# 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 zoonotic, arthropod-borne disease caused by the parasite *Trypanosoma cruzi*. This organism is known to infect many mammals and marsupials, though clinical cases are most widely recognized and understood in humans. Acute infections in healthy people are usually mild or subclinical except in foodborne outbreaks, and most people are unaware that they have become infected. However, they often continue to carry the organism, and while many carriers experience no apparent ill effects, a significant number eventually develop chagasic heart disease, megaesophagus, megacolon or other chronic medical conditions. Cases affecting the heart, which tend to have a high rate of complications such as arrhythmias and strokes, are particularly serious and have a high fatality rate. The effects of *T. cruzi* on animals are less well understood, but a pattern of illness similar to that in humans has now been recognized in some dogs and nonhuman primates, and sporadic clinical cases have been documented in other species.

Exposure to *T. cruzi* is most frequent in parts of Latin America; however, this organism is also endemic in parts of the U.S., and imported infections are common among Latin American immigrants in a number of nations. While this organism is not contagious during casual contact, it can be transmitted in blood transfusions and transplants, and pregnant women sometimes transmit *T. cruzi* to the fetus, resulting in ongoing propagation of the infection within families even in non-endemic regions. Locally acquired human cases seem to be infrequent in the U.S., where the triatomine vectors do not usually become established in homes, but animals are more likely to be exposed, particularly in some dog kennels, outdoor primate facilities and zoos.

## Etiology

Chagas disease results from infection with the protozoan parasite *Trypanosoma cruzi* subsp. *cruzi* (or *T. cruzi sensu lato*), a member of the family Trypanosomatidae. Another subspecies, *T. cruzi* subsp. *marinkellei*, only seems to occur in bats, and unless otherwise indicated, the term *T. cruzi* usually refers to *T. cruzi cruzi*.

*T. cruzi s.l.* has been divided into several genotypes, called discrete typing units (DTUs). They include TcI through TcVI, as well as Tcbat/ TcVII, which is specifically associated with bats (but not the only DTU found in bats). All of the currently recognized DTUs can infect humans, though Tcbat has been reported very rarely. DTUs, which may vary in prevalence between different regions, have been proposed to affect host specificity, clinical signs, the frequency of vertical transmission and other aspects of Chagas disease, though much of this remains to be proven.

## Species Affected

*T. cruzi* has a very wide host range. It is thought to be capable of infecting animals in most or all mammalian or marsupial orders, with virological or serological evidence of infections in more than 150 species including dogs, cats, wild canids and felids, livestock (pigs, sheep, goats, equids, South American camelids), lagomorphs, raccoons (*Procyon lotor*, *P. cancrivorus*), opossums (*Didelphis* and *Philander* spp.), coatis (*Nasua nasua*, *N. narica*), skunks, many wild or domesticated rodents including guinea pigs (*Cavia porcellus*), bats, anteaters, armadillos and non-human primates, among others. *T. cruzi*-related illnesses have mainly been seen in dogs and captive nonhuman primates, but rare clinical cases have also been reported in other hosts including cats, a horse, a llama, an aardwolf (*Proteles cristatus*), bears (American black bear, *Ursus americanus*; polar bear, *Ursus maritimus*), red pandas (*Ailurus fulgens*), slender-tailed meerkats (*Suricata suricatta*), African pygmy hedgehogs (*Atelerix albiventris*), sugar gliders (*Petaurus breviceps*) and a red-necked wallaby (*Macropus rufogriseus*). Most of the wildlife cases occurred in zoo animals or exotic pets, but an American black bear cub was infected in the wild. Histological lesions consistent with possible cardiac damage from *T. cruzi* have also been reported in some free-living wildlife including raccoons, opossums (*Didelphis* and *Philander* spp.) and coyotes (*Canis latrans*).

Which of these animals are important in maintaining *T. cruzi* is still debated and could vary between ecosystems. Some proposed reservoir hosts in North or South America include opossums (*Didelphis* spp. and *Philander opossum*), armadillos of the

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genera *Dasypus* and *Zaedyus*, raccoons, coatis, skunks, bats, certain primates, rodents (e.g., *Neotoma* sp. woodrats in California), dogs, cats and farmed guinea pigs.

In the past, birds, reptiles and fish were said to be refractory to infection with *T. cruzi*. However, new reports of PCR positive lizards, snakes and an American barn-owl (*Tyto furcata*), together with two reports of experimental transmission to triatomine insects from lizards, and a study from the 1950s, which reported experimental infection of some lizards, seem to challenge this assumption .

### *Zoonotic potential*

*T. cruzi* affects humans, who 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 triatomine insects in roughly the southern half of the country, and further north in some mild climate areas such as California. Research in the 1950s suggested the possibility of local transmission to humans and animals at some sites in the Caribbean, though no studies seem to have been done since that time. Triatomines are also present in Asia, but there is currently no evidence that they are infected with *T. cruzi* in this location.

## Transmission and Life Cycle

Chagas disease is transmitted primarily by triatomine (reduviid) insects, which are also known as "kissing beetles" or "assassin bugs" and are biological vectors for *T. cruzi*. More than 130 species of triatomines can transmit this organism with varying efficiency, with the most important species belonging to the genera *Triatoma*, *Rhodnius* and *Panstrongylus*. Whether any other insects might occasionally act as vectors is unclear, though bedbugs (*Cimex lectularius*) were shown to be competent vectors in the laboratory, and blood-sucking lice were infected in a captive primate facility.

Three basic cycles maintain *T. cruzi* in nature: a sylvatic cycle between sylvatic triatomines and wildlife, a peridomestic cycle that involves livestock and other domestic animals, and a domestic cycle between people and certain triatomine species, such as *T. infestans*, that have colonized houses. The domestic cycle was responsible for many infections in Latin America at one time, and can involve household pets as well as people. While this cycle is particularly common where cracks (e.g., in adobe walls) or thatched roofs provide good hiding places for triatomine insects to hide during the day, these bugs can find niches in more modern homes. The species from sylvatic or peridomestic cycles also invade homes and outbuildings sometimes in search of a meal, and may persist for a time.

Triatomine insects become infected with *T. cruzi* when they take a blood meal from a vertebrate host. After 2-4 weeks of parasite development in the insect's intestines, they release infective metacyclic trypomastigotes in the feces. The most effective vector species tend to defecate on the host during or immediately after feeding, which allows the parasites to immediately enter the body through mucous membranes or breaks in the skin, including the bite wound. Scratching by the host aids inoculation. Outside the insect, *T. cruzi* remains viable for a relatively short time: it was reported to lose motility and infectivity in triatomine feces within 30 minutes, and was nearly non-infectious for mice after 9 hours. Once inoculated into the vertebrate host, organisms circulate in the blood as trypomastigotes, but replicate as amastigotes in various tissues.

Some hosts also become infected by eating infected triatomine insects. This is thought to be a particularly important route in insectivores, but many other animals including dogs, cats and nonhuman primates may occasionally eat these insects. Eating undercooked meat or other animal tissues is thought to cause some infections in carnivores, and fruits, vegetables and unpasteurized fruit juices contaminated by insects or their feces regularly serve as sources of infections for people. Contamination of food by the secretions from opossum anal glands, a site of *T. cruzi* replication, was also proposed to account for a few human outbreaks. *T. cruzi* seems to remain viable in most fruits, vegetables and juices from 6 to 72 hours at room temperature, though it can survive longer (e.g., up to 16 days) when the food is refrigerated. It persists for only a short time on a few fruits, such as pineapple, and one study found that it was destroyed rapidly in non-distilled water.

Although *T. cruzi* is not spread by casual contact between hosts, it can be acquired in blood transfusions and transplanted organs. The significance, if any, of a recent finding of a PCR-positive salivary gland in a *Desmodus rotundus* vampire bat is uncertain. Transplacental transmission has been reported in a number of species, including humans and dogs. While transmission in milk also appears possible, the importance of this route is incompletely understood. In humans, the risk might be greater when the woman was recently infected and parasite levels are higher, or if the milk is contaminated by blood (e.g., trauma to the nipples). Sexual transmission has been demonstrated in acutely and chronically infected mice, and both nucleic acids and live organisms have been reported in human semen.

Laboratory infections usually occur when parasites contact mucous membranes or broken skin, or are accidentally injected via needlestick injuries. Aerosol transmission might also be possible in this setting. Under laboratory conditions, *T. cruzi* trypomastigotes survived for 24 hours at room temperature and at least 10 days at 4ºC (39°F) in human cells and tissues.

## Disinfection

*T. cruzi* is susceptible to many disinfectants including 1% sodium hypochlorite, 70% ethanol, iodine/alcohol solutions, Virkon, glutaraldehyde and formaldehyde. Blanching acai fruit at 70°C (158°F) for 10 seconds or pasteurization of acai juice at 82.5°C (181°F) for 1 minute was also reported to destroy the organism.

## Infections in Animals

### Incubation Period

Experimentally infected dogs may develop acute clinical signs several days to several weeks after inoculation; however, they and other species can also remain asymptomatic for many years before developing signs of heart disease or other chronic conditions.

### Clinical Signs

#### *Dogs*

Most of the information about Chagas disease in dogs comes from experimentally infected animals, which are often inoculated with high doses of the parasite and are more severely affected than most naturally infected dogs. In these animals, fever and nonspecific signs of illness (e.g., anorexia, lethargy, lymphadenopathy) and, in some cases, enlargement of the liver and/or spleen, diarrhea, ascites and palpebral edema may be seen soon after inoculation. A few dogs that received a South American strain of *T. cruzi* had a localized, painless induration (a chagoma) where the parasite entered the skin, though such lesions have rarely been documented in other studies. Some inoculated dogs also develop acute myocarditis around this time, sometimes resulting in arrhythmias or sudden collapse and death. However, most survive and become asymptomatic for a prolonged period. Some of these chronically infected animals eventually develop congestive heart failure, which tends to begin as right-sided heart failure and progresses to chronic myocarditis with cardiac dilatation and arrhythmias.

The limited number of reports in naturally infected dogs are consistent with this description, though early signs often seem to be milder. Some infected animals had a febrile illness with nonspecific clinical signs, lymphadenopathy, hepatomegaly, anemia, diarrhea, vomiting, edema, and/or weight loss, and a few congenitally infected puppies developed a persistent fever, weight loss, chronic diarrhea and signs of heart disease. However, the most prominent syndrome is heart disease in the chronic stage of the illness. Many of the deaths associated with this condition have been sudden. They sometimes occur in animals that have a history of cardiac dysfunction, but they are also seen in dogs with postmortem evidence of cardiac involvement but no previous signs of poor health or reduced performance. Various ECG abnormalities, especially supraventricular and ventricular arrhythmias and atrioventricular block, as well as echocardiographic changes and elevated levels of troponin (an indication of myocardial damage) have also been reported in seropositive dogs, even when overt clinical signs are not apparent.

Neurological signs such as lameness, ataxia, paresis or paralysis, incontinence, cranial nerve deficits and seizures have been reported occasionally in dogs, and one animal, which also had neurological signs, developed bilateral chorioretinitis and enophthalmos. Experimentally infected dogs can have 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 initially, though there are occasional reports of nonspecific signs of illness, anemia and/or hepatosplenomegaly, as well as a few reports of animals with evidence of cardiac involvement. Unusually, one rhesus macaque (*Macaca mulatta*) that appeared to be in the acute stage had orchitis, as well as nonspecific signs of illness, diarrhea and evidence of cardiomyopathy. An asymptomatic, acutely infected cynomolgus macaque (*Macaca fascicularis*) had abnormal laboratory findings that mainly consisted of large granular lymphocytosis and resolved spontaneously.

Like dogs, nonhuman primates subsequently enter a prolonged asymptomatic period, after which some chronically infected animals may develop myocarditis or dilated cardiomyopathy. Subclinical conduction and echocardiographic abnormalities have been documented sometimes before heart disease became apparent, and there are reports of megaesophagus or encephalitis. Some authors also mention abortions as a possible consequence of infection, and *T. cruzi* amastigotes were found in a placenta from a cynomolgus macaque after a stillbirth; however, a study of births in a colony of chronically infected rhesus macaques, as well as a comparison of seronegative and seropositive baboons (*Papio hamadrya*s spp.), found no evidence of significant reproductive dysfunction.

#### *Other species*

While symptomatic Chagas disease has been seen in cats, there are few published descriptions of clinical cases. The signs in some cats from South America were reported to include fever, edema, weight loss and neurological signs such as convulsions and paresis. A postmortem study of euthanized cats at an animal shelter in the U.S. found that seropositive cats were more likely to have histopathological evidence of mild to moderate cardiac inflammation, compared to seronegative cats, though a causative role remains to be proven. Reports of Chagas disease in other domestic animals are rare. An infected horse in the U.S., which had parasites in a spinal lesion, had a 6-month history of hindlimb lameness and ataxia. A llama with severe megaesophagus was PCR positive for *T. cruzi* in its digestive tract; however, llamas can develop megaesophagus unrelated to Chagas disease, and it is currently unclear whether or not this was an incidental finding.

Several clinical cases have been reported in captive African pygmy hedgehogs. One animal, which had evidence of a widely disseminated infection in its blood and tissues and was eventually euthanized, exhibited a progressive illness with nonspecific signs (decreased appetite and body condition, lethargy), oral ulcerations and erythema, greenish to bloody diarrhea, muscle atrophy, and progressive posterior paresis evolving into right hemiplegia. In another incident, pet hedgehogs and co-housed sugar gliders had

postmortem evidence of myocarditis and hemorrhagic gastrointestinal contents, but the clinical signs were limited to sudden death in both species and periorbital edema in the sugar gliders. There are also reports of cardiac lesions and arrhythmias in naturally infected rats.

Cardiac conditions have predominated in most case reports of other captive wildlife, often after sudden deaths or deaths associated with stressors, e.g., restraint during examination of a sick zoo animal. The strength of the evidence for a causative role in these cases varies, with most having been diagnosed on the basis of varying degrees of cardiac involvement ranging from mild myocarditis to gross ventricular dilatation, together with PCR evidence for the presence of the virus either in the heart or other tissues and, in some cases, observation of amastigotes identified as *T. cruzi* in the heart. A wallaby with heart disease was coinfected with *Toxoplasma gondii*, which can cause fatal toxoplasmosis in this species, though the lesions in the heart were mostly associated with *T. cruzi*. Histological evidence of inflammation in the heart has been seen in some infected but apparently healthy free-living wildlife including wild raccoons, opossums and coyotes.

Other lesions or clinical signs have also been reported occasionally. One red panda with a disseminated infection and serious cardiac involvement had mild anemia, pelvic limb weakness and lameness. The authors speculated that the latter signs might have been related to evidence of peripheral neuritis found at necropsy; however, protrusion of a lumbar intervertebral disk was also noted on a CT scan. Another red panda with evidence of myocarditis, which was co-infected with *Bacillus piliformis*, had ocular signs (keratitis, panuveitis, detached retina) and histological evidence of inflammation in the skeletal muscles. An aardwolf with ataxia, weakness and polypnea, which died during examination, had granulomatous myositis of the skeletal muscles, vaginitis, nephritis and inflammation in the intestinal tract as well as cardiomyopathy.

One unusual report described an acute infection in a 3-month-old orphaned black bear cub, which developed increasing weakness, anorexia, lethargy, anemia and elevated liver enzymes. The authors noted that some of these signs could also have been caused by concurrent proliferative bone lesions, which were considered unrelated to the infection. The illness eventually resolved without treatment, and no cardiac abnormalities were found.

## Post Mortem Lesions

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In experimentally infected dogs that die during the acute stage of the infection, the gross lesions, if any, mainly involve the heart, and are particularly prominent on the right side. Common findings include a pale myocardium, with subendocardial and subepicardial hemorrhages, and in some cases, multiple yellowish-white spots and streaks mainly involving the coronary groove. Pericardial effusion may be present. Some animals also have secondary pulmonary edema or congestion, congestion in the liver, spleen and kidneys, and a modified transudate in the peritoneal cavity, and inflammatory lesions are occasionally found in other organs including the digestive tract. Cardiac lesions in chronic Chagas disease can vary, but the heart is often bilaterally enlarged and flaccid, with thinning of areas in the ventricular walls, in the terminal stages. Some dogs with CNS signs had meningeal congestion with unilateral cerebral swelling, or multifocal areas of dark red to brown discoloration in the gray and/or white matter of the spinal cord, but no gross lesions were evident in others.

Similar lesions have been reported in other species. Cardiac involvement has been described most often, but focal congestion and hemorrhages were found in a segment of the spinal cord of a horse with neurological signs. Trypanosomes were detected in this segment by histopathology. In some naturally- infected animals that were probably in the acute stage, there was inflammation associated with parasite replication in most or all tissues examined, including skeletal muscles and the reproductive tract (e.g., testes).

## Diagnostic Tests

Direct detection of *T. cruzi* or its nucleic acids is most likely to be successful during the acute stage of the infection. It can often be found in the blood, and occasionally in other samples such as cerebrospinal fluid, as well as in various tissues, especially the heart, at necopsy. The number of parasites later drops to very low levels, though intermittent parasitemia may occasionally be detected in chronically infected animals by PCR. However, consistently PCR-negative blood samples are not unusual at this stage.

Microscopic examination can often detect *T. cruzi* trypomastigotes and amastigotes in blood or stained tissues, respectively, during the acute stage. Concentration techniques, such as examination of the buffy coat (microhematocrit method) or the Strout method (double centrifugation) can increase the probability of finding the organism in blood. Where available, PCR tests can identify *T. cruzi* DNA directly in clinical samples. It should be noted that there are a few reports of some *T. cruzi* PCR tests amplifying other trypanosomes not known to cause any illness in animals, such as *T. rangeli* or *T. dionisii*. *In situ* hybridization was used to diagnose Chagas disease in one acutely infected primate at a research laboratory.

Xenodiagnosis was historically considered to be one of the most accurate tests for Chagas disease in South America, but it has mostly been superseded by other tests such as PCR. In xenodiagnosis, laboratory-reared, *T. cruzi*-free triatomine insects are allowed to feed on an infected individual, and the intestinal contents of the insect are examined for the parasite 1-2 months later. Insect availability generally limits xenodiagnosis to some South American reference centers at present. *T. cruzi* can also be isolated in various specialized media (hemoculture) and some cell lines such as Vero cell lines, but culturing this organism requires expertise and may take several weeks to several months. It can be recovered by animal inoculation (guinea pig, mouse or rat), though this is generally discouraged for animal welfare reasons.

Chronic infections are usually diagnosed by serology. Antibodies to *T. cruzi* can be detected with various serological tests such as indirect immunofluorescence, direct and indirect hemagglutination, complement fixation, ELISAs and radioimmunoprecipitation. Many of these tests were originally developed for use in humans, and validation for most animal species is limited or nonexistent. IFA is used most often for diagnosis in dogs. Cross-reactions with other parasites, particularly *Leishmania* and various pathogenic or nonpathogenic species of *Trypanosoma* (e.g., *T. evansi, T. rangeli*, *T. caninum*) can sometimes be an issue, and discordant results between serological tests are not uncommon. In addition, the presence of antibodies alone does not necessarily prove an illness is caused by *T. cruzi*, as many seropositive animals are asymptomatic. However, a high titer, combined with the typical clinical signs, is sometimes used for a presumptive diagnosis in dogs.

## Treatment

Animals with acute stage symptoms may be treated with the anti-parasitic drugs used in human Chagas disease, such as benznidazole. Other agents have also been tried occasionally. Antiparasitic drugs have generally had little or no effect in substantially altering the course of the disease in chronically infected dogs, though some studies have reported possible histological improvement, slightly better cardiac function or somewhat longer survival times.

The currently used drugs and protocols do not always clear the organism in either humans or animals, even when they are given during the acute stage and the clinical signs disappear. However, some experimental dosing protocols for benznidazole, such as prolonged, intermittent (e.g., once or twice weekly) treatment with higher doses seemed to be more promising during their initial testing in animals.

## 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*

Completely preventing contact with triatomine insects may be difficult or impossible in some regions, but exposure to these vectors can be reduced. Where feasible, animals can be protected by strict indoor housing in well-constructed homes or other facilities. Bringing them indoors at night, when most of these insects are searching for blood meals, might also be helpful. Kennels and other facilities that may harbor triatomine insects are often sprayed periodically with insecticides. Some flea and tick preventatives for dogs, such as fluralaner, also appear to be promising in reducing triatomine populations. Netting or other barriers may help restrict entry to some facilities, though triatomines can enter even through small gaps. Because *T. cruzi* can remain viable in dead insects for a time, any dead or dying triatomines found at facilities should be removed. In addition, carnivores should not be fed raw tissues that might contain *T. cruzi*, especially tissues from commonly infected wildlife hosts such as opossums.

Testing bitches for T. cruzi in breeding kennels has been suggested as a way to decrease the incidence of congenital transmission. One study reported that early prophylaxis of puppies with twice weekly doses of benznidazole might be beneficial in kennels where triatomine exposure is high, though consideration should be given to the possibility of promoting drug resistance with this approach, as there are only two approved drugs for treating Chagas disease in people.

## Morbidity and Mortality

Infections with *T. cruzi* are common among wildlife and domestic animals in most endemic areas, with surveys in both North and South America reporting significant exposure among dogs and cats, especially animals that are feral, housed in multi-dog kennels, reside in rural locations and/or are regularly exposed to wildlife, such as hunting dogs. Some nonhuman primate facilities also report high rates of seropositive animals. Practitioners in parts of the U.S. regularly see clinical cases in dogs, but there is little information about other species, such as cats or equids. A case in one horse in the U.S., together with a report of antibodies in 40% of horses tested, suggest that some illnesses in this species might be missed.

Whether animals always remain infected for life is currently unclear, though spontaneous cure has been reported to be possible in humans and experimentally infected opossums. The role of immunosuppression, if any, is also unknown; however, apparent reactivation of *T. cruzi* infection occurred in two rhesus macaques coinfected with simian immunodeficiency virus. Morbidity and mortality rates, as well as the proportion of infected animals that develops chronic disease, are still uncertain even in dogs. While 50% or more of infected dogs died during the acute stage in some experiments, they were given large numbers of parasites and are unlikely to reflect the situation in naturally infected hosts. Based on human data, some authors have suggested that morbidity rates around 5% might be more likely during this stage in dogs, and many of these cases are probably mild,. However, this estimate still needs to be evaluated, as there may be differences in host susceptibility, immune responses, usual parasite dose, routes of exposure and other factors. In addition, dogs are short-lived compared to humans, which might limit the expression of cardiac damage. Once chronic cardiac disease has been diagnosed, naturally infected dogs survived for 0 months to 5 years.

# Infections in Humans

## Incubation Period

The incubation period for acute Chagas disease is usually 5-14 days after exposure via triatomine bites, ranges from 3 to 30 days in foodborne outbreaks, and is often around

30-40 days, but can be as long as 3 months, after acquiring the parasites in a blood transfusion. Many people have no symptoms until the chronic stage, which can develop years to decades after they were infected.

## Clinical Signs

The acute stage of Chagas disease often passes without any clinical signs in humans. Some people may develop a lesion at the site of parasite entry, either a chagoma (a localized painless, erythematous induration) on the skin, or painless edema of one (occasionally both) eyes, often accompanied by conjunctivitis and local lymph node enlargement. The latter syndrome, called Romaña's sign, usually persists for 1 to 2 months. Systemic signs, if any, are often limited to a nonspecific flu-like illness with regional or generalized lymphadenopathy, and may be accompanied by enlargement of the liver and spleen. Some patients also have gastrointestinal signs (nausea, vomiting, diarrhea, epigastric pain) and/or edema, either generalized or localized to the face and/or lower extremities, and there are infrequent reports of cases with jaundice, hemorrhagic signs (hematemesis, hematochezia, melena, epistaxis), coughing, a transient morbilliform rash, or unusual complications such as hematospermia. Facial edema is particularly common in foodborne outbreaks, which often result in a high proportion of people with clinical signs, as well as more severe illnesses. The most frequently reported serious complications, which are rare except in some foodborne outbreaks, are subclinical or clinical myocarditis, with signs ranging from ECG abnormalities and arrhythmias to death, and meningoencephalitis. Most acute cases without serious complications resolve spontaneously within weeks to months.

The following stage, called the indeterminate form of Chagas disease, is a prolonged or indefinite period with no clinical signs except, in some cases, a mild sensory-motor peripheral neuropathy. Many people in this stage have no further symptoms and have a normal life expectancy, though histopathological changes may be found in some tissues. However, others eventually develop signs of organ failure, usually involving the heart, and/or digestive system, though this may take years to decades to become apparent.

Signs of cardiac involvement can include arrhythmias and conduction abnormalities, which may progress to heart failure. Left ventricular dilatation and dysfunction tends to be seen initially, but biventricular congestive heart failure is common in the later stages. Chagasic cardiomyopathy can be particularly difficult to treat, as it is highly arrhythmogenic, is often associated with rapidly progressive heart failure, and has a high incidence of stroke or pulmonary embolism due to the tendency to form cardiac aneurysms, together with atrial fibrillation. Sudden cardiac death is relatively common in these patients.

Gastrointestinal involvement is usually characterized by esophageal and/or intestinal motility disorders, which can progress to megaesophagus with symptoms of dysphagia (e.g., pain during swallowing, excessive salivation, regurgitation, chest pain) and/or megacolon, which is associated with severe, prolonged constipation, abdominal pain and asymmetrical abdominal distention. While small intestinal motility issues can contribute to signs such as constipation, small intestinal dilatation is rare. Some other complications that have been reported in Chagas disease include gall bladder abnormalities (mega-gallbladder or cholelithiasis), gastric ulcers or chronic gastritis, and lower urinary tract dysfunction with incontinence.

Immunosuppressed patients may have a similar pattern of disease; however, they are also at risk for reactivation of *T. cruzi* replication during the indeterminate stage, particularly when the immunosuppression is significant (e.g., transplant patients, some HIV-infected people). Reactivation may occur either subclinically or with acute stage symptoms, acute myocarditis, accelerated progression of existing chronic Chagas heart disease, meningoencephalitis and/or brain masses, as well as unusual syndromes such as fever with erythematous nodules or other skin lesions, or parasite invasion of the gastrointestinal tract and peritoneum. CNS involvement is particularly common in HIV-infected people with low CD4 counts, and brain masses seem to be limited to this population. Ocular disease (e.g., retinitis) has also been reported.

Women who are infected with *T. cruzi*, either with or without symptoms, sometimes give birth to congenitally infected infants. Many or most of these infants are asymptomatic at birth, but a few have overt clinical signs, and others may become ill over the next few weeks or months, or develop chronic Chagas disease later in life. Common signs in symptomatic infants include hepatosplenomegaly, jaundice, anemia and thrombocytopenia; while megaesophagus, megacolon and/or ocular disease (e.g., chorioretinitis) are possible but infrequent; and respiratory distress syndrome, meningoencephalitis and/or signs of acute myocarditis/ cardiac insufficiency can be seen occasionally in severe cases. Whether chronically infected women have an increased incidence of premature births, abortions, stillbirths or low birth weight infants is still controversial.

## Diagnostic Tests

The tests used to diagnose acute or reactivated infections in humans are similar to those in animals, and include observation of parasites by light microscopy, PCR tests, xenodiagnosis and culture, though the latter two tests are rarely needed for routine diagnosis. Loop-mediated isothermal amplification tests to detect *T. cruzi* nucleic acids are also available for humans. PCR tests can be particularly useful for demonstrating rising levels of nucleic acids during reactivated infections in immunosuppressed patients, as occasional positive PCR tests can also be seen in immunocompetent, asymptomatic people during the indeterminate or chronic stage.

Serology is most often used to diagnose chronic infections, screen pregnant women, or identify congenitally infected infants after maternal antibodies decline if PCR tests were unable to find the organism. It is sometimes negative or

  

inconclusive in people who are immunosuppressed. The most commonly used serological tests in humans are IFA, hemagglutination and immunoassays (e.g., ELISAs, chemiluminescent immunoassay), though other assays such as radioimmunoprecipitation or trypomastigote excreted-secreted antigen immunoblot (TESA-blot) may be employed in some areas, often as confirmatory tests. Rapid diagnostic tests such as lateral flow assays are also available. Because cross-reactivity to other microorganisms is possible and discordant results between serological tests are common, the use of two different tests (e.g., ELISA and IFA) based on different antigens is generally recommended to confirm the infection in people.

## Treatment

People in the acute stage of Chagas disease, congenitally infected infants and immunosuppressed patients with reactivated infections are usually treated with the antiparasitic drugs benznidazole or nifurtimox. While the clinical signs are cured and the risk of chronic complications reduced or prevented, some treated people, including those who are immunocompetent, can remain infected. Treatment of all adults in the indeterminate or chronic stage remains controversial, with conflicting reports on benefit, though children seem to respond well at any stage and are usually treated. Benznidazole and nifurtimox can have significant side effects, including some that are serious, such as neuropathy or bone marrow suppression. Some countries reserve nifurimox for those who do not respond to benznidazole due to a higher incidence of side effects.

Complications in the chronic stage, such as megaesophagus or megacolon, are managed according to standard protocols for these diseases. The treatment of chagasic cardiomyopathy is similar to the usual treatments for heart disease, though it must often be modified due to issues such as a high rate of arrhythmias.

## Prevention

Prevention of Chagas disease includes the avoidance of bites from triatomine insects, food contamination, congenital transmission and person-to-person transmission from 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 (though feeding during the day has been reported in times of food stress). In areas where the domestic cycle occurs, hiding places can be reduced by measures such as plastering walls or improving floors and roofing materials. Sleeping under an insecticide-impregnated bed net is also helpful. However, regular insecticide treatment of the home is also necessary in many areas, and is an important component of vector control programs to reduce or eliminate domestic triatomine populations in South and Central America. Other measures with varying effectiveness, such as insect repellents, thermal treatment of household belongings or light traps ae also used by some inhabitants.

Sylvatic and peridomestic triatomine species that opportunistically colonize human dwellings can be controlled or eliminated similarly, but they often return, and may even invade niches formerly occupied by insects in the domestic cycle. Where sylvatic triatomines enter homes only sporadically, as in some parts of the U.S., frequent inspection of hiding places (e.g., between mattresses, under bedding, in pet beds, behind bookshelves), might be sufficient for control. Window/door screens and other barriers can help reduce intrusions from these insects, and animal pens and storage areas, which may contain triatomines, should be kept at a distance from the home. Because these insects are often attracted to light, particularly white lights, measures such as minimizing outdoor lighting, closing the curtains at night and, changing white outdoor lights to yellow bulbs may also help. However, the ability of these bugs to enter homes through even thin cracks can make it difficult or impossible to completely prevent entry, even in modern, well-constructed homes. DEET, as well as some essential oils (e.g., mint, lavender, citronella), appear to be useful as repellents outdoors. Thick clothing, including shoes and socks, can also help prevent bites.

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, their feces and animals. Foods that might be contaminated, including meats, can be cooked to at least 45°C (113°F) or pasteurized.

While benznidazole and nitrofurimox are contraindicated during pregnancy, treatment of women who intend to become pregnant appears to reduce the risk of congenital infections. Infants born to infected mothers are also tested, and treated if necessary. Donor screening is important in reducing the risk of infections from blood transfusions. Leucocyte depletion, irradiation or freezing are also helpful, though not completely protective. People who handle potentially infected animals, carcasses, tissues or *T. cruzi* cultures (e.g., veterinarians, hunters, laboratory workers) should protect their skin and mucous membranes from possible parasites in blood or tissues, and clean/decontaminate the skin immediately if exposure occurs.

## Morbidity and Mortality

In many endemic regions, most people are thought to become infected with *T. cruzi* in childhood, often with few or no clinical signs. Living in substandard housing and/or participating in activities that increase contact with triatomine vectors increases the risk of exposure. In addition, about half of mothers first infected during pregnancy, and 1-5% of women in the indeterminate and chronic stages, are thought to transmit *T. cruzi* to their infants. Transmission seems to be more common if the mother is co-infected with HIV. Foodborne exposure is also significant in some areas, often in outbreaks that involve fruit juices or other foods prepared under unhygienic conditions.

The incidence of Chagas disease has decreased in recent years in South America, where interventions such as control programs for the domestic insect cycle, pregnancy screening and treatment of congenital infections have reduced transmission. However, the number of cases in some other regions, where most or all cases occur in immigrants from Latin America, may be stable or even increasing. Locally acquired infections are absent in Europe and Asia and appear to be relatively uncommon in the U.S., even among those who are regularly exposed to triatomine insect vectors. The reasons for this are unclear, though the absence of the domestic cycle and the delayed defecation habits of many triatomine insects might play a role.

Morbidity and mortality rates in Chagas disease vary with the health of the individual and other factors, particularly the specific complications that develop. Most people in the acute stage are asymptomatic or have mild symptoms, with estimates suggesting that about 5% of infections acquired via insect bites will become symptomatic and < 1% of these cases will be severe. Serious illnesses and deaths are more common in young children, the elderly and people who are immunocompromised. Morbidity and mortality rates are higher in all age groups during foodborne outbreaks, which tend to cause more serious illnesses, with an increased risk for cardiac or CNS involvement. Mortality rates were reported to be as high as 33% in a few of these incidents, but rapid treatment can prevent or significantly reduce the number of deaths.

People in the indeterminate stage who have no symptoms and no evidence of organ damage have a life expectancy similar to those who are uninfected. What proportion of them will carry the organism for life, if left untreated, is not known, though some long-term studies have demonstrated that spontaneous clearance is possible. Some estimates of disease progression suggest that from 20% to 35% of people in the indeterminate phase will eventually develop cardiac or gastrointestinal involvement, but other reports suggest a lower proportion, including a recent study of seropositive blood donors, which found that about 9% developed *T. cruzi*-related cardiomyopathy over 10 years.

Cardiac involvement in chronic Chagas disease is particularly serious, with a case fatality rate that is higher than in other forms of heart disease, due to the high incidence of arrhythmias and other complications. Occasional deaths are also caused by conditions such as volvulus of a dilated megacolon, or by CNS involvement, particularly in immunocompromised patients. Why this infection progresses to chronic organ involvement in some people, but not others, is unclear; however, it is known that, while cardiomyopathy occurs in all areas where *T. cruzi* is found, megaesophagus and megacolon are rare in some regions.

## Internet Resources

Centers for Disease Control and Prevention (CDC), U.S.

Global Chagas Disease Coalition

The Merck Manual

The Merck Veterinary Manual

World Health Organization. Chagas Disease

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