

# CHAPTER 1

## Carlos Chagas

(1879–1934)

### Chagas Disease—A Kiss of Death

*Ecosystem disruption and subsequent loss of species have profound implications for humans. Damage to the ecosystem has caused changes in the equilibria between hosts, vectors, and parasites in their natural environment; for example, T. cruzi has switched from animals to humans as its primary host. In addition to global warming, acid rain, and pollution, Chagas disease warns us of a potential huge epidemic.*

Joseph Bastien

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The “kiss of death” is a macabre expression that brings back memories of Count Dracula and human-attacking vampires, along with tales of evil. The “kiss of death” is also a popularized term for Chagas disease, also called American trypanosomiasis, caused by a protozoan parasite and transmitted by “kissing bugs.” Chagas disease is endemic in Mexico and in South and Central America, putting 100 million people, about 25% of the population of Latin America, at risk. Its significance as a worldwide tropical disease is overshadowed only by malaria, a protozoan disease, and schistosomiasis, a worm disease. The epidemiology and biology of the disease are discussed later in the chapter.

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#### Microbe Hunter Carlos Justiniano Ribeiro Chagas

The disease is named after the Brazilian physician Carlos Ribeiro Justiniano Chagas (1879–1934), who first described it in 1909. Chagas stands out as the only researcher to describe all the major aspects of a new disease: pathogen, vector, epidemiology and clinical manifestations. He was born on July 9, 1879 in the town of Oliveira, Brazil;

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his parents were Jose Justiniano Das Chagas and Marina Candida Chagas. The family was well off and owned a small coffee farm. Their lineage traces back to descendants who emigrated from Portugal to Brazil in the mid-1600s. Carlos, who was one of four children, was 4 years old when his father died, leaving the responsibility of raising Carlos and his young siblings to his 24-year-old widow.

His early years were spent at a Jesuit boarding school, and, at his mother's urging, he planned to become a mining engineer. At the age of 14 he entered the School of Mining Engineering in Ouro Preto. It wasn't long before he realized that mining was not for him, and, partially influenced by an uncle who was a physician, he chose to study medicine. He entered into the Faculty of Medicine in 1897 at Rio de Janeiro and graduated with an M.D. in 1902. His thesis was "hematological aspect of malaria," and his mentor was Dr. Oswaldo Cruz, only 7 years his senior, a leading Brazilian parasitologist. Cruz had made his name in the fight against yellow fever and malaria in Rio de Janeiro and founded the Manguinhos Institute in Brazil (now named in his honor "The Oswaldo Cruz Institute") devoted to "preventative and sanitary medicine." The Institute was on its way to becoming a first-class facility for research and for the production of antiserum and vaccines.

Cruz offered Chagas an opportunity to work in the field of malaria at Manguinhos, but Chagas was attracted to the practice of medicine and elected to work (at a lower salary) in a hospital in Jurujuba in Rio de Janeiro. After a brief stint there, in 1905 he joined the Santos Docks Company in Sao Paulo, where the company was building port facilities, with the mission of eradicating a malaria epidemic. The antimalaria campaign focused on chemical agents to eradicate mosquitoes in houses and proved to be successful. In 1906 he joined Oswaldo Cruz at the Institute and worked there for the rest of his life.

In 1908 the Brazilian Government was attempting to connect an area in the Amazon region to Rio de Janeiro, but reminiscent of the building of the Panama Canal, which was halted on more than one occasion due to malaria and yellow fever, the railroad was not on track (pun intended). Based on his earlier success battling malaria, Chagas was commissioned by Cruz in 1909 to undertake a campaign against malaria in the small town of Lassance about 350 miles from Rio de Janeiro. The town served as the command post for the proposed railway; malaria was a formidable enemy, resulting in little progress on the railway project. He spent the next 2 years there living in a railroad car that doubled as a clinic.

Although Chagas was in Lassance to develop a campaign against malaria, he was on the trail of another disease that, ultimately, was to bear his name. He noted that some of the symptoms suffered by the railroad workers were not associated with malaria but, rather, appeared to him to be related to the bites of bloodsucking insects that harbored a protozoan flagellate-bearing organism in their hindgut. Frequently, the bugs sucked the blood at night from the face of their victims and, hence, earned the name "kissing bugs."

The protozoan parasite resembled the species of trypanosomes responsible for African trypanosomiasis, also known as “sleeping sickness,” which is transmitted by tsetse flies. (“Sleeping sickness,” sometimes approaching epidemic levels, has also been observed in the author’s classroom during lectures.) Chagas named this new protozoan *Schizotrypanum cruzi* (later renamed *Trypanosoma cruzi*) in honor of Oswaldo Cruz. Experimentally, it was shown that the parasite could be transmitted by infected kissing bugs to marmoset monkeys. In 1909 Chagas discovered the trypanosomes in the blood of a 3-year-old child and subsequently described 27 cases of the acute form and over 100 cases of the chronic form of the disease; these studies established the relationship between trypanosomes and a specific disease that came to be called Chagas disease. The original publication by Chagas in 1909 was entitled *New Human Trypanosomiasis: Studies About the Morphology and Evolutive Cycle of Schizotrypanum cruzi, Etiological Agent of a New Morbid Entity of Man*.

Oswaldo Cruz died in 1917, and Chagas succeeded his mentor as director of the Institute, a position he held over the next 17 years, in conjunction with an appointment as Director of the National Department of Public Health, until his own death in 1934 at the young age of 55. His contributions were numerous and highlighted campaigns against a variety of microbial diseases, including leprosy, Spanish flu, sexually transmitted diseases and tuberculosis; further, he advanced the cause of sanitation and hygiene.

Chagas’ identification of trypanosomiasis as a disease entity earned him international fame and established him as an eminent microbe hunter of the 20th century. His pioneering work has been recognized in his own country and internationally, as evidenced by the many prestigious awards and honors bestowed upon him, including election into The National Academy of Medicine in 1910. In 1912 he was chosen as the recipient of the Schaudinn Prize, awarded every 4 years in recognition of outstanding work in protozoology and tropical medicine. He was the recipient of the Great Prize of the Pasteur Centenary Commemorative Exposition. Harvard University awarded him an honorary doctorate—the first Brazilian recipient to be so honored. He also held honorary degrees from universities in Paris, Hamburg and Brussels. Although nominated twice for the Nobel Prize (1913 and 1928), he never received the award; some historians cite opposition from jealous colleagues to be the reason.

Chagas disease, also known as American trypanosomiasis, is the only protozoan disease described in this text. Protozoa, along with viruses, bacteria, fungi and prions, are microbes and, like fungi, are eukaryotic cells. Bacteria are described as prokaryotic cells; viruses and prions are acellular (i.e., less than cells). Eukaryotic cells are more complex forms and are distinct from prokaryotes in that they have a nuclear membrane and other organelle-bound structures. *Trypanosoma cruzi* is the infectious agent responsible for American trypanosomiasis.

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### The Life Cycle of *T. cruzi* and Direct Transmission of Chagas Disease

The cycle of Chagas disease in humans involves three organisms: *T. cruzi* as the parasite, Triatominae insects (kissing bugs) as the vector, and a human host. Trypanosomes reproduce asexually by binary fission; they measure 20 microns long and 3 microns wide and have a tail-like flagellum that serves as an anchor to the intestinal wall of its insect vector and as a whip, providing motility through the blood of an infected individual. A membrane surrounds the body giving the organism an undulating-like movement; they are fascinating to observe under the microscope. The parasite has three major forms, known as metacyclic trypomastigotes, amastigotes, stubby trypomastigotes, and epimastigotes, each of which is identified in the description of the cycle of Chagas disease, below.

The vector of American trypanosomiasis is called the kissing bug (Figure 1-1) because of its tendency to suck blood from unclothed parts of the body, particularly at night, and “kissing” its host’s eyelids, ears or lips with its sucking mouth parts. They are known by a variety of colloquial names, depending on the country, including cone-nosed bugs, Mexican bed bug, vinchuca, Reduviid bugs (Reduviidae family) and barbeiros. In the United States most kissing bugs are in the genus *Triatoma*. Bugs harboring trypanosomes are not harmed by the parasite (unlike the case in typhus [see Chapter 3] where body lice, the vectors, become infected and die). There are a number of significant species, of which *Triatoma infestans* is a major one.

The parasite undergoes a sequence of morphological changes in the gut of the insect. Kissing bugs are large (2 to 3 cm in length) obligatory blood-feeders that feed on a variety of mammals, including opossums, armadillos, deer, foxes, livestock, rodents, cats and dogs, all of which serve as natural reservoirs; opossums and armadillos are the most common reservoirs for *T. cruzi*. People who are fed on describe the process as painless and accompanied only by a mild tingling. Feeding takes approximately 10 minutes and is repeated at about 3-week intervals. The insects are able to survive for 3 to 6 months between feedings. Although unusual, severe allergic reactions to proteins in the insect’s saliva can occur, possibly resulting in death due to anaphylactic shock. Although not unique to Chagas disease, infection is linked to poverty and poorly constructed houses. The bugs are light sensitive and live in thatched roofs (Figure 1-2), crevices, cracks and other dark places in the mud walls characteristic of adobe huts and are frequently found in the bedding and bed frames, favoring transmission.

In the early days of the discovery of the disease it was thought that the trypanosomes were in the saliva of the insect vector and injected into the skin by the insect’s mouthparts. It is now known, however, that the trypanosomes reside in the hindgut of the insect. While feeding, the insect defecates, thereby depositing the (metacyclic) trypomastigotes on the skin. The accompanying itchiness leads to



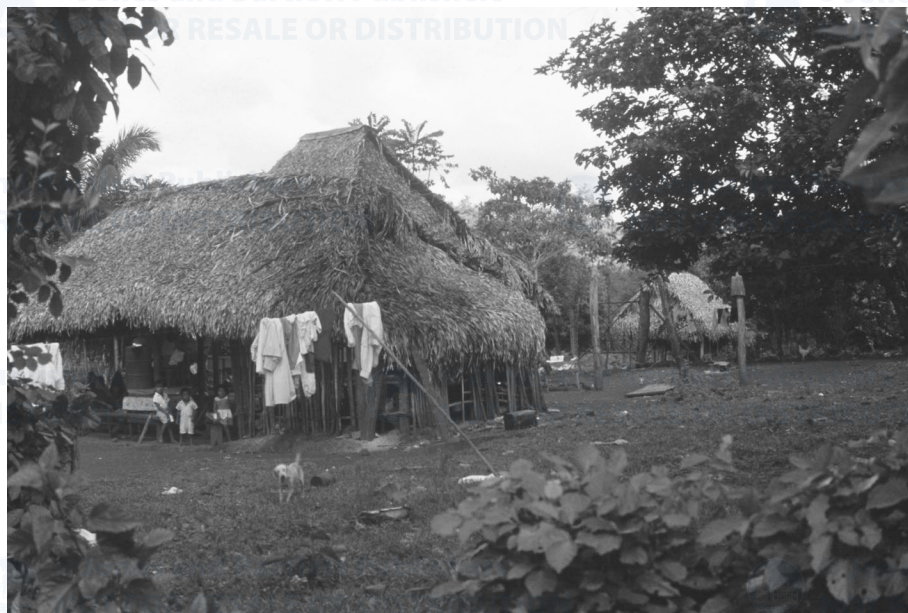
**Figure 1-1** A triatomid, “kissing bug,” the vector of *Trypanosoma cruzi*. (Courtesy of World Health Organization/CDC.)

scratching and rubbing of the area, resulting in inoculation of trypanosomes into the skin and their subsequent invasion into the blood. The habits of the vectors are significant in their infectivity. For example, studies have shown that *Triatoma infestans* defecate soon after taking a meal, whereas other species wait 20 to 30 minutes, after which time the insect has no further contact with the person.

A number of years ago the author spent 3 months as part of a small group studying tropical diseases in Central America and in Mexico under the auspices of the U.S. Public Health Service and the Louisiana State University School of Medicine—Inter-American Training Program in Tropical Medicine. In San Jose, the capital city of Costa Rica, the group made a field trip to a rural village on the outskirts of San Jose in which Chagas disease was endemic. The village was spotted with one-room, dirt floor, thatched-roof adobe huts. These huts were generally occupied by five or six children and two, or more, adults and had neither bathroom nor cooking facilities. In one particular hut, occupied by a family of eight, we searched for the presence of kissing bugs. The only item in the hut was a broken-down bed frame with a sheet of cardboard over a dilapidated bed spring partially covered by an old and ragged blanket. The “bug hunt” was easy: Within a brief period of time close to 100 bugs were



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**Figure 1-2** A village with thatched roof huts; this type of structure is a habitat for triatomids. (Courtesy of Robert I. Krasner.)

collected, primarily around the bed frame. Subsequent laboratory analysis revealed that approximately 60% of the bugs were infected with *T. cruzi*.

### Cycle of Chagas Disease

The cycle of Chagas disease is a function of the interplay of the parasite, vector and host (Figure 1-3). Metacyclic trypomastigotes are present in the feces of the insect vector and gain entrance by the host scratching at the wound site. Painful nodular swellings, called chagomas, sometimes develop. Next, the parasite invades into cells where it transforms into amastigotes and cluster-forming cysts. These intracellular amastigotes cause serious damage, resulting in pathological conditions described below. Amastigotes erupt from invaded cells and spread to other tissue cells, during which time rapid proliferation by binary fission takes place. Amastigotes evolve into stubby trypomastigotes that are released into the host's blood. If the insect takes a blood meal, the trypomastigotes travel to the insect's midgut, where they transform into epimastigotes and multiply. Further development into infective metacyclic trypomastigotes occurs, completing the cycle. Residence in the insect is anywhere from 6 to 16 days. The transformation exhibited by trypanosomes is an example of a highly developed adaptive strategy.

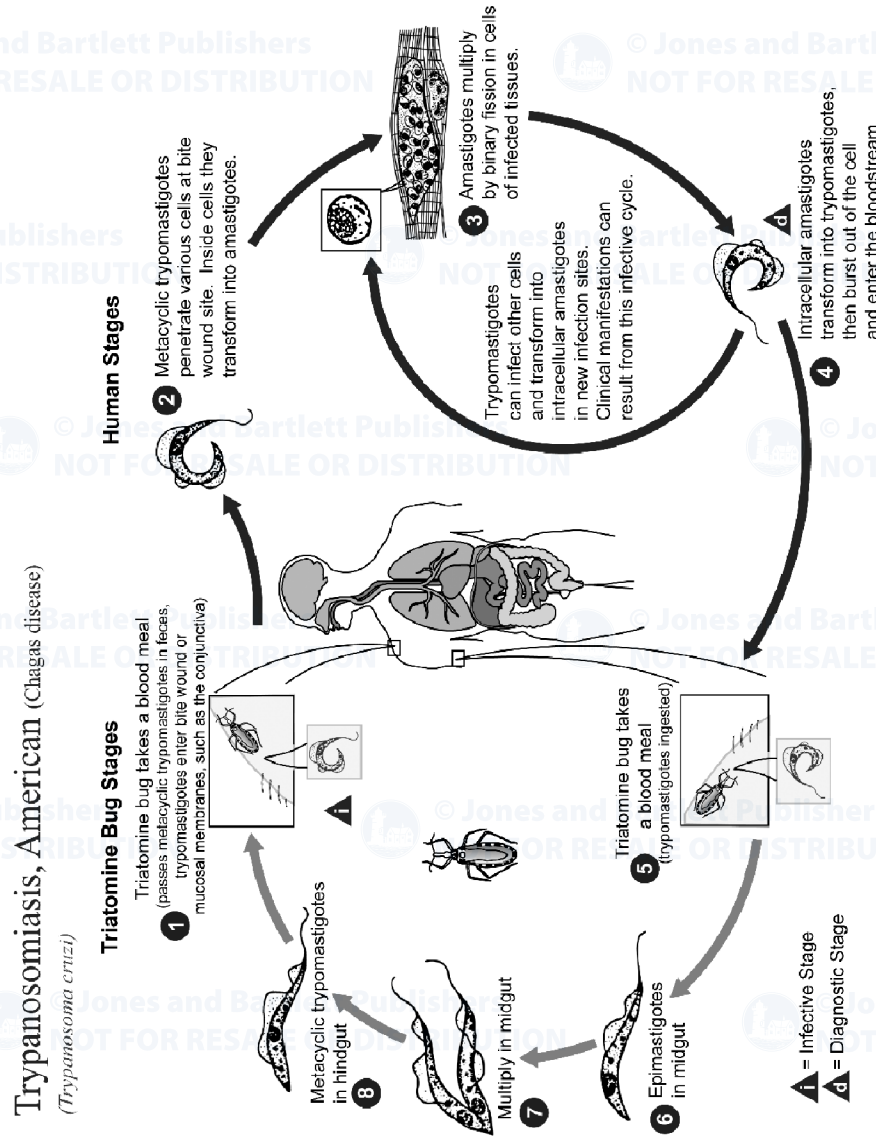


Figure 1-3 The cycle of Chagas disease, involving interplay between a human host, disease vector, and parasite. (Courtesy of Alexander J. da Silva, PhD/Melanie Moser/CDC.)

**8** Chapter 1**Indirect Transmission of Chagas Disease**

Although the primary mechanism of transmission is direct (i.e., vector to host), alternate mechanisms play a role in the epidemiology of Chagas disease. In parts of Central America and in Mexico, where the disease is endemic, infected persons are responsible for transmitting the disease into the population through blood transfusion. Immigration of people from *T. cruzi*-endemic areas and increased international travel have caused concerns about the potential for transfusion-acquired Chagas disease. In 1998 the Centers for Disease Control and Prevention reported five cases of Chagas disease in North America traced to transfusions.

Solid-organ transplantation in the United States resulted in five cases of acute Chagas disease in heart transplant recipients. The last two cases were reported by two Los Angeles county hospitals in February 2006. The first case was in December 2005 in a 64-year-old man who received a heart transplant. Subsequent investigation revealed that the organ donor was positive for *T. cruzi*; he had been born in the United States but had traveled to a Chagas disease-endemic area of Mexico. Three patients, respectively, received a liver and both kidneys from the same donor and, to date, show no evidence of infection; they continue to be monitored. A second case occurred in January 2006 in a 73-year-old man who had received a heart transplant. A few months later he became ill, and *T. cruzi* trypomastigotes were present in his blood. The source of infection turned out to be an organ donor who had been born in El Salvador and was residing in Los Angeles at the time of his death. Three other patients received solid organs from the same donor and continue to show no evidence of harboring trypanosomes; they continue to be monitored. Because organ donors may have received blood transfusions, infections can be transmitted to recipients either by transfusion or by transplant.

Currently, there are no screening tests in the United States for *T. cruzi*, and the risk of getting the disease may be higher than the risk of getting acquired immunodeficiency syndrome or hepatitis B and C. Progress is being made in some countries of Latin America to screen the blood supply. Organs sold on the black market by poverty-stricken and desperate people are a potential risk for American trypanosomiasis.

Studies of outbreaks have documented that trypanosomiasis can also be acquired by the oral route. In 1991 farm workers in Brazil were infected with food containing opossum feces; several years later an outbreak occurred in which 17 people were infected by drinking assai palm fruit juice believed to be contaminated with triatomids crushed in the juice pressing. More recently, in March 2005, several people in Santa Catarina, Brazil acquired Chagas disease as a result of consuming sugar cane juice at a local roadside kiosk. As in the previous case, transmission occurred by infected trypanosome-bearing insects. Just as food can carry triatomid feces infected with trypanosomes, so too can fingers that have been in contact with trypanosome-bearing feces, resulting in possible transfer into eyes, skin and mouth. Finally,



Chagas disease can be transmitted from mother to fetus by transplacental transfer and by breast milk.

### Pathology and Epidemiology

For those who develop chronic infection, life expectancy is decreased by an average of 9 years. Chagas can cause serious illness and is a major cause of death worldwide due to congestive heart failure. The infection progresses in two stages: the acute stage and the chronic stage. The acute stage primarily manifests in children and is accompanied by symptoms that are not unique to Chagas disease and so go unnoticed. Symptoms may include fever, swelling of lymph glands and enlargement of liver and spleen; in about 1% to 2% of the acute cases a swelling occurs around the eye and on the eyelid, referred to as Romaña's sign (**Figure 1-4**). The more serious chronic stage develops in about 30% of those infected but may not occur for as long as 30 to 40 years later. The chronic stages includes the possibility of neurological disorders, enlargement of the bowel and esophagus (with associated difficulty in swallowing), and, most significantly, damage to the heart, which occurs in about 40% of those infected. Left untreated, the disease can be fatal, primarily due to cardiac damage leading to heart failure.



**Figure 1-4** Romaña's sign found in some cases of Chagas disease, characterized by swelling around the eye and on the eyelid. (Courtesy of the WHO/TDR.)

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The following case study was described in *Kiss of Death—Chagas' Disease in the Americas* by Joseph William Bastien:

Bertha (psuedonym) lives in La Paz, Bolivia, and her medical history provides insight into the effects of Chagas. She suffers from chronic heart ailments from Chagas disease.

As a child living in the 1930s, she was bitten by vinchucas (“kissing bugs”) and infected with *T. cruzi* when she lived in Tupiza, a small rural village in Bolivia. She later married and bore four daughters. In 1960, she moved to La Paz after her husband abandoned the family. She made a living sewing for wealthy people, but in 1974 she was diagnosed with Chagas disease.

She tells the story of her life and how she copes with Chagas. Until she was forty-four she was healthy, going up and down the hills of La Paz to do her sewing. In 1974 she felt fatigue. She began to get a swollen throat and spit blood. She didn't know what it was; she had no idea it had to do with the vinchucas bites years before. She would get tired, fatigued and experience dizzy fainting spells. She continued to do her sewing though she sometimes would faint while she was working. The fainting spells continued for a year; the next year her fainting got more severe and she eventually suffered a stroke. Her children took her to a doctor, Dr. Jauregui, who hospitalized her. She underwent testing, xenodiagnosis, that indicated she had Chagas disease. X-rays showed that she didn't suffer from cardiomegaly (an enlarged heart), but that she probably had lesions in her heart's electrical system. These were caused by *T. cruzi* amastigotes being encysted in her cardiac tissue. This condition can be fatal.

Dr. Jauregui implanted a pacemaker in 1980 when Bertha's heart rhythm worsened. The pacemaker keeps the heart rhythm constant and Bertha's condition improved. She was able to resume her seamstress work, although she suffered minor fatigue as she climbed the streets of La Paz at 12,000 feet. (Reprinted courtesy of the University of Utah Press.)

Regarding diagnosis of Chagas disease, xenodiagnosis is an old, but still used, procedure. The test sounds like it is out of the Dark Ages and is performed by placing uninfected triatomid bugs in a jar that is turned upright and placed on the skin, allowing the insects to feed for 30 minutes. Their feces are then examined for the presence of trypanosomes at 30 days, 60 days and whatever other intervals are prescribed. Needless to say, the test, although reliable, is not popular; the thought of having live insects sucking blood is disgusting. Other immunological diagnostic procedures are also used as indirect tests to detect the presence of antibodies against trypanosomes; the presence of these specific antibodies is proof of infection by *T. cruzi*.

It is thought that Charles Darwin may have died of Chagas disease, an idea advanced by Professor Saul Adler of the Hebrew University, a recognized expert in trop-

ical diseases; he pointed out that Darwin had been in contact with *Triatoma infestans*, a principle carrier of Chagas disease, on a number of occasions in South America. Darwin describes in the *Voyage of the Beagle* an encounter with a “bug” at night: “I experienced an attack (for it deserves no less a name) of the Benchucha, a species of Reduviid, the great black bug of the Pampas. It is most disgusting to feel soft wingless insects about an inch long crawling over ones body. Before sucking, they are quite thin but after become round and bloated with blood.” Some claim, however, that Darwin’s poor health, which commenced about 5 years after his experience as a naturalist on the frigate *Beagle*, was psychosomatic.

American trypanosomiasis affects 16 to 18 million people worldwide, and estimates are that 50,000 will die each year. The disease remains a major public health problem throughout Latin America, with estimates that more than 10 million in that population are infected and 1 million will die at the current rate, pointing the need for new measures of prevention, diagnosis and treatment. More than 100 million people, about 25% of the population of Latin America, are at risk for infection. Further, the disease is not limited to Latin America because another 100,000 chronically infected immigrants now live in the United States, in Canada, and in Europe, increasing the potential for transfusion and transplantation-acquired Chagas disease. The condition extends from the southern United States through Argentina, focused primarily on Central and South America.

### Treatment and Prevention of Chagas Disease

In general, drug therapy for Chagas disease has not yet “arrived,” although two medications, nifurtimox and benzadiazole, are effective when given during the acute and early chronic phase of infection. The earlier the diagnosis is made and the earlier treatment is implemented, the greater the chances of recovery. A diagnostic problem is that only a small number of people show specific signs of infection (Romaña’s sign), but many then go on to develop serious illness as they progress years later into the chronic phase. The antiparasitic drugs are highly toxic, cause serious side effects and are expensive, all factors that limit their use.

Once the chronic stage is reached the disease is not curable, and treatment is focused on management of the patient’s symptoms. Heart disease resulting from Chagas disease can be handled by heart transplantation, although there is uncertainty in the medical community about the wisdom of transplantation because the heart recipient would be on immunosuppressive therapy, affording increased opportunity for infection.

Prevention, as is the general rule in microbial disease, is the best means of maximizing control. Vaccines are not available; one was developed in the 1970s that used “pieces” of the parasite but proved to be impractical because of the high cost and other factors. Several research groups are currently working on preventive immunizations.

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A significant problem is that some of the *T. cruzi* proteins (antigens) are similar to human tissue proteins, possibly leading to a problem of autoimmunity, meaning that parasite proteins can stimulate the production of antibodies that react with tissue proteins, leading to their destruction.

Reducing the kissing bug population is the focus of preventive measures and is accomplished by the use of insecticides. Over the past 30 years, urbanization has resulted in a shift in the epidemiological pattern of the disease from a rural disease to a suburban and urban disease that can be transmitted by blood transfusion. Control measures are aimed at preventing the insects from gaining access into houses. National projects with community participation in building better houses made of solid adobe, plaster walls and zinc roofs to replace thatched roofs aim to minimize entrance and nesting of kissing bugs in houses.

### Progress in the Fight Against Chagas Disease

In 1991 the governments of six Southern Cone Countries of South America (Argentina, Bolivia, Brazil, Chile, Paraguay and Uruguay) launched the Initiative for the Southern Cone Countries aimed at eliminating *Triatoma infestans* and at screening blood transfusion donors. The Initiative has been successful; more than 2 million houses in rural areas have been sprayed. Target dates for interruption of transmission were set for each of the six countries ranging from Uruguay in 1997 to Paraguay in 2005. Following suit, the Andean Countries (Colombia, Peru, Ecuador and Venezuela) Initiative was established in 1997 with similar goals of eradication by 2010. In 1997 the Central America Initiative was formed. These Initiatives are in accord with the 1998 World Health Assembly Resolution control strategy for elimination of Chagas disease over the period 1996 to 2010. Venezuela and Colombia are in the midst of providing better houses constructed to minimize vectorial transmission. Parallel significant progress in reducing transfusion transmission has made headway. According to the World Health Organization the following major achievements have been accomplished:

- Reduction of 72% is seen of the incidence of human infection in children and young adults in the countries of the Initiative of Southern Cone.
- In 1997 Uruguay is certified free of vectorial and transfusional transmission of Chagas disease.
- In 1999 Chile is certified free of vectorial and transfusional transmission of Chagas disease.
- In 2000 10 of 12 endemic states of Brazil were certified free of vectorial and transfusional transmission.

In December 2006 the U.S. Food and Drug Administration approved a new test to screen blood for the presence of *T. cruzi* antibodies. The test is not required by all

blood centers, but in January 2007 the American Red Cross and the Association of Blood Banks (the two agencies responsible for 65% of the nation's blood supply) implemented the test. It is expected that the U.S. Food and Drug Administration will require screening in the near future.

To defeat a disease it is necessary to understand it in all its dimensions. Biologists and medical scientists are becoming increasingly aware that strategies to control diseases must take into account societal factors, including religious beliefs, cultural habits and ethnic customs. For example, the battle against Guinea worm (see Chapter 10), a disease nearing eradication, has been hampered by the belief that the souls of ancestors reside in bodies of water that are known to be a source of the disease, prohibiting the use of insecticides to rid the water of infective worm larvae. Further, the practice of cannibalism in the Fore Tribe of New Guinea that resulted in Kuru perpetuated the existence of that disease (see Chapter 7). In the case of Chagas disease, there is strong resistance on the part of some Andean natives to the use of insecticides; these natives consider insects as life forms not to be destroyed, and the presence of kissing bugs is considered a sign of fertility. The fact that symptoms of Chagas disease may not occur for 30 to 40 years after exposure to infective *Triatoma* feces makes it difficult to accept a causal association between exposure and disease.

There is no doubt that societal factors that hinder disease control can be as difficult, and maybe even more difficult, to solve than biological factors. But there is no doubt that Chagas disease, a neglected and forgotten epidemic, needs to be addressed without further delay. Chagas disease, along with other neglected diseases, affects populations primarily in developing countries. The not-for-profit organization Drugs for Neglected Diseases Initiative seeks to develop drugs that are affordable and suitable for the "poorest of the poor" and lists Chagas disease in the category of the "Most Neglected Diseases."

In 2005 the genome of *T. cruzi* was deciphered, allowing new strategies to prevent and to treat Chagas disease. To add further emphasis for research and public health in an effort to reduce the incidence of Chagas disease, it is now recognized as an opportunistic infection and coinfects individuals with acquired immunodeficiency syndrome; this may further Carlos Chagas' dream of eliminating the disease that bears his name. He would be proud to witness the Oswaldo Cruz Institute as now a leading institute for biomedical research in Latin America. The Institute covers all major areas in the biomedical arena, especially those related to the basic aspects of parasitic and infectious diseases.

### The Legacy of Carlos Chagas

Chagas died of a heart attack at the young age of 55. His life and his achievements to combat microbial disease are an inspiration to all who know of him. Chagas' detective-like work, leading to a description of the causative agent, vector, clinical manifestations,



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epidemiology and the animal reservoirs and of a particular protozoan disease earns him a unique place in medical history. One year before his death, in addressing a group of graduate doctors, he remarked, “gentlemen, the hygienic practical applications in Tropical Medicine have destroyed the prejudice of a terrible climate; the scientific method has conquered the tropics unhealthiness.”

Thank you, Carlos Justiniano Ribeiro Chagas.

**Suggested Readings**

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