- Jussara
- Mara Roas
- Mimosa de Goias
- Minacu Municipio
- Niquelandia Municipio
- Nova Roma
- Novo Brazil
- Padre Bernardo
- Planaltina de Goias
- Santa Fe de Goias
- Sao Joao da Alianca Municipio
- Terezina de Goias
- Uruacu
- Uruana
- Vila Propicio

Maranhao State:
- Batra do Corda Municipio
- Mirador Municipio

Mato Grosso State:
- Barra do Garca Municipio
- Campinapolis Municipio
- Diamantino
- Marcolandia Municipio
- Nobre
- Nova Canaa do Norte
- Nova Guarita Municipio

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- Nova Mutum
- Peixoto de Azevedo Municipio

Minas Gerais State
- Bom Despacho
- Conceicau do Para
- Leandro Ferreira
- Luz
- Martinho Campos
- Nova Serrana
- Natalandia
- Planura
- Santo Antonio do Monte
- Sao Gotardo

Para State:
- Afua Municipio
- Agua Azul do Norte Municipio
- Alenquer Municipio
- Bannach Municipio
- Breves Municipio
- Conceicao do Araguaia Municipio
- Foresta Municipio
- Gurupa Municipio
- Itaituba Municipio
- Obidos Municipio
- Parauapebas Municipio
- Rendencao Municipio
- Santa Maria de Barreira Municipio
- Sao Felix do Xingu Municipio
- Tucuma Municipio

Rio Grande do Sul State

Roraima State:
- Alto Alegre Municipio
- Canta Municipio
- Mucajai Municipio
- Uiramuta Municipio

Sao Paulo State
- Oureste
- Santa Albertina

Santa Catarina State (western and southern region)

Tocantins State
- Almas
- Ananas Municipio
- Arraias Municipio
- Aurora do Tocantins
- Campos Lindos
- Figueiropolis Municipio
- Guarai Municipio
- Parana Municipio
- Pedro Afondo Municipio
- Recursolandia Municipio

As of 2009, the southern, coastal area of Bahia state and the northern, coastal area of Espiritu Santo are no longer considered risk areas for yellow fever transmission, while the yellow fever risk areas have expanded in the states of Sao Paulo and Parana.

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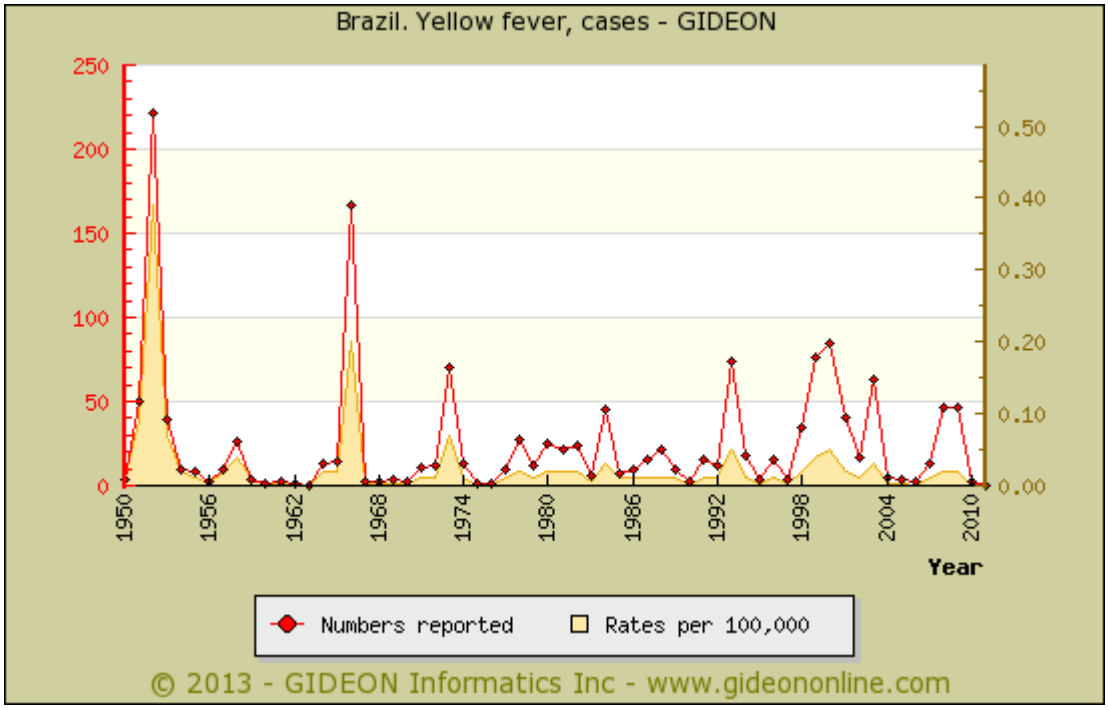

Graph: Brazil. Yellow fever, cases

Notes:
1. 4,000 fatal cases were reported in Rio de Janeiro in 1850.
2. 23,000 fatal cases were registered in Rio de Janeiro during 1851 to 1883; 5,000 in 1894; 314 in 1900; 80 in 1904.
3. The last cases of urban yellow fever were reported in 1942. [6]
4. 898 cases were reported during 1930 to 1992, (including 150 during 1981 to 1988) - 80.1% of these from Para, Goias and Mato Grosso; and 1.6% from Maranhao.
5. 537 cases were reported during 1980 to 2000; 251 (111 fatal) during 1998 to 2002. [7]
6. A total of 376 cases (216 fatal) of sylvatic yellow fever were reported during 1980 to 1998.
7. Parana reported 32 fatal cases in 1966; 0 cases during 1967 to 2007; 2 (1 fatal) in 2008. [8]
8. A review of yellow fever in Brazil during 1954 to 1999 - see reference [9]
9. 46 cases (27 fatal) were confirmed by WHO in 2008, 42 (11 fatal) in 2009. [10]
Individual years:
1993 - Cases from Maranhao (including 62 cases in Mirador), Amazonas and Para States.
1998 - 19 of these from Afua Municipality (Para State), with additional cases in Amapa, Amazonas, Mato Grosso and Roraima States.
2000 - Most in Goias State; with cases in Sao Paolo State (the first in this area since 1953) and Bahia State (the first since 1948).
2001 - Included 20 cases in Minas Gerais State (Bom Despacho, Conceicao do Para, Leandro Ferreira, Luz, Martinho Campos, Nova Serrana, Santo Antonio do Monte and Sao Gotardo).
2004 - From Parauapebas (Para State).
2006 - 83 suspect cases were reported during January to June. [11] Confirmed cases reported in Amazonas and Mato Grosso. [12]
2007 - 9 fatal cases were reported, from Amazonia, Para, Roraima, and Goias. [13] [14]
2008 - 2 fatal cases were reported in Sao Paulo State. [15]

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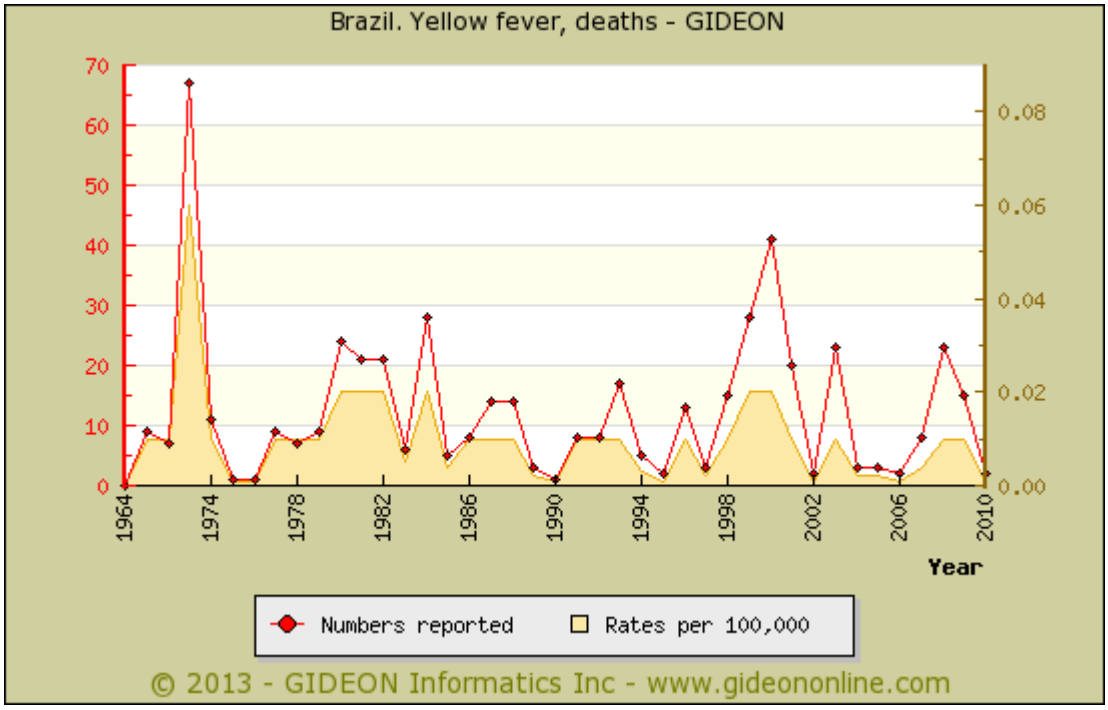

Graph: Brazil. Yellow fever, deaths

Exported cases:
1996 - Two travelers (Switzerland and United States) developed fatal yellow fever on return from Brazil. [16]
2002 - An American traveler died of yellow fever in Texas following return from Manaus. [17]

Vaccine Schedule:
BCG - birth; 6-10 years; [in contact with leprosy]
DT - 2 months; [conform to indications]
DTwP - 15 months; 4-6 years
DTwPHib - 2, 4, 6 months; Certain regions
DTwPHibHep - 2, 4, 6 months; Certain regions; [CRIES indications]
HepA - 1 year; [conform to indications]
HepB - birth; 1, 6 months
Hib - 2, 4, 6 months; [CRIES indications]
Influenza - 6 months; [in special cases and by campaigns for >= 60 years]
IPV - 2, 4 months; [CRIES indications]
MenC-conj - 2 months; [CRIES indications]
MMR - 1 year
MR - 12 years
OPV - 2, 4, 6 months
Pneumo-conj - 2 months; [CRIES indications]
Pneumo- ps - 2 years; [CRIES indications]
Rotavirus - 2,4 months
Td - 7 years
Varicella - 1 year; [CRIES indications]
Vitamin A - 6, 12, 18, 24, 30, 36, 42 months; Certain regions [puerperal]
Yellow fever - 9 months; 10 years

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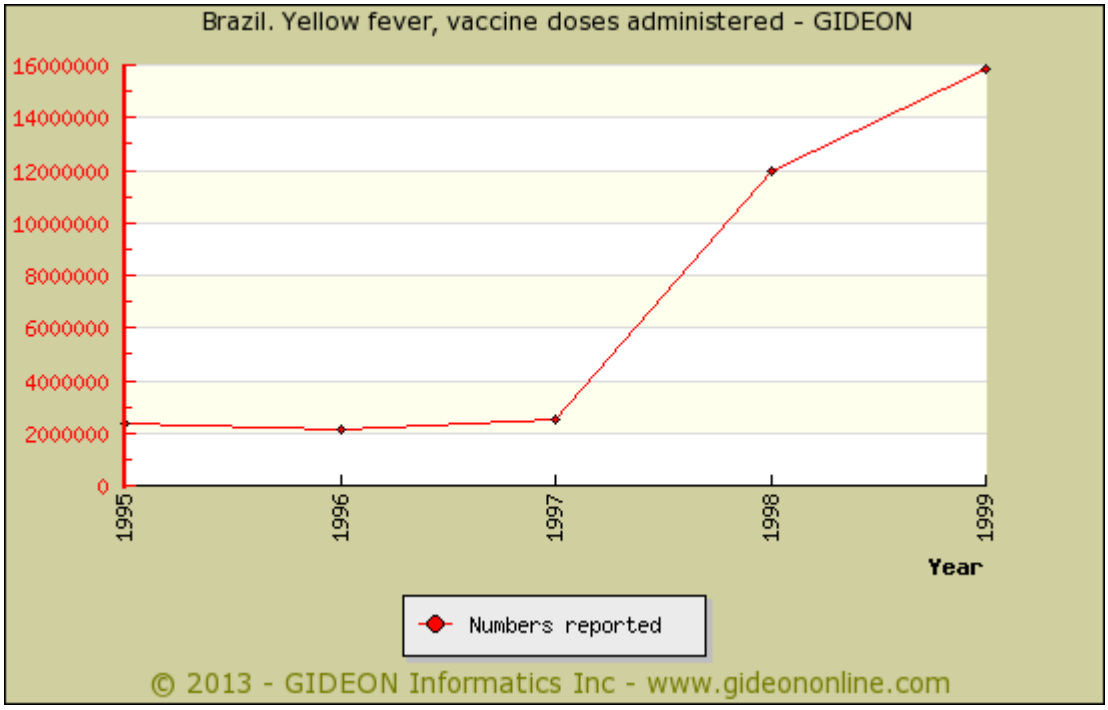
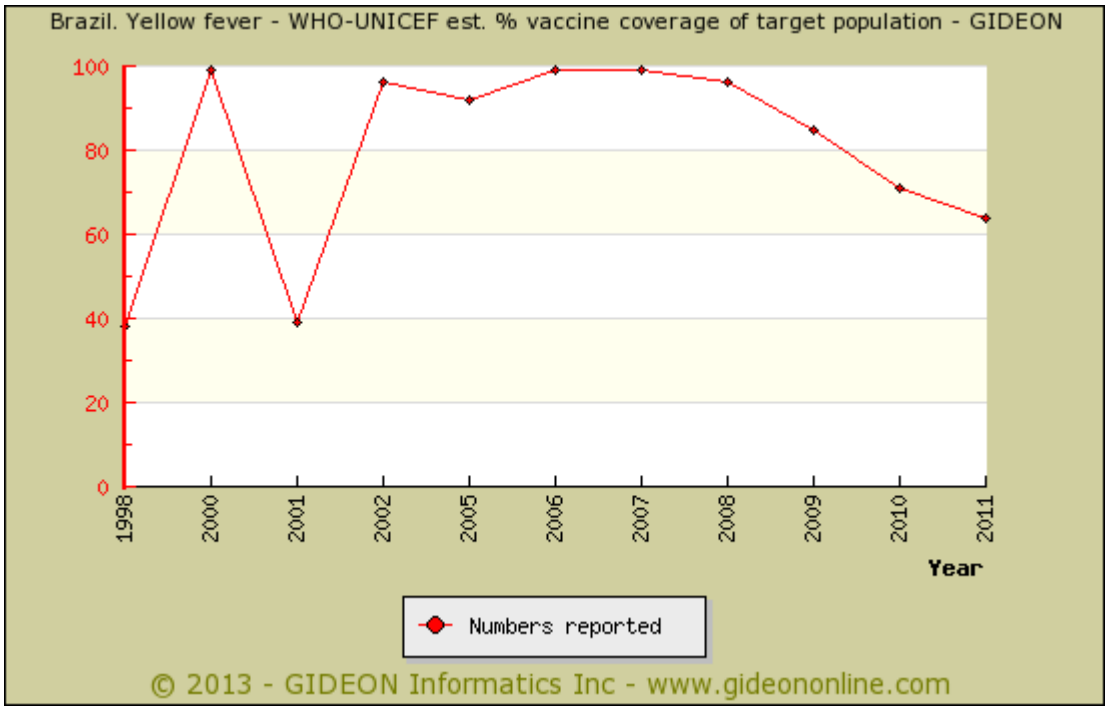


Graph: Brazil. Yellow fever, vaccine doses administered

Notes:
1. 8 million were vaccinated annually during 1986 to 1988.



Graph: Brazil. Yellow fever - WHO-UNICEF est. % vaccine coverage of target population

Notes:
1. A survey showed that vaccine coverage by 12 months of age was 79%, 86% by age 18 months (27 capital cities, 2010 publication) [18]

Proof of vaccination is **NOT** required for travelers entering Brazil.
- CDC (The United States Centers for Disease Control) For all travelers >=9 months of age going to the following areas at

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risk for yellow fever transmission, including the ENTIRE states of Acre, Amapa, Amazonas, Distrito Federal (including the capital city of Brasilia), Goias, Maranhao, Mato Grosso, Mato Grosso do Sul, Minas Gerais, Para, Rondonia, Roraima, Tocantins, and designated areas of the following states: northwest and west Bahia, central and west Parana, southwest Piaui, northwest and west central Rio Grande do Sul, far west Santa Catarina, and north and west Sao Paulo. Vaccination is recommended for travelers visiting Iguacu Falls.
- Note that the Federal District of Brasilia is also an endemic area.
- Coastal cities, including Rio de Janeiro, Sao Paulo, Salvador, Recife, and Fortaleza, are NOT within the endemic zone.

Yellow fever in monkeys:
- *Alouatta fusca* is the most likely species, although *A. fusca* and *A. caraya* are sympatric from Northern Argentina to the Western region of Parana.
2000 - An outbreak of yellow fever was reported among monkeys in the Grande river Parana basin of northwestern region of Sao Paulo state [19]
2001 - Over 30 cases were confirmed among howler monkeys (*Alouatta* spp.) in the municipalities of Santo Antonio das Missoes and Garruchos (Rio Grande do Sul), in association with infected mosquitoes (*Haemagogus leucocelaenus*). [20] [21]
2004 - An epizootic was reported in Goias and Tocantins, involving the municipalities of Sao Miguel do Passa Quatro, Goianapolis, Terezopolis de Goias, Minacu, and Santa Cruz do Goias. Affected species included howler monkeys (*Alouatta* spp.) and *Cebus apella*.
2006 - An outbreak of yellow fever was reported among monkeys in Rio Grande do Sul. [22]
2007 - Epizootics were notified in 48 municipalities in 9 states: Goias, Tocantins, Minas Gerais, Mato Grosso, South Mato Grosso, South Rio Grande, Piaui, the Federal District, and North Rio Grande Epizootics in monkeys were notified in 48 municipalities in 9 states: Goias [23] , Tocantins, Minas Gerais, Mato Grosso, South Mato Grosso, South Rio Grande, Piaui, the Federal District, and North Rio Grande. [24]
2008 to 2009 - Yellow fever was confirmed in 18 dead monkeys in Sao Paulo State [25-29] , 7 in Mato Grosso State [30] , 1 in Bahia. [31] and 299 in Rio Grande do Sul. [32-37]
2008 to 2009 - An outbreak was reported among howler monkeys (*Alouatta caraya*) in Rio Grande do Sul. [38]
2009 - An outbreak (8 deaths) of suspected yellow fever was reported among primates in Parana State [39] [40] ; and an infected monkey was identified in Sao Paulo State. [41]
2011 - An outbreak (11 deaths) of suspected yellow fever was reported among monkeys in Mato Grosso State. [42]
2012 - Two monkeys in Rio Grande do Sul died of yellow fever. [43]

Vectors:
- The vector for human infection is *Aedes aegypti*
- During 5 outbreaks of sylvatic yellow fever, the following mosquito species were implicated: *Haemagogus janthinomys* [44] , *Hg. submaculatus*, *Sabethes chloropterus* and *Sa. soperi*.
- *Haemagogus (Conopostegus) leucocelaenus* was implicated in zoonotic transmission during an outbreak in Rio Grande do Sul State (2001). [45]
- *Haemagogus janthinomys* has been identified in Pernambuco [46]
- *Haemagogus leucocelaenus* and *Aedes serratus* were active during an epizootic in Rio Grande do Sul during 2008. [47]
- Yellow fever virus was identified in *Haemagogus (Conopostegus) leucocelaenus* in Sao Paulo State (2011 publication) [48]
- A single case of vertical transmission was reported in 2009. [49]
- Transmission of vaccine virus through breast feeding has been reported. [50] [51]

Notable outbreaks:
1849 to 1850 - An outbreak of yellow fever was reported in Rio de Janeiro. [52]
1896 to 1902 - Outbreaks (3 outbreaks) were reported in Sao Simao - in 1896, 1898, and 1902. [53]
1903 - An outbreak was reported in Ribeirao Preto occurring in 1903.
1952 to 1953 - An outbreak of yellow fever was reported in Alta Sorocabana. [54]
1972 to 1973 - An outbreak (71 cases, 44 fatal) was reported in Goias State. [55-57]
1993 to 1994 - An outbreak (70 cases) was reported in Maranhao State. [58]
2000 - An outbreak (85 cases, 41 fatal) was reported. [59]
2001 - An outbreak was reported in Minas Gerais State. [60] [61]
2002 to 2003 - An outbreak (84 cases, 62 confirmed, 23 fatal) was reported in Minas Gerais State. [62]
2007 to 2008 - An outbreak (61 cases, 35 confirmed, 21 fatal) was reported, including 21 cases in Goias, 7 in Mato Grosso do Sul, 5 in Federal District, 2 (fatal) in Sao Paulo [63] [64] and 6 in Mato Grosso. [65-75] 45 cases (25 fatal) were reported to July 2008, in Sao Paulo and west Parana states. [76]
2008 to 2009 - Outbreaks of yellow fever were reported in Sao Paulo (28 cases, 11 fatal) [77-83] and Rio Grande do Sul (18 cases, 7 fatal) [84-92]]

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Yersiniosis

Agent	BACTERIUM. Yersinia enterocolitica and Yersinia pseudotuberculosis A facultative gram-negative bacillus
Reservoir	Pig Rodent Rabbit Sheep Goat Cattle Horse Dog Cat Bat
Vector	None
Vehicle	Food Water Meat Dairy products Vegetables Fecal-oral Blood
Incubation Period	4d - 7d (range 1d - 11d)
Diagnostic Tests	Culture stool, blood. Alert laboratory when these organisms are suspected.
Typical Adult Therapy	Stool precautions; diarrhea is self-limited. If severe disease - Ciprofloxacin 500 mg BID X 5 to 7d. OR Sulfamethoxazole/trimethoprim
Typical Pediatric Therapy	Stool precautions; diarrhea is self-limited. If severe disease - Sulfamethoxazole/trimethoprim 20 mg-4 mg/kg BID X 5 to 7d
Clinical Hints	Fever, diarrhea, right lower quadrant pain; fecal leucocytes present; may be associated with rheumatologic manifestations such as erythema multiforme, Reiter's syndrome and chronic arthritis.
Synonyms	Yersinia enterocolitica, Yersinia pseudotuberculosis, Yersiniose. ICD9: 008.44 ICD10: A04.6,A28.2

Clinical

Yersinia enterocolitica infection typically presents as febrile diarrhea, and occasionally bloody diarrhea.

• Lower abdominal pain without diarrhea occurs in over 15% of cases, and may mimic acute appendicitis. [1-5] Several instances of intestinal intussusception have been associated with *Yersinia enterocolitica* [6-15] and *Y. pseudotuberculosis* infections. [16]

• Pharyngitis [17] and tonsillitis [18] are encountered ; and metastatic infection of bone, soft tissues, spleen, meninges [19] or other organs may occur. [20-23]

• Chronic arthritis, erythema nodosum, Reiter's syndrome [24] , Sweet's syndrome [25] , glomerulonephritis [26-32] , pneumonia [33] and endocarditis [34-47] are also reported.

• Reactive arthritis has been reported in over 20% of cases [48] [49]

• *Yersinia enterocolitica* septicemia (associated with transfusion of contaminated red blood cell products) is fatal in over 50% of cases. [50]

Yersinia enterocolitica is one of at least a dozen *Yersinia* species encountered in humans. See the Microbiology module for further details.

This disease is endemic or potentially endemic to all countries.

Yersiniosis in Brazil

Prevalence surveys:

0% of urban children with diarrhea in Salvador (2000 to 2002) [51]

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Zygomycosis

Agent	FUNGUS. Zygomycota, Zygomycetes, Mucorales: Mucor spp., Rhizopus spp., Lichtheimia (formerly Absidia) spp, Saksenaea spp, et al
Reservoir	Saprophytes
Vector	None
Vehicle	Air Bandages Contact
Incubation Period	Variable
Diagnostic Tests	Fungal smear and culture.
Typical Adult Therapy	Amphotericin B to maximum dose 0.8 mg/kg/d; and to total dose of 3g. Excision as indicated
Typical Pediatric Therapy	Amphotericin B max dose 0.8 mg/kg/d; and to total dose of 40 mg/kg. Excision as indicated
Clinical Hints	Periorbital pain, sinusitis, and palatal, nasal or cerebral infarcts; occurs in the setting of preexisting acidosis (diabetes, uremia); pulmonary infection may complicate leukemia.
Synonyms	Absidia, Actinomucor, Apophysomyces, Cokeromyces, Cunninghamella, Hormographiella, Lichtheimia, Lichtheimia, Mucor, Mucormycosis, Mycocladus, Phycomycosis, Rhizomucor, Rhizopus, Saksenaea, Syncephalastrum. ICD9: 117.7 ICD10: B46

Clinical

Infection is most commonly associated with hyperglycemia, metabolic (diabetic, uremic) acidosis, corticosteroid therapy and neutropenia [1] , transplantation, heroin injection or administration of desferoxamine. [2]
- Major risk factors identified in children are neutropenia, diabetes mellitus, and prematurity. [3]
- Virtually any organ can be involved [4-7] ; however, most infections involve the paranasal sinuses and contiguous structures (orbit, cavernous sinus, cranial nerves, cerebral arteries), lungs, skin [8] [9] and gastrointestinal tract. [10]

Disease manifestations reflect the mode of transmission, with rhinocerebral and pulmonary diseases being most common. [11]
- Cutaneous [12] , gastrointestinal, and allergic diseases are also seen.
- The Mucorales are associated with blood vessel invasion, often leading to thrombosis, infarction and tissue destruction.
- Rare cases of sinusitis have been ascribed to *Actinomucor elegans*.
- Dissemination is common.
- Therapy must be started early and consists of antifungal drugs, surgical intervention, and reversal

Rhinocerebral zygomycosis initially manifests with headache (often unilateral), fever, facial pain, diplopia, lacrimation, and nasal stuffiness.
- As the infection spreads, necrotic lesions appear in the turbinates, nose, paranasal skin or hard palate. [13]
- Rare cases of mycotic mandibular osteomyelitis have been reported.
- Chemosis, proptosis, and external ophthalmoplegia may occur.
- Cranial nerve abnormalities are common (nerves II through VII, IX, and X) [14] , and blindness may ensue following invasion of the cavernous sinus, ophthalmic artery, and orbit.
- Hemiparesis, seizures, or monocular blindness suggest advanced disease.
- Invasion of the internal carotid artery in the cavernous sinus can occur, with metastatic lesions in the frontoparietal cortex and deepening coma.

Pulmonary zygomycosis presents with nonspecific symptoms such as fever, cough and dyspnea. [15]
- Hemoptysis may occur with vascular invasion.
- Radiological findings include segmental consolidation which progresses to contiguous areas of the lung and may cavitate.
- In 74% of pulmonary zygomycosis cases, the infection is limited to the lung. [16]

Gastrointestinal zygomycosis usually affects patients with severe malnutrition, and may involve the stomach, ileum, colon [17] or peritoneum. [18]
- Clinical findings mimic intra-abdominal abscess.
- The diagnosis is often made at autopsy.

Cutaneous zygomycosis may present as primary infection, characterized by necrotic lesions following trauma; or

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secondary extension from a focus of rhinocerebral infection. [19] [20]

Renal zygomycosis may mimic malignancy [21-23]

59 case reports (38 fatal) of neonatal zygomycosis had been published to July 2007 • 77% premature infants, 54% gastrointestinal and 36% dermal. [24]

Zygomycosis has a poor prognosis, with a mortality rate of 44%. [25]

This disease is endemic or potentially endemic to all countries.

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