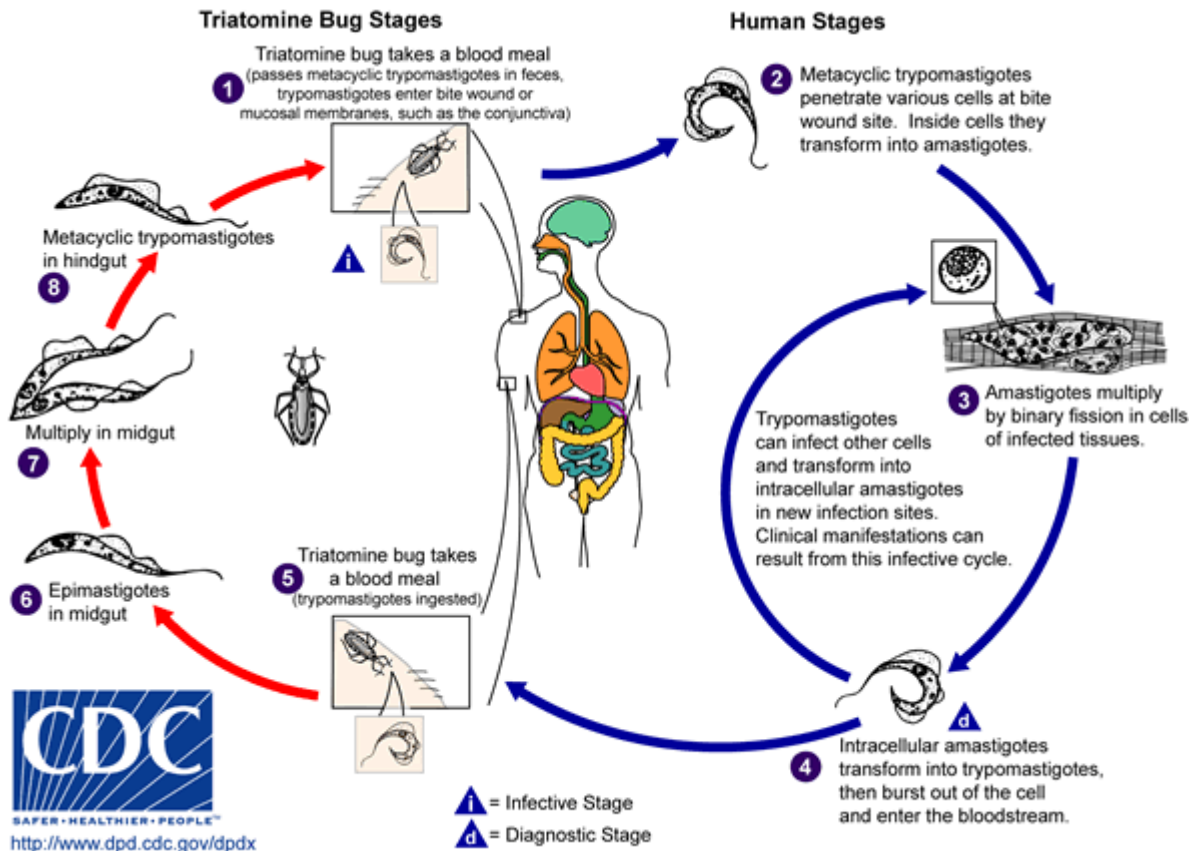


Trypanosomiasis, American

Causal Agent:

The protozoan parasite, *Trypanosoma cruzi*, causes Chagas disease, a zoonotic disease that can be transmitted to humans by blood-sucking triatomine bugs.

Life Cycle:



An infected triatomine insect vector (or “kissing” bug) takes a blood meal and releases trypomastigotes in its feces near the site of the bite wound. Trypomastigotes enter the host through the wound or through intact mucosal membranes, such as the conjunctiva **1**. Common triatomine vector species for trypanosomiasis belong to the genera *Triatoma*, *Rhodnius*, and *Panstrongylus*. Inside the host, the trypomastigotes invade cells, where they differentiate into intracellular amastigotes **2**. The amastigotes multiply by binary fission **3** and differentiate into trypomastigotes, and then are released into the circulation as bloodstream trypomastigotes **4**. Trypomastigotes infect cells from a variety of tissues and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle. The bloodstream trypomastigotes do not replicate (different from the African trypanosomes). Replication resumes only when the parasites enter another cell or are ingested by another vector. The “kissing” bug becomes infected by feeding on human or animal blood that contains circulating parasites **5**. The ingested trypomastigotes transform into epimastigotes in the vector’s midgut **6**. The parasites

multiply and differentiate in the midgut ⁷ and differentiate into infective metacyclic trypomastigotes in the hindgut ⁸.

Trypanosoma cruzi can also be transmitted through blood transfusions, organ transplantation, transplacentally, and in laboratory accidents.

Geographic Distribution:

The Americas from the southern United States to southern Argentina. Mostly in poor, rural areas of Central and South America. Chronic Chagas disease is a major health problem in many Latin American countries. With increased population movements, the possibility of transmission by blood transfusion has become more substantial in the United States.

Clinical Features:

A local lesion (chagoma, palpebral edema) can appear at the site of inoculation. The acute phase is usually asymptomatic, but can present with manifestations that include fever, anorexia, lymphadenopathy, mild hepatosplenomegaly, and myocarditis. Most acute cases resolve over a period of 2 to 3 months into an asymptomatic chronic stage. The symptomatic chronic stage may not occur for years or even decades after initial infection. Its manifestations include cardiomyopathy (the most serious manifestation); pathologies of the digestive tract such as megaesophagus and megacolon; and weight loss. Chronic Chagas disease and its complications can be fatal.

Laboratory Diagnosis:

Demonstration of the causal agent is the diagnostic procedure in acute Chagas disease. It almost always yields positive results, and can be achieved by:

- Microscopic examination: a) of fresh anticoagulated blood, or its buffy coat, for motile parasites; and b) of thin and thick blood smears stained with Giemsa, for visualization of parasites.
- Isolation of the agent by: a) inoculation into mice; b) culture in specialized media (e.g. NNN, LIT); and c) xenodiagnosis, where uninfected reduviid bugs are fed on the patient's blood, and their gut contents examined for parasites 4 weeks later.

Note: In certain circumstances, investigational molecular diagnostic tools, such as PCR, may be useful.

Diagnostic Findings

- Microscopy
- Antibody detection

Treatment:

Medication for Chagas disease is usually effective when given during the acute stage of infection. The drugs of choice are benznidazole or nifurtimox (under an Investigational New Drug protocol from the CDC Drug Service). In the chronic stage, treatment involves managing the clinical manifestations of the disease, e.g., pacemaker for heart block; the decision about whether to use antiparasitic therapy should be individualized in consultation with an expert.