American Trypanosomiasis

Chagas Disease,
New World Trypanosomiasis,
South American Trypanosomiasis,
Mal de Chagas,
Chagas-Mazza Disease

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Importance

American trypanosomiasis (Chagas disease) is an important cause of heart disease, megaesophagus and megacolon among people in Mexico, Central and South America. Many mammals can be infected with the parasite that causes this disease; however, among animals, clinical cases have been reported mainly in dogs. Chagas disease is transmitted by the bites of triatomine insects, or “kissing bugs.” Some infected insects occur in sylvatic environments, where humans are exposed only occasionally. These “sylvatic cycles” are found from the U.S. through South America. From Mexico through South America, triatomine insects have also become adapted to human dwellings, particularly substandard housing where the insects hide in cracks during the day and emerge to feed on humans and animals at night. Most human cases are acquired from insects in these “domestic cycles,” and campaigns to eliminate the bugs, together with testing to prevent congenital cases and transmission in blood transfusions, have significantly reduced the incidence of Chagas disease. Antiparasitic treatment is most effective early, before irreversible damage occurs to the heart or gastrointestinal tract.

Etiology

Chagas disease results from infection with the protozoan parasite Trypanosoma cruzi, a member of the family Trypanosomatidae. Most strains of this parasite can be classified into two major groups, T. cruzi I and T. cruzi II, which can be separated further into various lineages (e.g., T. cruzi Ila). Lineages tend to be associated with certain host species, although this relationship is not absolute.

Geographic Distribution

T. cruzi can be found in the Americas from the U.S. to Chile and central Argentina. In the U.S., this parasite is thought to be endemic in approximately the southern half of the country, as well as in California.

Transmission and Life Cycle

Chagas disease is a vector-borne disease transmitted primarily by triatomine insects, which are also called reduviid insects, “kissing beetles/ bugs” or “assassin bugs.” More than 130 species of these insects appear to be capable of transmitting T. cruzi, with the most important species in the genera Triatoma, Rhodnius and Panstrongylus. The parasite usually completes its life cycle by cycling between an insect species and a mammalian species with which the insect lives in close association. The mammalian hosts include wildlife, domesticated animals and humans.

In its mammalian host, T. cruzi can be found in the blood as trypomastigotes (extracellular nondividing forms) and in cells as amastigotes (replicative forms). When an insect takes a blood meal from an infected mammal, it ingests the trypomastigotes. After 2 to 4 weeks of development, some of the parasites migrate to the insect’s hindgut, where they are transformed into the infective metacyclic trypomastigotes. The insect defecates after it feeds, releasing the trypanosomatid in its feces. These parasites can enter the mammal’s body through mucous membranes or breaks in the skin. Scratching may inoculate trypomastigotes into the bite wound, or allow the parasites to enter through the scratches. Triatomine insect species that exhibit delayed defecation after feeding are less likely to transmit Chagas disease than species that defecate on the host. Humans and animals can also be infected if they eat either the insect or uncooked food that contains insect feces.

There are three basic cycles of transmission for T. cruzi. In the sylvatic (wild) cycle, this organism cycles between wildlife and triatomine insects that live in sylvatic environments. Humans and domesticated animals are infected occasionally when they contact these bugs in the wild. Under some conditions, the insects may also invade houses or outbuildings when they are attracted to light, heat or certain odors, and may contaminate food. Wild triatomine insects can also be transported accidentally to human houses. The sylvatic cycle is responsible for relatively few cases of Chagas disease. It is the only cycle in the U.S. A domestic transmission cycle
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also exists in Mexico and parts of Central and South America. In this cycle, some insect vectors have colonized primitive adobe, grass and thatched houses, resulting in transmission between humans and insects. Important species in the domestic cycle include *Triatoma infestans*, *T. dimidiata* and *Rhodnius prolixus*. Transmission cycles between insects and domesticated animals (peridomestic cycles) can also provide opportunities for the parasite to infect humans.

*T. cruzi* is not spread between mammals by casual contact; however, it can be transmitted directly via blood (e.g., in a blood transfusion) and in donated organs. Carnivores can acquire this organism when they eat infected prey. Vertical transmission has been reported in dogs and other animals, both *in utero* and in the milk. Transmission in milk is very rare in humans, but transplacental transmission can occur at each pregnancy, and during all stages of infection. Laboratory infections usually occur when the parasites contact mucous membranes or broken skin, or are accidentally injected via needlestick injuries, but aerosol transmission might be possible in this setting.

**Disinfection**

*T. cruzi* is an obligate intracellular parasite. It is destroyed by several hours of exposure to direct sunlight or other harsh environments. *T. cruzi* is susceptible to many disinfectants including 1% sodium hypochlorite, 70% ethanol, iodine/alcohol solutions, glutaraldehyde and formaldehyde. It can be inactivated by moist heat (121°C for a minimum of 15 min) or dry heat (160 to 170°C for a minimum of an hour).

**Infections in Humans**

**Incubation Period**

The incubation period is usually at least 5 to 14 days after exposure to triatomine insect feces, and 20 to 40 days after infection by blood transfusion. Many people do not become symptomatic until the chronic stage, which can occur 5 to 40 years after infection.

**Clinical Signs**

The acute phase is defined as the period during which the parasites can be found easily in the blood. Many people, particularly adults, are asymptomatic during this stage. The symptoms of the acute phase are highly variable and may include fever, headache, anorexia, malaise, myalgia, joint pain, weakness, nausea, vomiting, diarrhea, hepatomegaly, splenomegaly, and generalized or localized lymphadenopathy. Edema, either generalized or localized to the face and/or lower extremities, occurs in some cases. Sometimes, a chagoma (a localized painless induration) is seen where the parasite has entered through the skin. If entry occurs via the ocular mucous membranes, there may be painless edema of one or occasionally both eyes, often accompanied by conjunctivitis and enlargement of the local lymph nodes. This syndrome, which is called Romaña’s sign, usually persists for 1 to 2 months. Patients occasionally develop a rash, but this usually disappears within several days. In most cases, the clinical signs resolve within weeks to months without treatment; however, some acute cases can be fatal. Deaths are most likely to occur in young children and patients who are immunosuppressed (e.g., HIV-infected individuals), who may develop acute myocarditis or meningoencephalitis.

The acute phase is usually followed by an asymptomatic period of varying length; this stage is called the indeterminate phase. During the indeterminate phase, the parasites disappear from the blood. Although estimates vary, approximately 70% to 90% of the patients in the indeterminate phase never become symptomatic. Most of the remaining patients enter the chronic phase after 5 to 15 years, but in a few patients, the indeterminate phase can last as long as 40 years.

The chronic phase is typically represented by organ failure, usually of the heart or digestive system. Heart disease, the most common form of chronic Chagas disease, may be characterized by arrhythmias and conduction abnormalities, cardiac failure, apical aneurisms, embolic disease including stroke or pulmonary embolism, and sudden death. Signs of isolated left heart failure may occur first. Biventricular congestive heart failure with peripheral edema, hepatomegaly, pulmonary congestion and dyspnea is common in the later stages. Some patients develop chest pains without evidence of coronary artery disease.

Digestive system abnormalities lead to megaesophagus and/or megacolon, which can occur concurrently with heart disease. The symptoms of megaesophagus may include pain during swallowing, excessive salivation, regurgitation and chest pain. Dysphagia mainly occurs with dry, solid and cold food. Aspiration pneumonitis can be a sequela and there is an increased risk for cancer of the esophagus. In severe cases, there may be weight loss or cachexia, and the esophagus may rupture. The symptoms of megacolon include severe constipation, which can last for a few days to months, and abdominal pain that is often associated with episodes of constipation. The abdomen may be asymmetrically distended. Complications may include intestinal occlusion, volvulus, fecalomas, ulceration, or intestinal perforation with peritonitis. Patients with Chagas disease also have an increased chance of developing gastric ulcers or chronic gastritis, due to abnormalities in the stomach. Gall bladder abnormalities may include mega-gallbladder or cholelithiasis. Small intestinal dilatation is rare.

Women who are infected with *T. cruzi* can give birth to infected children. Congenital infections may occur during any of the woman’s pregnancies, whether she is symptomatic or not. In congenitally infected infants, the most common symptoms are prematurity birth, hepatosplenomegaly, meningoencephalitis, changes in the
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retina and signs of acute myocarditis/ cardiac insufficiency. Transplacental infections are also associated with abortions.

Patients with AIDS suffer a more severe form of the disease with a high percentage of neurological and cardiac signs. Many of these patients develop T. cruzi brain abscesses, which are not seen in immunocompetent patients. HIV-infected individuals and others who are immunosuppressed, including those who receive organ transplants, are at risk for reactivation of T. cruzi replication.

Communicability

Chagas disease is not spread from person to person by casual contact, but infected humans can transmit the parasite to the insect vectors. Congenital infections occur in 1-10% of infants born to infected mothers, and this may occur at any stage of the disease. Transmission through the milk is very rare. T. cruzi can also be spread in tissue transplants or blood transfusions.

Diagnostic Tests

Chagas disease can be diagnosed by microscopy, isolation of the parasite, serology or molecular techniques.

**Microscopic examination**

Light microscopy can sometimes detect *T. cruzi* in Giemsa or Wright stained samples of blood, cerebrospinal fluid or tissues. *T. cruzi* can be found in the heart, skeletal and smooth muscle cells, and the glial cells of the nervous system. It sometimes occurs in chagomas. In immuno-compromised patients, parasites may also be detected in atypical sites such as the pericardial fluid, bone marrow, brain, skin and lymph nodes. Active parasitemia is much more likely to be found during the acute than the chronic stage. Either thick or thin blood smears may be examined, but parasite morphology is clearer in thin smears. Blood concentration techniques can increase the probability of finding the parasites; examination of the buffy coat is often used. *T. cruzi* can be difficult to distinguish from *Trypanosoma rangeli*, which is avirulent.

**Isolation of the agent**

*T. cruzi* can be cultured from heparinized blood samples or tissues. Various specialized media including liver infusion tryptose medium or Novy-MacNeal-Nicolle medium, as well as Vero cell lines can be used. Culture may take 1 to 6 months. The agent can also be isolated by inoculating the blood into a guinea pig, mouse or rat; this procedure is often successful in chronic cases. Xenodiagnosis is considered to be the gold standard where it is available. In xenodiagnosis, laboratory-reared, *T. cruzi*-free triatomine insects are allowed to feed on an infected individual; the parasites may be found in the intestinal contents of the insect 1 to 2 months later. Because these methods of diagnosis are slow and labor-intensive, and require highly trained personnel, they are generally used only to confirm a diagnosis if other tests are inconclusive, in research, or to isolate strains of parasites.

**Serology**

Serology is most often used to diagnose chronic infections. Commonly used serological tests in humans include indirect immunofluorescence (IFA), hemagglutination and enzyme-linked immunosorbent assays (ELISAs). Other tests including radioimmunoprecipitation and complement fixation may also be used. Cross-reactions can occur with other parasites, particularly *Leishmania* species.

**Molecular techniques**

Polymerase chain reaction (PCR) techniques can be used for diagnosis. Immunoblotting (Western blotting) is another option.

**Treatment**

Acute Chagas disease can be treated with antiparasitic drugs. In the U.S., drugs may be available only under an Investigational New Drug protocol from the CDC Drug Service. Treatment of acute or congenital cases is recommended to prevent the development of chronic disease. Antiparasitic drugs are less effective in the indeterminate and chronic stages, and treatment recommendations may vary with the age of the patient and other factors. There are significant side effects with these drugs, which must be given long term.

In the chronic stage, treatment of cardiomyopathy is mainly symptomatic and similar to the treatment of other causes of heart disease. A pacemaker may be necessary, and a heart transplant can be considered. Surgery, balloon dilation of the gastroesophageal junction and/or symptomatic relief may be used for chagasic megaesophagus or megacolon.

**Prevention**

Vaccines are not available for humans; however, precautions can be taken to reduce the risk of infection, particularly in countries where the prevalence of Chagas disease is high. Triatomine insects usually feed at night and withdraw to their hiding places in daylight. In endemic areas, houses can be improved by plastering walls, improving flooring and taking other measures to remove the cracks where these insects hide. Triatomine insects are often found in basements, which should be avoided. Sleeping inside a screened area, under a permethrin-impregnated bed net, or in an air-conditioned room is safest. Bed nets should be tucked tightly under the mattress before dusk. Animal pens and storage areas should be kept away from homes. Regular spraying of insecticides in and around houses can reduce the number of insects, and in some cases, eliminate them.

Some triatomine bugs such as Triatoma infestans and Rhodnius prolixus have adapted to human dwellings, and do not exist in the wild. These insects can be eliminated completely by control measures, greatly reducing the risk of Chagas disease. Since 1991, the Pan American Health
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Organization and the World Health Organization have run a joint program to eliminate T. infestans, the most important vector for Chagas disease in humans. This program has decreased the distribution of this insect by more than 80%, although foci can still be found in some regions. Triatomine species that occur in sylvatic cycles but colonize human dwellings can also be controlled or eliminated by insecticides and other measures, but they may recolonize homes. Foods that might be contaminated should be cooked.

Blood and organ donors should be screened to prevent transmission by these routes. In the U.S., transfused blood has been screened for Chagas disease since 2007. Pregnant women can be tested to identify cases where congenital transmission may occur, and the infant should be monitored and treated if necessary.

People in occupational risk groups should take additional precautions. Veterinarians and technicians should protect their skin and mucous membranes from contamination with parasites in blood or tissues. This includes using gloves and/or other barriers while drawing blood samples from T. cruzi-infected animals, taking care of IV catheters or performing other invasive procedures. Needles and other “sharps” must be handled and disposed of properly to prevent needlestick injuries. Individuals who work with wildlife and hunters should also take precautions, especially when handling blood and tissues. Laboratory personnel should use appropriate personal protective equipment, including gloves and eye protection, while processing blood samples, cultures or infected insects. If an accidental exposure occurs, the site should be disinfected immediately if possible, and antiparasitic drugs may be given prophylactically.

Travelers to areas where Chagas disease is common should wear thick clothing that covers as much of the body as possible; heavy long-sleeved shirts, long pants, socks and shoes are recommended. Sleeping in sub-standard housing should, if possible, be avoided.

Morbidity and Mortality

Chagas disease is most common among people who live in substandard housing. Most cases are acquired by exposure to insects in domestic or peridomestic cycles, or by congenital transmission. In some areas, outbreaks have been linked to foods such as fresh fruit juices contaminated by sylvatic triatomines. In endemic areas, many people become infected in childhood. In South and Central America, campaigns to control the vectors in the domestic cycle have significantly decreased the number of new human infections. Triatomine vectors for T. cruzi are present in the U.S.; however, only a few locally acquired, vector-borne cases have been diagnosed in people. The lower prevalence rate in the U.S. is mainly due to higher standards of living and the absence of triatomine species that are well adapted to living in human houses. In addition, some of the insects involved in sylvatic transmission in the U.S. exhibit delayed defecation after feeding, which may reduce the risk of transmission.

The morbidity and mortality rates vary with the stage of the disease. Approximately 5% of people infected with T. cruzi develop acute symptoms. Estimates of the case fatality rate for acute Chagas disease range from less than 5% to approximately 8%; among immunologically competent individuals; deaths occur mainly in young children with acute myocarditis or meningoencephalitis. The CDC estimates that 20-30% of humans infected with T. cruzi eventually develop chronic disease; estimates from other sources vary from 10% to 50%. The reason for the progression of disease in some patients but not others is unknown. It may be related to host genetic factors, the dose of the parasites, the number of inoculations, the strain of the parasite, and immunological or nutritional factors. Cardiac disease is often fatal. Occasionally, deaths are also caused by volvulus of a dilated sigmoid megacolon.

Infections in Animals

Species Affected

Trypanosoma cruzi occurs in more than 100 species of mammals throughout the Americas; infections have been reported among carnivores including dogs and cats, as well as in pigs, goats, lagomorphs, rodents, marsupials, bats, xenarthra (anteaters, armadillos and sloths) and non-human primates. In the U.S., opossums, armadillos, raccoons, coyotes, rats, mice, squirrels, dogs and cats are among the most frequent hosts. Birds, reptiles and fish are not susceptible to infection.

Incubation Period

The incubation period for acute disease in dogs appears to be 5 to 42 days; in experimental infections, symptoms of acute heart disease are usually reported after 2 to 4 weeks. Like humans, some dogs may not develop clinical signs until the chronic stage, which occurs after a few years; the exact length of this period is not known.

Clinical Signs

Dogs

Acute, latent and chronic stages of infection occur in experimentally infected dogs. Clinical signs reported in the acute stage include fever, anorexia, lethargy, an unkempt hair coat, lymphadenopathy, hepatomegaly and splenomegaly. Anorexia, diarrhea, ascites and/or weight loss may also be seen. Some dogs may have palpable edema. Chagomas appear to be rare, but they have been reported in a few dogs inoculated with a South American strain of T. cruzi. Cardiac dysfunction can occur during the acute phase; acute myocarditis may cause arrhythmias or sudden collapse and death. After the acute phase, infected dogs enter the indeterminate (latent) stage, during which the
parasites are difficult to find and the animal is asymptomatic. The indeterminate stage can be as short as 27 days in some experimentally infected animals, but it seems to last for years in some natural infections. Congestive heart failure is the most common sign during the chronic stage. Right-sided heart failure usually occurs first. Eventually, dogs with heart disease develop chronic myocarditis with cardiac dilatation and arrhythmias. Sudden death can occur.

Limited information is available from naturally infected dogs. In one retrospective study from Texas, sudden death was the most common presentation in more than 500 confirmed cases. Other common clinical signs included anorexia, lethargy, ascites, an enlarged heart, cardiac conduction disturbances and respiratory difficulties. Hepatomegaly, anemia, diarrhea, vomiting and edema were reported less often. Heart disease was the most common presentation in both puppies and adults. In Mexico, only mild disease was reported in naturally infected stray dogs, although ECG changes were seen in some of these animals. A limiting factor in the latter study is that stray dogs would be expected to have a relatively short lifespan, and may not have lived long enough to develop more severe signs.

Atypical cases have also been reported in dogs. In one dog, the clinical signs included anorexia, lethargy, weight loss, generalized lymphadenopathy, fever and dependent, pitting edema, without cardiac signs or lesions. Neurologic signs such as limping or coma have occasionally been reported. One case of Chagas disease in a 13-month-old Doberman pinscher presented as slowly progressive paraparesis with temporal, supraspinatus and infraspinatus muscle atrophy, bilateral enophthalmos, superficial inguinal lymphadenopathy, tachycardia with pulse deficits, and chorioretinitis. In addition to neurologic abnormalities in the hindlegs and forelegs, this dog had cranial nerve deficits and a delayed gag reflex.

Experimentally infected dogs can develop histological abnormalities in the esophagus and stomach, but unlike humans, megaesophagus and megacolon have not been reported.

**Cats**

Symptomatic Chagas disease has been rarely described in cats. Reported clinical signs include fever, edema, weight loss and neurological signs such as convulsions and paresis.

**Other species**

There is little information about Chagas disease in other species, including wild animals. Mild, histologically evident myocarditis (without clinical signs of heart disease) has been reported at necropsy in infected wild raccoons and opossums. Naturally infected rats can develop arrhythmias and more severe cardiac lesions. Infertility, fetal losses, reduced birth weights, and early postnatal deaths have been reported in pregnant mice.

**Post Mortem Lesions**

In dogs, acute disease is characterized mainly by cardiac lesions, which are particularly prominent on the right side. The myocardium may be pale, and subendocardial and subepicardial hemorrhages are often present. Multiple yellowish-white spots and streaks, mainly involving the coronary groove, may be found in the heart. In addition, there may be secondary pulmonary edema, congestion in the liver, spleen and kidneys, and a modified transudate in the peritoneal cavity. Pericardial effusion has been reported in some naturally infected dogs. Gross lesions may be absent in some infected animals.

In chronic Chagas disease, the heart is bilaterally enlarged and flaccid, with thinning of areas in the ventricular walls. Atypical cases without cardiac lesions have also been reported.

Mild myocarditis (multifocal interstitial inflammation) has been reported in wild raccoons and opossums at necropsy.

**Communicability**

Chagas disease is not contagious during casual contact, but the parasite can be transmitted in blood or by contact with tissues. Carnivores can be infected if they eat T. cruzi-contaminated tissues. Many species of animals, including dogs, can serve as amplifying hosts, and infect the insect vectors. Vertical transmission can also occur in some animals.

**Diagnostic Tests**

Chagas disease can be diagnosed by microscopy, isolation of the parasite, serology and molecular techniques.

**Microscopic examination**

A presumptive diagnosis can be made if T. cruzi is observed by light microscopy in a stained blood smear or buffy coat sample, or in tissues such as the heart. The trypomastigotes occur as either short, broad forms or long, thin forms in the blood; in stained preparations, they form a C-shape. Amastigotes, which are found inside cells, are round or ovoid, and 1.5-4 μm in diameter. Differentiating T. cruzi from the morphologically similar, non-pathogenic species T. rangeli is difficult.

**Isolation of the agent**

T. cruzi can be cultured from blood samples or tissues; culture is usually more successful in the acute stage. This organism often occurs in the myocardium, but it can also be isolated from other organs such as the lymph nodes, liver, gastrointestinal tract, brain, cerebrospinal fluid and adrenal gland. Various specialized media including liver infusion tryptose medium or Novy-MacNeal-Nicolle medium, as well as Vero cell lines may be used. The agent may also be isolated by inoculating blood into a guinea pig, mouse or rat. Xenodiagnosis is considered to be one of the most accurate tests for Chagas disease in South America.

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America, but it is of little practical importance for veterinarians in the U.S. Isolation of the agent may take 1 to 6 months, and requires highly trained personnel.

Serology

Indirect immunofluorescence is the most commonly used serological test. Other assays include radio-immuno-precipitation, direct and indirect hemagglutination, complement fixation and ELISAs. Cross-reactions can occur with other parasites, particularly Leishmania species.

Molecular techniques

Molecular detection methods including PCR and western blot (immunoblot) analysis techniques can detect T. cruzi DNA in tissues and blood.

Treatment

Occasionally, dogs have been treated with anti-parasitic drugs. These drugs appear to be more effective in the early stages; by the time of diagnosis, treatment may be too late to prevent the progression of the disease.

Prevention

Dogs and cats should not be allowed to eat tissues from potentially infected wild animals. Strict indoor housing in well-constructed homes or other facilities reduces the risk of infection. Housing animals indoors at night, when triatomine insects are active, may also be helpful. Residual insecticides sprayed regularly in kennels and surrounding structures may decrease the number of insect vectors. In breeding kennels, testing bitches for T. cruzi-might also decrease the incidence of Chagas disease by reducing vertical transmission. No vaccines are available.

Morbidity and Mortality

The prevalence rate can be high among wildlife in endemic areas. Surveys of wildlife in the U.S., conducted mainly in raccoons and opossums, have reported prevalence rates from 2% to 62%. In one study, 19 of 134 coyotes in Texas had antibodies to T. cruzi. In the U.S., the reported seroprevalence rates in dogs from Texas, Oklahoma, Louisiana, Georgia and other southern states vary from 1.1% to 8.8%. Over a 15-year period, more than 500 clinical cases of Chagas disease were reported among dogs in Texas, suggesting that this disease occurs regularly in some areas. In the Yucatan region of Mexico, one study reported that 10-14% of dogs were seropositive, but when both serology and PCR were used, the prevalence as high as 17%. The infection rate is highest among dogs that are regularly exposed to wildlife and insect vectors. Cats are frequently infected with T. cruzi in South America.

There is little information on the morbidity and mortality rates in dogs, except in experimentally infected animals, where these rates are high. In one study, 13 of 26 dogs infected with blood trypomastigotes died during the acute phase. In another, 26 of 38 dogs died during the acute phase, while the 12 survivors developed chronic disease and lived for 1 to 2 years. In a study using North American strains of T. cruzi from armadillos or opossums, 7 of 13 dogs died or were euthanized due to the severity of acute disease. Based on human data, some sources suggest that approximately 5% of naturally infected animals would be expected to develop symptoms during the acute stage. In naturally infected dogs with chronic cardiac disease, the survival time after diagnosis ranged from 0 months to 5 years.

Internet Resources

Centers for Disease Control and Prevention (CDC)
http://www.cdc.gov/chagas/

Public Health Agency of Canada - Disease Information.
American Trypanosomiasis or Chagas Disease
http://www.phac-aspc.gc.ca/tnp-pmv/info/am_trypan_e.html

Public Health Agency of Canada - Transfusion Transmitted Diseases/Infections
http://www.phac-aspc.gc.ca/hcai-iamss/tti-it/ttdi_e.html#chagas

Medical Microbiology
http://www.ncbi.nlm.nih.gov/books/NBK7627/

The Merck Manual
http://www.merck.com/pubs/mmanual/

The Merck Veterinary Manual
http://www.merckvetmanual.com/mvm/index.jsp

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*Link defunct as of 2009*