## CHAGAS DISEASE

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<th>Animal Group(s) Affected</th>
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<td>All mammals are susceptible to infection.</td>
<td>Contamination of blood-feeding lesion or mucous membrane by feces of insect vector; ingestion of infected vector, or food or water contaminated with bug feces; trans-placental or trans-mammary; blood transfusion</td>
<td>Dogs and humans-range from asymptomatic to acute myocarditis and sudden death. Chronic disease signs are related to cardio-myopathy; and in humans, mega-esophagus and/or megacolon can be seen.</td>
<td>Dogs and humans-variable; can cause severe disease or death. The degree to which wildlife reservoirs present disease is unknown.</td>
<td>No FDA approved treatment is available.</td>
<td>Prevent exposure to vectors; control vector populations; minimize wildlife reservoir access; blood donor screening; prevent seropositive female dogs from breeding.</td>
<td>Yes.</td>
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<td>Wildlife reservoirs include woodrats, opossums, armadillos, and raccoons. Disease is reported in dogs, humans, and non-human primates.</td>
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### Fact Sheet compiled by: Sarah A. Hamer

**Sheet completed on:** 4 September 2013

**Fact Sheet Reviewed by:** Tom Sidwa; Susan Montgomery

### Susceptible animal groups:
All mammal species are considered to be susceptible to *Trypanosoma cruzi*, including more than 150 species of 24 families that have been reported to be infected. Disease is best described from humans and dogs; the degree to which other domestic or wild animals present disease upon infection is unknown.

### Causative organism: *Trypanosoma cruzi* is a flagellated protozoan parasite that maintains many life stages. The parasite is spread by triatomine bugs. Triatomines are blood-sucking vectors commonly referred to as kissing bugs or cone-nosed bugs. After ingesting trypomastigotes from the blood of a vertebrate host, the bug’s hindgut contains epimastigotes which also can multiply in the vector. Metacyclic trypomastigotes appear in the insect’s rectum 8-10 days after infection. These metacyclic forms pass in the feces and can enter the body of a vertebrate host through the bite, scratched skin, or mucous membranes. Trypomastigotes are the abundant blood form that circulates in the mammalian host after infection. Amastigotes develop in muscle and other tissue cells and multiply by binary fission. Amastigotes differentiate into trypomastigotes which lyse the host cell and burst free and this stage can then attack other host cells. Pseudocysts of parasites may form in muscle cells.

### Zoonotic potential:
Many kinds of wild and domestic mammals serve as reservoirs for *T. cruzi*. This parasite can be bridged to humans from mammalian reservoirs through kissing bug vectors. Zoonotic potential is high in areas of Mexico and South and Central America, where kissing bugs maintain peridomestic cycles and colonize human dwellings. In contrast, the housing structures in US are generally less able to be colonized by bugs, and therefore zoonotic potential is reduced relative to areas with peridomestic cycles.
### Distribution:
Chagas disease in humans or animals can occur wherever there is overlap among kissing bug vectors, the *Trypanosoma cruzi* parasite, and vertebrate reservoir hosts. The disease is endemic in many areas of Mexico, South and Central America, and is increasingly recognized across the southern US. In the US, 11 species of kissing bugs occur, and are distributed across the southern half of the country and range as far north as the California/Oregon border and New Jersey. In Latin America, an estimated 12-19 million people were infected in the early 1990s, with an annual incidence exceeding 500,000. Since then, control campaigns have assisted in reducing the disease burden. The disease burden in the US is largely unknown due to lack of awareness, testing, and reporting. However, CDC has estimated that more than 300,000 cases of Chagas disease are found in US among immigrants from endemic countries of Latin America. The American Association of Blood Banks maintains The Chagas Biovigilance Network for reporting of screening and confirmatory results from the testing of US blood donors for antibodies to *T. cruzi*.

### Incubation period:
Once the metacyclic trypomastigotes enter the host, an acute local inflammatory reaction may occur. In humans, within 1-2 weeks of infection, the parasites spread to lymph nodes and multiply within phagocytic cells. The intracellular amastigotes multiply and pseudocysts may form. Within days, some organisms may transform to trypomastigotes and burst free from the pseudocyst. A generalized parasitemia can occur, followed by parasite invasion of many tissues within body. The incubation period may be up to several months if contaminated blood from transfusion is the source of infection.

### Clinical signs:
Chagas disease manifests as acute and chronic phases; in the absence of treatment, the host is infected for life. The chronic phase of infection has two forms, an indeterminate form during which the host is asymptomatic followed by development of clinical disease years to decades later. In humans and dogs, the initial acute phase of infection is usually asymptomatic or undetected; regional or generalized lymphadenopathy, fever, myalgia, headache, hepatosplenomegaly, edema, rash, vomiting, diarrhea, or anorexia may occur. Humans may note a lesion (chagoma) where the parasite enters the body. Severe manifestations, such as acute myocarditis or meningoencephalitis are rare. Chronic phase of disease may develop in a subset of human patients who survive the acute phase of infection. In chronic disease, cardiac abnormalities may be noted including right bundle branch block and left anterior hemiblock, atrio-ventricular conduction abnormalities, and arrhythmias. Megacardia may be noted on radiographs. In humans and dogs, systolic dysfunction is indistinguishable from dilated cardiomyopathy. Weakness and exercise intolerance may be noted. Humans with Chagas disease may also have complications of the digestive system, including megaesophagus and megacolon, with or without cardiac manifestations.

### Post mortem, gross, or histologic findings:
Gross cardiac changes may include megacardia and focal thinning of the myocardium including apical aneurysm. Dilatation and thinning of the wall of the esophagus and colon may occur. Histologically, in canines, examination of the heart may reveal unruptured pseudocysts with no inflammatory response, or ruptured pseudocysts with characteristic infiltration of lymphocytes, monocytes, and/or polymorphonuclear leukocytes.

### Diagnosis:
During acute infections, the trypomastigotes (blood stage of the parasite) may be identified by microscopy of a peripheral blood sample or through culture techniques; the organism has a single flagellum and a large kinetoplast at the posterior end of the cell and appears as a characteristic ‘C’ shape in Giemsa stains of bloodsmears. Additionally, PCR can be used to amplify the DNA of the parasite from a blood sample. Serologic tests may be of limited utility during acute infections. Because the level of circulating parasites decreases within months, parasites are undetectable in blood by most methods during the chronic phase of disease. During chronic disease, serologic tests are used to detect antibodies to the parasite. To increase sensitivity and specificity, a standard serodiagnostic approach is to apply two or more tests that use different techniques or different antigens. Two commonly used techniques are enzyme-linked immunosorbent assay (ELISA) and immunofluorescent antibody test (IFA). Some serological tests are cross-reactive and will also detect
antibodies to *Leishmania* species. Rapid immunochromatographic ‘dipstick’ assays have been developed for the detection of antibodies to *T. cruzi* in humans and dogs. While sensitivity and specificity meet or exceed the characteristics of other available tests, their use for Chagas disease diagnosis is considered experimental. Two tests are FDA approved for use as screening tests for human blood donations; most samples that screen positive are then subjected to a supplemental test with greater specificity. Blood donors who screen positive are notified of results, are urged to contact their physician, and are no longer able to donate blood. In chronic disease, particular ECG abnormalities combined with positive serology results can be highly indicative. The only parasitological techniques currently considered useful in the chronic phase of disease would be xenodiagnoses and hemoculture although it is no longer used in human diagnostics. In humans, PCR and IHC also are used and PCR would be considered more sensitive. Postmortem, heart or other tissues may be examined using histopathology for the amastigotes (tissue stage of the parasite) and associated inflammation.

**Material required for laboratory analysis:** Whole blood, plasma, serum, and/or cardiac tissue.

**Relevant diagnostic laboratories:**
Texas A&M Veterinary Medical Diagnostic Laboratory  
PO Box Drawer 3040  
College Station, TX 77841-3040  
(979) 845-3414  
(888) 646-5623  
http://tvmdl.tamu.edu/

*T. cruzi* rapid immunoblot assay  
Primate Diagnostic Services Laboratory (PDSL)  
Washington National Primate Research Center  
University of Washington  
Seattle Washington 98195-7330  
diagnostic@wanprc.org  
http://www.wanprc.org/pdsl/

**Treatment:** Although two antiparasitics can be used to treat human patients with Chagas disease (nifurtimox and benznidazole), these drugs are not approved by FDA so in the US, they are available only from CDC under investigational protocols. For both drugs, side effects are fairly common, and contraindications for treatment include severe hepatic disease and renal disease. However, antiparasitic treatment is indicated for all cases of congenital, acute or reactivated Chagas disease and for chronic *T. cruzi* infection in children. Treatment is recommended for adults up to 50 years old with chronic infection who do not already have advanced Chagas cardiomyopathy. For adults older than 50 years with chronic *T. cruzi* infection, the decision to treat with antiparasitic drugs should be individualized.

**Prevention and control:** In the absence of a human or veterinary vaccine and given the limited treatment options, prevention and control of Chagas disease across the Americas relies heavily on vector control and community education. Improvement of housing structures combined with insecticide treatment inside homes has significantly reduced peridomestic transmission of the *T. cruzi* parasite in Central and South America. To reduce the attraction of kissing bugs to homes or kennels, outdoor lights should be eliminated, and rodent habitat immediately surrounding the home or kennel should be removed. Screening of blood donations is an important public health tool for prevention of disease transmission through blood transfusion. Early detection and treatment of acute disease, including congenital cases, can reduce the burden of disease.

**Suggested disinfectant for housing facilities:** The duration of time the parasite can live outside a vector or host on environmental surfaces contaminated by bug feces is unknown, but the parasite will be destroyed by direct exposure to sunlight and other harsh environments. Surfaces that have come in contact with bugs or
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- **Notification**: States are not required by federal law to report cases of Chagas disease. However, Chagas disease in humans is reportable in 4 states: Arizona, Massachusetts, Tennessee, and Texas. Chagas disease in animals is reportable in Texas.

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<tr>
<th>Measures required under the Animal Disease Surveillance Plan:</th>
<th>N/A</th>
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<tr>
<td><strong>Measures required for introducing animals to infected animal:</strong></td>
<td>The risk of animal to animal direct transmission in the absence of the kissing bug vector is minimal. However, infected animals may increase the infection prevalence in vectors in a local environment. Efforts should be made to prevent seropositive female dogs from breeding due to congenital transmission.</td>
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<td><strong>Conditions for restoring disease-free status after an outbreak:</strong></td>
<td>N/A</td>
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### References