

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 human heart disease, megaesophagus and megacolon in Latin America, where the causative organism, *Trypanosoma cruzi*, is endemic. While this organism occasionally causes an acute illness, most people do not realize they are infected until several years or decades later, when some develop a chronic medical condition. Antiparasitic treatment is most effective in the acute stages of the infection, and may be ineffective once this period has passed. In addition to humans, *T. cruzi* can infect many other mammals and marsupials, some of which act as reservoir hosts. Clinical cases have been reported mainly in dogs and captive non-human primates, with few illnesses documented in other species. However, cases in animals might be underdiagnosed.

Chagas disease is usually transmitted by the bites of triatomine insects, also known as “kissing bugs.” These insects or their feces can also contaminate foods such as fruit juices, resulting in foodborne, sometimes life-threatening, outbreaks of acute Chagas disease in people. In South and Central America and parts of North America, *T. cruzi* cycles between triatomines in the environment and wild or domesticated animals. Some triatomine species occur in the wild, or invade human homes only opportunistically; others have adapted to live within substandard dwellings, where they hide in cracks during the day and emerge to feed on people and animals at night. Campaigns to eliminate triatomines in domestic cycles, together with diagnostic testing to prevent congenital or blood transfusion associated infections, are gradually reducing the incidence of Chagas disease in Latin America. However, this disease has become an increasing problem in non-endemic regions, where infections may not be recognized in immigrants and travelers, and where the few effective drugs may not be widely available.

Etiology

Chagas disease results from infection with the protozoan parasite *Trypanosoma cruzi* (*T. cruzi sensu lato*), a member of the family Trypanosomatidae. This organism is widely referred to as "*T. cruzi*," without indication of the subspecies. Another subspecies, *T. cruzi marinkellei*, occurs in bats but is not known to infect other animals.

Historically, *T. cruzi s.l.* was divided into two major groups, I and II, for epidemiological purposes, with further subdivisions in group II. Since 2009, it has been classified into several genotypes, also called discrete typing units (DTUs). Currently, there are seven recognized DTUs, TcI through TcVI and the bat-associated genotype Tcbat/ TcVII. Some DTUs may predominate in certain sylvatic or domestic cycles or occur in different geographic areas, and some might have a tendency to cause different syndromes; however, this is still incompletely understood. Mixed infections with more than one DTU are common. All DTUs including Tcbat can infect humans.

Species Affected

T. cruzi has been detected in more than 150 species of mammals and marsupials, which appear to be widely susceptible to infection. It is known to infect carnivores (e.g., dogs, cats, wild canids and felids), livestock (pigs, sheep, goats, horses), lagomorphs, raccoons (*Procyon lotor*, *P. cancrivorus*), coatis (*Nasua nasua*, *Nasua narica*), skunks, numerous rodents, bats, xenarthra (anteaters, armadillos and sloths) and non-human primates, among others. Clinical cases have mainly been reported in dogs and captive non-human primates; however, evidence of pathology or clinical signs have been documented rarely in other species, including a horse, cats and a captive hedgehog (*Atelerix albiventris*). Birds, reptiles and fish seem to be refractory to infection.

Which hosts act as reservoirs is still debated and may vary between ecosystems. Some proposed reservoir hosts include opossums (*Didelphis marsupialis* and *D. virginiana*), armadillos of the genus *Dasypus*, raccoons, coatis, bats, certain primates such as the golden lion Tamarin (*L. rosalia*), rodents (e.g., woodrats [*Neotoma* sp.] in California), dogs, cats and domesticated guinea pigs.

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Zoonotic potential

T. cruzi affects humans. People are also involved in maintaining this organism in domestic transmission cycles.

Geographic Distribution

T. cruzi is endemic in the Americas from the U.S. to Chile and central Argentina. In the U.S., this parasite has been found in triatomines in approximately the southern half of the country and California. Imported human infections also occur in non-endemic regions. Triatomine insects that might have the potential to transmit this organism have been described in Africa, parts of Asia, and northern Australia, but *T. cruzi* is not currently endemic in any of these regions,

Transmission and Life Cycle

Chagas disease is a vector-borne illness transmitted primarily by triatomine insects, which are also called reduviid insects, “kissing beetles/ bugs” or “assassin bugs.” More than 130 species of triatomines appear to be capable of transmitting *T. cruzi* with varying efficiency. The most important species belong to the genera *Triatoma*, *Rhodnius* and *Panstrongylus*, but other genera are also significant in some areas.

T. cruzi completes its life cycle by cycling between these insects and vertebrate hosts. Triatomines sometimes live in close association with a host species (e.g., in the nests of coatis); however, they will opportunistically bite other animals or people. In the sylvatic (wild) cycle, *T. cruzi* cycles between wildlife and triatomine insects in a variety of habitats. These triatomines sometimes invade houses or outbuildings when they are attracted to light, heat or certain odors. A domestic transmission cycle also exists in Mexico and parts of Central and South America. In this cycle, some triatomine species have colonized primitive adobe, grass and thatched houses, resulting in continuous transmission between humans and insects. Certain vectors such as *T. infestans* primarily exist in such domestic cycles. There are also transmission cycles between triatomine insects and domesticated animals (peridomestic cycles). Other insects might transmit *T. cruzi* occasionally but their significance is still unclear. Bedbugs (*Cimex lectularius*) were shown to be competent vectors in the laboratory, and blood-sucking lice were infected in a captive primate facility.

In its mammalian host, *T. cruzi* occurs in the blood as trypomastigotes (extracellular nondividing forms) and in cells as amastigotes (replicative forms). Insects acquire trypomastigotes via a blood meal. After 2-4 weeks of development, some of the parasites migrate to the insect’s hindgut, where they are transformed into infective metacyclic trypomastigotes and released in the feces. The most effective vector species tend to defecate on the host, either during or after feeding, facilitating parasite transmission through mucous membranes or breaks in the skin. Scratching can help inoculate them into the bite wound. Humans and animals can also be infected by eating

triatomine insects or insect feces. Human outbreaks have been linked to triatomine-contaminated fruits, vegetables and unpasteurized fruit juices. Experiments suggest that infectious *T. cruzi* can persist for more than 24-72 hours in some juices held at room temperature, and sometimes longer when they are refrigerated. Contamination from the anal gland secretions of opossums, which can contain infectious *T. cruzi*, was thought to be involved in at least one outbreak involving vegetables. Undercooked meat or other animal tissues, especially game meat from reservoir hosts, has been implicated uncommonly in human cases, and could also be an important source for some animals.

T. cruzi is not contagious by casual contact; however, it can be transmitted in blood transfusions and transplanted organs. Transplacental transmission can occur in humans. It has also been documented in some other species, including dogs and bats. *T. cruzi* may be present occasionally in milk, but transmission to human infants by breast-feeding seems to be very rare. However, the risk may be higher when the milk is contaminated by blood, or at times when the parasite levels are higher, particularly the early stages of the infection. Sexual transmission has been demonstrated in acutely and chronically infected mice in the laboratory, but its significance in other species (if any) is still uncertain.

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, an obligate intracellular parasite, can be destroyed by several hours of exposure to direct sunlight and drying, or other harsh environments. This organism is also susceptible to many disinfectants including 1% sodium hypochlorite, 70% ethanol, iodine/alcohol solutions, glutaraldehyde and formaldehyde. It can be inactivated by standard heat sterilization methods: moist heat (121°C/ 250°F for a minimum of 15 minutes) or dry heat (160 to 170°C/ 320-338°F for at least one hour).

Infections in Animals

Incubation Period

Experimentally infected dogs may develop acute clinical signs several days to several weeks after inoculation; however, dogs can also remain asymptomatic for a prolonged period before developing heart disease. In some naturally infected dogs, this seems to take years.

Clinical Signs

Dogs

Most of the information about Chagas disease in dogs comes from experimentally infected animals. Dogs that become ill shortly after inoculation are reported to have a fever and nonspecific signs of illness (e.g., anorexia,

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lethargy, an unkempt hair coat, lymphadenopathy), and, in some cases, additional signs including enlargement of the liver and/or spleen, diarrhea, ascites and palpebral edema. Some dogs can develop acute myocarditis at this stage, sometimes resulting in arrhythmias or sudden collapse and death. A few animals inoculated with a South American strain of *T. cruzi* had a localized painless induration, called a chagoma, where the parasite entered the skin; however, chagomas have rarely been documented in other dogs. After the acute phase, experimentally infected dogs become asymptomatic for a prolonged period, but some animals eventually develop congestive heart failure. Right-sided heart failure is usually seen initially, but progresses to chronic myocarditis with cardiac dilatation and arrhythmias.

Limited information is available from naturally infected dogs. In one large retrospective study from Texas, heart disease was the most common syndrome in both puppies and adults. Sudden death was the most common presentation; other common findings included various signs that can be associated with heart disease, such as ascites, an enlarged heart, cardiac conduction disturbances, lethargy and respiratory difficulties. Hepatomegaly, anemia, diarrhea, vomiting and edema were reported less often. In another report, a few congenitally infected puppies had a persistent fever, weight loss, chronic diarrhea and signs of heart disease.

Atypical cases have also been reported in dogs. One infected dog had a fever, anorexia, lethargy, weight loss, generalized lymphadenopathy and dependent, pitting edema, without cardiac signs or lesions. Neurological signs such as lameness or coma have been reported occasionally. A 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 neurological 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 in this species.

Nonhuman primates

Captive nonhuman primates usually have few or no signs during the acute stage of the disease, but they can develop irreversible myocarditis and dilated cardiomyopathy during the chronic stage. Subclinical conduction and echocardiographic abnormalities may be seen before heart disease becomes apparent. Megaesophagus and encephalitis have also been reported in nonhuman primates.

Other species

Information about Chagas disease in other species is limited. Symptomatic Chagas disease has been rarely described in cats. The clinical signs in reported cases from South America include fever, edema, weight loss and

neurological signs such as convulsions and paresis. An infected horse in the U.S., which had parasites in a spinal lesion, had a 6-month history of hindlimb lameness and ataxia. Experimentally infected pigs did not develop acute clinical signs and had minimal tissue lesions.

Mild, histologically evident myocarditis, without obvious clinical signs, was reported in some infected wild raccoons and opossums. Naturally infected rats can develop arrhythmias and more severe cardiac lesions. The death of one captive hedgehog was also attributed to Chagas disease. Infertility, fetal losses, reduced birth weights and early postnatal deaths were reported in some pregnant mice.

Post Mortem Lesions [Click to view images](#)

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. There may also be multiple yellowish-white spots and streaks, mainly involving the coronary groove. Pericardial effusion has been reported in some naturally infected dogs. Additional lesions may include secondary pulmonary edema, congestion in the liver, spleen and kidneys, and a modified transudate in the peritoneal cavity. Gross lesions may be absent in some acute cases. In most dogs with chronic Chagas disease, the heart is bilaterally enlarged and flaccid, with thinning of areas in the ventricular walls. In some atypical cases, however, the heart did not have gross lesions.

Microscopic and/or gross cardiac lesions of varying severity (myocarditis, epicarditis) have usually been the most prominent lesions in other animals, including nonhuman primates, wild raccoons and opossums. However, the heart and other internal organs were unaffected in a horse with neurological signs. In this animal, the lesions were limited to focal congestion and hemorrhage in a segment of the spinal cord where trypanosomes were detected on histopathology.

Diagnostic Tests

T. cruzi or its nucleic acids may be found in the blood or myocardium, and sometimes in other organs and tissues (e.g., lymph nodes, liver, gastrointestinal tract, cerebrospinal fluid). Direct detection of the organism is more likely to be successful in the acute stage of the infection. Organisms may sometimes be observed by light microscopy in a stained smear or tissues, but must be distinguished from other pathogenic and nonpathogenic trypanosomes (e.g., *T. rangeli*) by additional testing. Culture of *T. cruzi* may take 1- 6 months, and requires highly trained personnel. This organism can be isolated in various specialized media including liver infusion tryptose medium or Novy-MacNeal-Nicolle medium, as well as Vero cell lines. Xenodiagnosis is considered to be one of the most accurate tests for Chagas disease in South America, but it is not usually available in other regions. 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 1-2 months

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later. Animal inoculation (guinea pig, mouse or rat) has also been used to isolate *T. cruzi*, though mainly in the past. Some laboratories may have PCR tests that can detect *T. cruzi* DNA directly in clinical samples.

Various serological tests, including indirect immunofluorescence (IFA), direct and indirect hemagglutination, complement fixation, ELISAs, radioimmunoprecipitation and other assays, can detect antibodies to *T. cruzi*. Some of these tests were originally developed for humans, but have been used in dogs and other animals. IFA is used most often in dogs. There are no validated serological tests for some species. Some tests may be complicated by cross-reactions with other parasites, particularly *Leishmania*. The presence of antibodies alone does not prove that an illness is caused by *T. cruzi*, as some infected animals are asymptomatic. However, a high titer, combined with characteristic clinical signs, is one criterion that has been used to diagnose Chagas disease in dogs.

Treatment

Dogs have occasionally been treated with anti-parasitic drugs. Benznidazole and nifurtimox, the two drugs used to treat Chagas disease in Latin America, are not widely available outside this area. These drugs are more likely to be effective in the early stages; by the time of diagnosis, treatment may be too late to prevent the progression of the disease. In one recent study, treatment of experimentally infected dogs with benznidazole during the chronic stage did not prevent increases in the size of the heart chambers, compared to untreated dogs, although cardiac function was slightly better.

Control

Disease reporting

Veterinarians who encounter or suspect Chagas disease should follow their national and/or local guidelines for disease reporting. This disease is reportable in some U.S. states, but not others.

Prevention

Preventive measures mainly depend on reducing exposure to triatomine insects and preventing the ingestion of infected raw tissues, especially tissues from potentially infected wild animals. Strict indoor housing in well-constructed homes or other facilities is likely to reduce exposure to infected insects. Housing animals indoors at night, when most triatomine insects are active, may also be helpful. Some triatomines have been found to colonize kennels in the U.S., contributing to cases of Chagas disease in dogs. Infected insects have also been found at some primate facilities. Residual insecticides may decrease the number of insect vectors in these situations, and netting or mesh barriers to exclude bugs may also be helpful. In breeding kennels, testing bitches for *T. cruzi*-might decrease the incidence of Chagas disease. However, all dogs do not seem to pass the parasite to their offspring.

Morbidity and Mortality

Infection with *T. cruzi* can be common among wildlife and domestic animals in endemic areas, but the prevalence varies with the region and species. Surveys in South America and the Caribbean have reported seroprevalence rates ranging from 4% to > 60% in dogs, and from 2% to 79% in cats. Approximately 1% to 22% of dogs in various southern U.S. states also have antibodies to this organism. In the U.S., the infection rate is highest among dogs in rural locations, and in dogs that are regularly exposed to wildlife (e.g., hunting dogs) and/ or insect vectors. Up to 50-60% of kenneled hunting dogs and dogs in triatomine infested indoor-outdoor kennels were seropositive in two studies.

In some parts of the U.S., practitioners may regularly see clinical cases in dogs. One study documented more than 500 cases over a period of 15 years. Low levels of local transmission have also been reported in some U.S. captive primate colonies. There is little information about other domestic animal hosts, including cats. A clinical case in one horse in the U.S., and a report of antibodies in 40% of horses tested, suggest that some cases in this species might be missed.

There is little information on the morbidity and mortality rates in dogs, except in experimentally infected animals, where large numbers of parasites may be administered, and 50% or more of the animals may die during the acute stage. Based on human data, some sources suggest that approximately 5% of naturally infected dogs would be expected to become symptomatic during the acute stage. After the diagnosis of chronic cardiac disease, naturally infected dogs survived for 0 months to 5 years.

Whether immunosuppression ever plays a role in the severity or onset of clinical signs in animals is generally unknown. However, apparent reactivation of *T. cruzi* infection occurred in two rhesus macaques (*Macaca mulatta*) coinfecting with an immunosuppressive virus (simian immunodeficiency virus).

Infections in Humans

Incubation Period

In people who develop symptoms in the early stages of infection (acute Chagas disease), the incubation period is usually 5-14 days after exposure via triatomine bites, and it can be 3-30 days in foodborne outbreaks. Infections acquired in a blood transfusion seem to become apparent somewhat later, with incubation periods ranging from 20 days to 3 months. Many people have no symptoms until the chronic stage, which can occur years or decades after they were infected.

Clinical Signs

The acute phase is defined as the period during which parasites can be found easily in the blood. This stage often passes without any clinical signs except in foodborne outbreaks, where the dose of the organism is likely to be

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higher, or in more vulnerable populations such as young children. 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. Some patients may develop edema, either generalized or localized to the face and/or lower extremities. Epigastric pain, jaundice, hematemesis, hematochezia, melena, epistaxis and cough have also been reported, especially in foodborne cases. Lesions sometimes occur at the site of parasite entry: there may be a chagoma (a localized painless, erythematous induration) on the skin, or painless edema of one (or occasionally both) eyes, often accompanied by conjunctivitis and enlargement of the local lymph nodes. The latter syndrome, called Romaña's sign, occurs after entry via the conjunctiva, and usually persists for 1 to 2 months. Patients occasionally develop a diffuse morbilliform rash, but this usually disappears within several days. Serious complications during the acute phase can include subclinical or clinical myocarditis, with signs ranging from ECG abnormalities and arrhythmias to death, as well as meningoencephalitis. These serious syndromes tend to be uncommon, but have been reported more often in foodborne outbreaks. In most cases, the clinical signs of acute Chagas disease resolve within weeks to months without treatment; however, some acute cases can be fatal.

After the acute stage, parasites disappear from the blood, and infected people become asymptomatic for a prolonged or indefinite period. The period between the acute stage and the onset of chronic clinical signs is sometimes called the indeterminate phase; others consider it to be part of the chronic stage. In some cases, a mild sensory-motor peripheral neuropathy has been found during the indeterminate as well as the chronic stage.

Years after they were infected, some people develop signs of organ failure, usually involving the heart, and/or the digestive system. Signs of cardiac involvement can include arrhythmias and conduction abnormalities, heart failure, apical aneurisms, embolic signs including stroke or pulmonary embolism, and sudden death. Left ventricular dilation and dysfunction may be apparent initially, but biventricular congestive heart failure is common in the later stages. Some patients have chest pains without evidence of coronary artery disease. The most common gross abnormalities in the digestive tract are megaesophagus and megacolon. 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 and esophageal rupture are possible complications, and there is an increased risk for cancer of the esophagus. Megacolon results in severe constipation, which can last for a few days to months. There may also be abdominal pain, often associated with episodes of constipation, and asymmetric distention of the abdomen. Potential complications of megacolon include intestinal occlusion, volvulus, fecalomas, ulceration or intestinal perforation with peritonitis. Patients with Chagas disease

may also develop gall bladder abnormalities (mega-gallbladder or cholelithiasis), and they have an increased chance of developing gastric ulcers or chronic gastritis. Small intestinal dilatation is rare.

Infected women can give birth to congenitally infected infants, whether or not they have clinical signs at the time of the pregnancy. Many or most of these infants are asymptomatic at birth, but a few are born with clinical signs. Others can become ill over the next few weeks or months, or develop chronic Chagas disease later in life. The most common symptoms in infants are premature birth, low birth weight, hepatosplenomegaly, jaundice, anemia and thrombocytopenia. Respiratory distress syndrome, meningoencephalitis and/or signs of acute myocarditis/cardiac insufficiency can be seen in severe cases. Uncommonly reported conditions include megaesophagus and megacolon, which may even be present at birth, and ocular disease with chorioretinitis and opacification of the vitreous body. Transplacental infections are also suggested to cause some abortions, but this remains to be proven.

Immunosuppressed individuals are at risk for reactivation of *T. cruzi* replication. While reactivation may be subclinical, it can also result in the reappearance of acute stage symptoms including nonspecific signs (e.g., malaise, weight loss, hepatosplenomegaly), cardiac complications, meningoencephalitis, or other syndromes such as fever and skin lesions, especially erythematous nodules or plaques. Cardiac involvement can appear as acute myocarditis, but it may also present as an accelerated progression of existing chronic Chagas heart disease. In transplant patients, the signs of reactivation can be mistaken for rejection of the organ. Both encephalitis and brain masses caused by *T. cruzi* are both common in HIV infected people with low CD4 counts. These mass lesions is not usually seen in immunocompetent people or in patients with other immunosuppressive conditions, although encephalitis is possible. Other complications, including parasite invasion of the gastrointestinal tract and peritoneum have been reported.

Diagnostic Tests

Light microscopy can sometimes detect *T. cruzi* in stained samples of blood, cerebrospinal fluid or tissues (e.g., heart, chagomas) during the acute stage or in reactivated infections. Concentration techniques, such as examination of the buffy coat, increase the probability of finding the parasites in blood. In immunocompromised patients, parasites may also be present in various atypical sites, including skin lesions. PCR can be used for diagnosis during the acute stage and in congenitally infected infants. Quantitative (real time) PCR may demonstrate rising levels of nucleic acids when parasites are reactivated in chronically infected, immunosuppressed individuals. *T. cruzi* can also be identified by xenodiagnosis, cultured from blood samples or tissues, or isolated by animal inoculation. However, these isolation methods are generally used only to confirm a diagnosis if other tests are inconclusive, in research, or to isolate strains of parasites.

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Serology is most often used to diagnose chronic infections, or to identify congenitally infected infants after maternal antibodies decline. The World Health Organization currently recommends the use of two serological tests based on two different antigens. Commonly used assays include IFA, hemagglutination and immunoassays such as ELISAs. Other tests such as radioimmunoprecipitation or trypomastigote excreted-secreted antigen immunoblot (TESA-blot) may be employed, often as confirmatory tests, in some areas. Serology is sometimes negative or inconclusive in people who are immunosuppressed.

Treatment

People with acute or reactivated infections, and congenitally infected infants, are treated with the antiparasitic drugs benznidazole or nifurtimox. In reactivated infections, this treats the clinical signs but does not eliminate the parasite from the body. Outside Latin America, these drugs may be available only through special programs and government sources (e.g., as an investigational new drug from the CDC Drug Service in the U.S.). They must be given for prolonged periods, and can have significant side effects. New drugs and new combination treatments with other drugs are being researched.

Treatment early in the infection can prevent the development of chronic complications. After this stage, antiparasitic drugs become less effective, and drug treatment recommendations may vary with the age of the patient and other factors. For instance, children seem to respond well, and they are treated whether the infection is acute or chronic. Chronic cardiac disease is treated similarly to heart conditions from other causes. 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

Prevention relies on preventing bites or food contamination from triatomine insects, congenital transmission, and person-to-person transmission in blood transfusions or organ transplants.

Most species of triatomine insects, including all species involved in domestic cycles, usually feed at night and withdraw to their hiding places in daylight. Sleeping in poorly constructed houses should, if possible, be avoided. Houses can be improved by plastering walls, improving flooring and taking other measures to remove the cracks where these insects hide, and using screens and other barriers to exclude peridomestic and sylvatic triatomines. Animal pens and storage areas and wild animal nests, which may contain triatomines, should be kept at a distance from the home. Where triatomine insects may be present, sleeping under an insecticide-impregnated bed net can be helpful. Bed nets should be tucked tightly under the mattress before dusk.

Regular spraying of insecticides in and around houses can reduce the number of insects, and in some cases, eliminate

them. Vector control programs to eliminate domestic populations of *T. infestans* and other important triatomines are being conducted in a number of areas in South and Central America. Triatomine species that occur in sylvatic cycles, but opportunistically colonize human dwellings, can also be controlled or eliminated within the home; however, they may recolonize. Where these bugs enter homes only occasionally, such as in some parts of the U.S., frequent inspection of hiding places (e.g., between mattresses, under bedding, in pet beds), may be effective. Measures to reduce the attraction of sylvatic triatomines to houses include minimizing outdoor lighting, changing white outdoor lights to yellow bulbs, and closing blinds/ curtains when indoor lights are on. Thick clothing, including shoes and socks, may help prevent bites outdoors. Preliminary work suggests that citronella may be an effective repellent.

Preventive measures for foodborne Chagas disease include thoroughly washing vegetables, practicing good hygiene (e.g., washing the hands before food preparation or eating) and protecting all foods from contamination by insects or their feces. Foods that might be contaminated, including meats, should be cooked to $> 45^{\circ}\text{C}$ (113°F) or pasteurized.

Pregnant women can be tested for Chagas disease, and their infants monitored and treated if necessary. Some recent evidence suggests that antiparasitic treatment of women before a pregnancy may significantly reduce the risk of transmission, although infected women may not themselves be cured. Blood supplies can be protected by screening donors. The risk of infection can be decreased, but not eliminated, by leucocyte depletion, irradiation or freezing. Some countries do not allow high risk donors to donate blood. Heart or intestinal transplants from infected people are contraindicated, but policies on the transplantation of other organs vary. A limited number of studies suggest that *T. cruzi* is transmitted in $< 35\%$ of kidney or liver transplants, and post-transplant monitoring and treatment can minimize the risk.

People who handle potentially infected live animals, carcasses, tissues or *T. cruzi* cultures (e.g., veterinarians, hunters, laboratory workers) should protect their skin and mucous membranes from contamination with parasites in blood or tissues. If an accidental exposure occurs, the site should be disinfected immediately.

Morbidity and Mortality

Risk factors for Chagas disease include living in substandard housing and/or participating in activities that increase contact with triatomine vectors or the tissues of infected animals. People who spend significant amounts of time outdoors in triatomine habitats (e.g., piassava gatherers in the Amazon) are among those at risk. In some areas, outbreaks have been linked to foods such as fresh fruit juices, vegetables and even water contaminated by sylvatic triatomines. Reports of such outbreaks seem to be increasing, especially where vectors have been controlled inside homes.

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Occasional cases have also been associated with undercooked meat, especially game meat.

In endemic areas, many people become infected with *T. cruzi* in childhood. Congenital infections occur in both endemic and non-endemic regions. Approximately half of mothers first infected during the pregnancy, and 5% of chronically infected women, are thought to transmit *T. cruzi* to their infants. The latter rate may differ between regions, with reported transmission rates ranging from 1% to 10%. Transmission also seems to be more common if the mother is coinfecting with HIV. All infants born to an infected mother are not necessarily infected.

In South and Central America, campaigns to control domestic cycle vectors have significantly decreased the number of new human infections, but Chagas disease is still a widespread problem. Most cases of Chagas disease outside Latin America occur in immigrants and travelers. The number of infected people, and the risk of transmission to blood transfusion recipients and infants, have become significant concerns in the U.S., Europe and other areas. Infections acquired locally from triatomines seem to be uncommon in the U.S., with < 50 cases documented as of 2017; nevertheless, small numbers of cases have been identified regularly in recent years.

The morbidity and mortality rates vary with the stage of the disease and syndrome. Approximately 5% of people infected with *T. cruzi* via insect bites are thought to develop acute symptoms, and < 1% of these cases are severe overall. Severe cases and deaths are more common in young children, the elderly and people who are immunocompromised. Higher morbidity and mortality rates have been reported among healthy people after foodborne transmission: in outbreaks affecting 10 people or more, 0% to 33% of the infected people died. Neurological involvement can have a high case fatality rate, especially in severely immunosuppressed patients.

An estimated 20-30% of humans infected with *T. cruzi* eventually develop chronic disease, with some estimates ranging from 10% to 50%. Why this infection progresses to chronic organ involvement in some patients, but not others, is not known. Cardiac disease occurs throughout the regions where Chagas disease is endemic, but digestive abnormalities seem to be rare in some areas. Cardiac involvement is eventually fatal in many chronic cases. Occasionally, deaths are also caused by other chronic complications, such as volvulus of a dilated megacolon.

Internet Resources

[Centers for Disease Control and Prevention \(CDC\), U.S.](#)

[Global Chagas Disease Coalition](#)

[International Federation of Associations of People Affected by Chagas Disease \(FINDECHAGAS\)](#)

[Southern Cone Initiative to Control/Eliminate Chagas Disease \(INCOSUR\)](#)

[The Merck Manual](#)

[The Merck Veterinary Manual](#)

[World Health Organization. Chagas Disease](#)

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