Disease: The etiologic agent of Chagas disease (American trypanosomiasis) is *Trypanosoma cruzi* (1). **Transmission:** *Trypanosoma cruzi* is usually transmitted by arthropod vectors. The insect vectors of this disease are triatomine bugs, which are also known as “kissing bugs.” The bug acquires *T. cruzi* by feeding on the blood of an infected animal or human and then passes the disease in its feces after each blood meal (1). The parasite can also be transferred by the fingers to other body sites such as the mucous membranes of the mouth, nose and eyes, but it cannot penetrate unbroken skin (6). In addition, Chagas disease can be transmitted through blood and organ transfusions, transplacental transfer from mother to fetus, and occasionally by ingestion of contaminated food or drinks (2). **Reservoirs:** The most common reservoirs for Chagas disease are armadillos, raccoons, opossums, and rodents (3). However, since these wild animals are usually killed or forced out of Chagas disease endemic areas by the presence of humans, there is an increase in the incidence of human and domesticated animal reservoirs. Examples of domesticated animal reservoirs include dogs, guinea-pigs, and rats (2).

**General Characteristics:** *Trypanosoma cruzi* is a hemoflagellate. It is a flagellated protozoan parasite that belongs to the order Kinetoplastida and the family Trypanosomatidae (4). *T. cruzi* takes on three main forms during its life cycle: the epimastigotes, then transform into the infective metacyclic trypomastigotes (circulating), which then differentiate into the intracellular amastigotes (reproductive) (6). **Key Tests For Identification:** Chagas disease can be identified in the acute stage by microscopic examination of the blood for motile *T. cruzi* parasites. To aid visualization, thin and thick blood smears are stained with Giemsa. The Chagas parasite can be isolated by inoculation inside an animal, grown in special media. This technique is called xenodiagnosis. To perform a xenodiagnosis, an uninfected triatomine bug is placed in a jar placed on the armpit of the individual with a suspected infection. The bug is allowed to feed for 30 minutes, removed, and its feces is examined 30-60 days later for the *T. cruzi* parasite. Although it is effective, there is difficulty in obtaining uninfected bugs for the purpose of xenodiagnosis, and most people are hesitant to be willingly bitten by a triatomine bug (8). In addition, one of the two serologic tests utilized for detection of the Chagas parasite is the indirect immunofluorescence test. An
indirect ELISA test is also used to detect antibodies in patients no longer in the acute stage of the disease. One drawback of the indirect ELISA test is that it may not properly distinguish between \( T. cruzi \) antibodies and those produced by other parasites (8, 9). **Signs and Symptoms of Disease:** There are two distinct phases of symptoms for Chagas disease: acute and chronic. Symptoms of the acute phase can include fever, body aches, fatigue, headaches, loss of appetite, diarrhea and vomiting. The signs can include mild enlargement of the liver or spleen, swollen glands, and local swelling (\( c \ hagoma \)) where the parasite entered the body. Romana’s sign is the most distinctive sign of Chagas disease, which is a swelling of the eyelids near the bite or where the feces was rubbed into the eye (6). The acute phase typically lasts a few weeks to a few months and eventually gives way to an asymptomatic chronic phase. During the chronic phase, the disease may lay dormant for decades, but it could manifest into more serious health problems, such as cardiomyopathy (enlarged heart), heart failure, altered rate, and sudden death due to cardiac arrest. Additional chronic phase problems include enlargement of the esophagus and colon that can make eating and bowel movements difficult (4,6).

**Historical Information:** Chagas disease obtained its name in honor of the Brazilian physician, Carlos Ribeiro Justiniano Chagas, who discovered the disease in 1909 (5).

**Virulence Factors:**

The parasite’s two main virulence factors include its life cycle and immune evasion. Within the host, intracellular phagocytosis allows amastigotes to proliferate inside tissue cells where the parasite is undetectable by the immune system. It has the ability to “cleave” antibodies present on its surface, thereby concealing its antigenic signal from the host’s immune system. Additionally, the short period of time the circulating trypomastigotes are present in the bloodstream also contributes to the parasite’s ability to avoid host defenses, essentially by not allowing enough time for the appropriate immunoglobulins to be activated and released (9). A large part of parasite’s destructive power inside the body is due to its ability to stimulate an autoimmune response. The body begins creating antibodies that attack epitopes shared by both the \( T. cruzi \) parasite and the normal host tissues most often in the heart and gastrointestinal tract. This enables \( T. cruzi \) antigens to create an autoimmune response that present a problem in developing a vaccine (4). In addition to stimulating an autoimmune response, the \( T. cruzi \) parasite also has the ability to cause immunodeficiency. Both the trypomastigote and the amastigote
secrete a protein known as TC52 that suppresses interleukin-2, therefore suppressing the proliferation of T-lymphocytes (4,6).

**Control/Treatment:** The Centers for Disease Control and Prevention and The World Health Organization uphold that the two main strategies for control of the disease are vector control and transfusion control. Vector control involves the spraying of residual insecticides to eliminate the disease from rural homes in endemic areas (6). Transfusional control involves the screening of blood for the Chagas parasite to ensure no contaminated blood is taken from donors. This was approved by the FDA on December 13, 2006 (2). Patients in the acute stage of Chagas disease are typically treated with either benznidazole or nifurtimox, which aid in elimination of the parasite. Young patients in the acute or intermediate stage of the disease are more likely to be cured. In contrast, the probability of parasitologic cure among adult patients (over 18 years old) with long-standing T. cruzi infections, most of whom were infected while young, is less than 10%, according to the CDC (4).

**Prevention/Vaccine Information:**

There is no vaccine for Chagas disease, however, there are antiparasitic and symptomatic therapy treatments (7). The two drugs available for antiparasitic treatment are Nifurtimox and Benznidazole. These treatments are usually most effective when given during the acute or early chronic phase of the disease (9). Symptomatic treatments are designed to treat people experiencing immunodeficiency who are in the chronic stage of the disease. Examples include pacemakers and medications to regulate the patient’s irregular heartbeat (4,7).

**Local Cases or Outbreaks:** According to the Texas Department of State Health Services, locally-acquired human cases in Texas are relatively uncommon. In 2014 and 2014, 39 human cases have been reported to the Texas DSHS, of which 24 were foreign-acquired, 12 were locally-acquired, and the location of acquisition was unknown for 3 cases (10).

**Global cases or Outbreaks:** About 6 million to 7 million people worldwide, mostly in Latin America, are estimated to be infected with T. cruzi, according to the World Health Organization (7). Annually, Chagas disease is

References


8. Bastien, Joseph. Testing and Treatment of Chagas Disease in *The Kiss of Death: Chagas*


<http://www.cdc.gov/dpdx/trypanosomiasisAmerican/dx.html>