Chagas disease (American trypanosomiasis) was named after the Brazilian physician Carlos Justiniano Ribeiro Chagas, who in 1909 announced to the world the discovery of this new parasitic disease in animals and humans, in the town of Lassance, State of Minas Gerais, Brazil. In 1908, Chagas observed, for the first time, flagellate forms of the parasite in the intestine of the hematophagous bug *Panstrongylus megistus* (initially called *Conorhinus megistus*), which he found residing in human dwellings in Brazil. A few months later, he studied the parasite by experimentally infecting monkeys, rodents, and dogs. At the beginning of 1909, Chagas discovered the same flagellate in the blood of a cat and in a 2-year-old girl and realized that he had discovered a new disease-causing agent, transmitted by hemipteran insects in the family Reduviidae, subfamily Triatominae. He named the new trypanosome *Schizotrypanum cruzi*, which was later renamed *Trypanosoma cruzi*. The enzootic condition of the new trypanosomiasis was also demonstrated by Chagas after he found a natural infection in an armadillo (*Dasypus novemcinctus*) and a bug (*Panstrongylus geniculatus*) sharing the same burrow (Chagas, 1909a, 1909b, 1912; Coura, 1997).

According to the classical WHO data, it was estimated that Chagas disease affected 16–18 million people with at least 100 million at risk of contracting the infection in 21 countries throughout Latin America. There were an estimated 1 million new cases of chronic disease and some 45,000 deaths annually (WHO, 1991, 1995). Recent data indicate that these figures have been reduced drastically to less than 10 million, mainly due to the action of the various control “initiatives” throughout Latin America. Marked reductions in incidence and prevalence have been observed since the Southern Cone Initiative was launched in 1991, with consequent important savings in healthcare expenditures for the countries (Moncayo and Ortiz-Yanine, 2006; Schofield et al., 2006; Yamagata and Nakagawa, 2006). Likewise, the efforts toward the total elimination of *Rhodnius prolixus* from the Central American subregion, a target of the Central American Initiative, have been extremely successful (cf. Zeledón et al., 2008). In fact, the transmission of
Chagas disease by *R. prolixus* is presently considered to be interrupted in the entire subregion, due to its apparent elimination (OPS, 2011).

In the United States, the parasite was first observed in California in another species of bug, *Triatoma protracta*, a few years after Chagas’ initial observation (Kofoid and McCulloch, 1916). Nevertheless, when these authors failed to find the blood forms in the natural host of the bug, the wood rat (*Neotoma fuscipes*), they thought that the flagellate they had found was a different species and named it *Trypanosoma triatomae*. Furthermore, according to Kofoid and Donat (1933a) at that time, they were unable to transmit the parasite to laboratory albino rats through the bite of the infected insects. Later on, Kofoid and Donat (1933a, 1933b) succeeded in infecting laboratory and wild *Neotoma* rats plus one opossum (*Didelphis virginiana*) through *T. protracta*-infected feces, demonstrating that the previously observed flagellate present in the bug was indeed the same trypanosome described by Chagas in Brazil several years earlier. These authors stressed the fact that, of the six subspecies of *N. fuscipes* present in California, they found that the bugs (*T. protracta*) in *Neotoma fuscipes macrotis* and *Neotoma fuscipes annectans* nests, but only those associated with the former wood rat were infected with *T. cruzi* (Kofoid and Donat, 1933b).

The experiments were extended by Wood (1934a, 1934b), who proved that in fact *Triatoma protracta* and *N. fuscipes* are natural hosts of the parasite, that it is possible to experimentally infect different mammals (including rhesus monkeys and dogs), that amastigotes are formed in tissues of the infected animals, and that the infections tend to be light, suggesting a low virulence of the trypanosome.

Also in the 1930s, *T. cruzi* infections were discovered in other species of bugs such as *Triatoma uhleri* (also known as *Triatoma rubida*) in Arizona (Kofoid and Whitaker, 1936) and *Triatoma sanguisuga* and *Triatoma gerstaeckeri* in Texas (Anonymous, 1938; Packchanian, 1939). Additional species were also later found infected as indicated below. An interesting antecedent is that *T. sanguisuga* was reported from the state of Georgia as early as 1855 by Le Conte, who made the observation that people, particularly children, were bitten by the bug there (Le Conte, 1855). The same species was confirmed in Georgia by Stal (1859) a few years later. Similarly, this species was found in beds and reported to bite humans in Illinois in counties such as Madison, Jersey, Union, and Adams (Walsh and Riley, 1869). Uhler (1876, 1878) made reference to *T. sanguisuga* as
inhabiting Virginia, Maryland, Ohio, Texas, Florida, and Illinois and pointed out that it was a “blood-thirsty tenant of the beds in houses.” Ryley and Howard (1892) presented evidence of Conorhinus sanguisugus (T. sanguisuga) biting humans in Missouri and Oklahoma (Indian Territory). In the latter place, the bugs were in a bed in a log house, and apparently representatives of the species were also reported in the forests. Kimball (1894) found this bug, in large numbers, in poultry houses and in barns attacking horses, and occasionally in houses in Manhattan, Kansas, causing serious allergic reactions in people. A similar situation was pointed out by Marlatt (1896) in parts of Texas and Kansas, where the insect was a frequent visitor of homes.

John Lembert made observations of humans bitten by T. protracta in the Yosemite Valley in the 1860s (Mortensen and Walsh, 1963). Thurman (1944) mentioned the first finding of Triatoma neotomae in Texas by Schwartz in 1898, even though the specimens were not properly identified at that time.

In 1899, there were several nationwide newspaper releases, originating with the story of a lady from Washington D.C. who developed a severe reaction when bitten on the face by one of these insects (T. sanguisuga). This seems to be the origin of the common name “kissing bug,” used for the first time on that occasion (Howard, 1899; Shields and Walsh, 1956). Other common names found in the American literature are cone-nosed bug, bloodsucker, Mexican bed bug, China bug, and assassin bug.

Stal (1859) makes reference to other species of the same group being present in the United States, including C. gerstaeckeri and C. variegatus (aka Triatoma lecticularia) in Texas. Uhler (1876) also mentions the presence of the latter species in California, Georgia, Louisiana, and Illinois. Ryley and Howard (1893) reported that in Washington County, Florida, T. lecticularia “frequently fly into houses.” Howard (1899) added that C. protracta was present in California, Arizona, and Utah and included Missouri in the distribution of C. sanguisuga.

Collectively, these reports demonstrate that triatomine vectors of Chagas diseases have existed in the United States, under wild conditions, for many centuries, and that some species have been associated with human dwellings for a long time, causing allergic reactions of varying degrees in people. Also, Barnabé et al. (2001), on the basis of phylogenetic studies, are of the opinion that T. cruzi not only is native to the
United States but also has been part of the native fauna for a very long time. Interestingly, Reinhard et al. (2003) suggest that a prehistoric mummy from the Chihuahuan Desert, at the Texas-Coahuila border, might represent an ancient North American case of Chagas disease. This individual presented intestinal alterations consistent with megacolon, a condition that can be associated with the disease.

Details on the distribution and infection of various bug species occurring in the United States are presented in this chapter along with the unique epidemiological aspects of Chagas disease in this region. Additionally, we present evidence that Chagas disease is a rather common zoonotic infection, involving many species of North American wild mammals, domestic pets, and, in a small but important number of instances, human beings. Even though we believe that human infection in the United States has been underestimated for years, the incidence and prevalence rates of the disease in the United States are clearly much lower than in endemic regions of Latin America, for reasons we will discuss.