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Chapter

Amazonian Reservoir Hosts of *Trypanosoma cruzi*

Jocelyn Ginette Pérez Lazo, Pedro Mayor and Andrés G. Lescano

Abstract

The epidemiology of *Trypanosoma cruzi* in the wild is a particular and highly dynamic scenario that needs attention due to the increased alteration of the environment caused by different factors including anthropogenic change. This chapter is an updated summary about the known reservoir hosts of *T. cruzi* identified in the countries that share the Amazon rainforest. This information will provide a better understanding of the ecology of *T. cruzi* in sylvatic environments. This chapter will also contribute to address the potential risks of *T. cruzi* infection in Amazonian communities who are in contact with wild animals through hunting and wild meat consumption.

Keywords: *Trypanosoma cruzi*, Chagas disease, Amazon, parasites, wild animals, zoonosis, host

1. Introduction

The Amazon basin comprises multiple South American countries: Brazil (63.9%), Peru (15.6%), Bolivia (11.7%), Colombia (5.6%), Ecuador (2.1%), Venezuela (0.9%) and Guyana (0.2%). It covers over 1.3 billion hectares with 60% of total forest area (**Figure 1**) [1]. Population density in the Amazon basin is low and more than 70% live in urban areas. However, this region is in transition due to both climate change as well as anthropogenic activities such as the expansion of agriculture, road paving and logging that lead to accelerated population growth [1, 2].

The environmental conditions of the Amazon basin are favorable for the transmission of multiple vector-borne diseases. Well-known endemic diseases such as malaria and Leishmaniasis show the highest incidence in the Americas, and recent data shows the circulation of multiple arboviruses [3], with an increasing incidence of Dengue fever over time. Trypanosomatids exist in nature since millions of years ago, and Chagas disease has been identified in 4000–9000 year-old human mummies from Chile and Peru [4, 5]. Over time, the spread of Chagas disease in the Americas expanded from a wild and peridomiciliary cycle to a domestic cycle. This occurred through the domiciliation and domestication of triatomines, the primary vectors of *T. cruzi*. Thus, it was suggested that triatomines evolved from non-blood sucking insects and became mandatory hematophagous insects after undergoing morphological modifications [6, 7]. Likewise, trypanosomes also evolved and adapted to this new opportunity [6]. The evolution of *T. cruzi* was influenced by factors related to its dependence on the host and its environment and the reliance

Figure 1.
The Amazon Basin location in South America. Source: FAO, 2015.

on vertebrates as a blood source. *Trypanosoma cruzi* can spread geographically by both triatomines flying into different areas and passive carriage in vertebrate hosts moving across broad areas [8].

The deforestation of the Amazon basin, 3.6 million hectares of forest lost per year between 2000 and 2010, has become an important factor in the domestication of triatomines due to the scarcity of blood sources among wildlife. Triatomines and other disease vectors have looked for new blood sources and reached areas closer to human dwellings. Social inequality and poor public health systems exacerbate the impact of these factors, especially in rural areas, and further contribute to the transmission of Chagas disease and the emergence of new pathogens.

2. Chagas disease transmission in the Amazon basin

Trypanosomiasis is an ancient enzootic parasitosis in nature, maintained by wild animals and infected vectors. In the wild, it is presumably transmitted primarily by the oral route through predation of infected vectors or mammals, or by contamination of animal nests or shelters with metacyclic forms of the parasite released in the feces of infected triatomines or from scent glands of marsupials i.e. *Didelphis marsupialis* (**Figure 2**) [9, 10]

Besides the wild enzooty, other scenarios for the transmission of *T. cruzi* in the Amazon have been proposed (**Figure 2**). For instance, antropozoonosis, through the accidental transmission of *T. cruzi* from infected vectors or wild animals to humans. This occurs commonly with the invasion of infected vectors and marsupials in human dwellings or when humans invade the forest for different activities such as hunting, fishing, logging, tourism, etc. [11]. The invasion of vectors in human dwellings may occur during deforestation or when their blood sources such as wild mammals are scarce due to deforestation itself, over hunting or during

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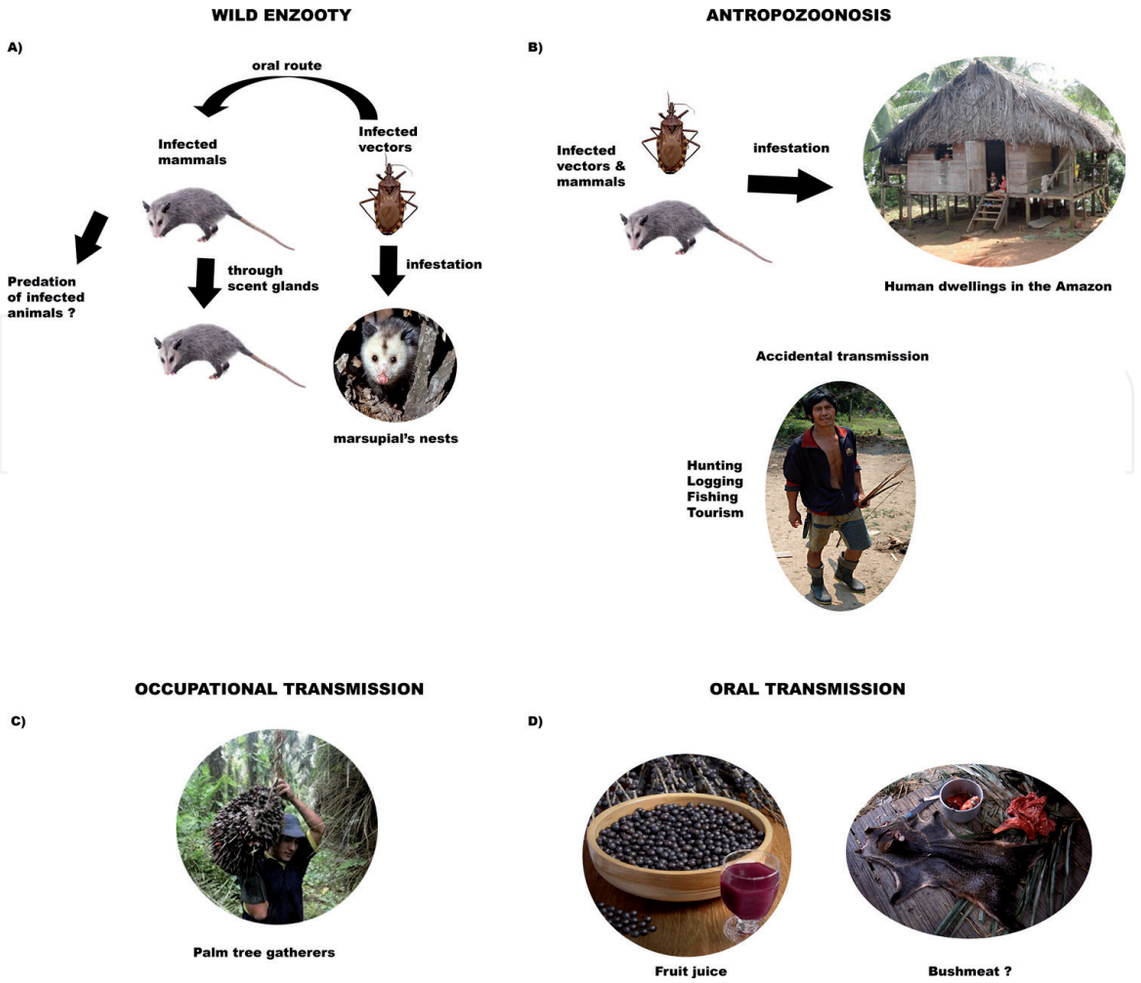

Figure 2.
T. cruzi transmission scenarios in the Amazon Basin. Photo credits: Dr. Pedro Mayor.

the wet season. Thus, vectors fly from the trees at night attracted by the lights and invade the houses in search of blood meals [11, 12]. Nymphs and adults of triatomines infected with *T. cruzi* have been found on palm trees in backyards and inside the houses in Amazonian villages. Blood meals from birds, opossum, rodents, humans, dogs and horses have been found in the gut of triatomines captured in such settings [12].

Trypanosoma cruzi may have different transmission features between areas of different degrees of disturbance. One study carried out in the Brazilian Amazon found that areas with low and intermediate degree of disturbance had higher prevalence of wild mammals with parasitemia detected by hemoculture than in areas with greater environmental disturbance caused by human occupation and agriculture. Areas with intermediate degrees of disturbance also had the highest prevalence of positive domestic mammals detected by IFAT [13]. In contrast, seroprevalence did not differ between areas with difference disturbance levels; one explanation for this finding may be the difference on the ability of reservoirs to infect vectors between geographic areas despite the comparable exposure to the parasite.

Occupational transmission scenarios are well documented among palm tree gatherers, an economically important activity. Palm trees provide shelter for different wild mammals, amphibians and insects. Gatherers of the piassava palm (*Leopoldinia piassaba*) are reportedly bitten by wild triatomines during their camping months in the forest, possibly due to the lack of other blood sources such as wild mammals [14]. Palm gatherers are therefore at higher risk to become infected compared to individuals not involved in this activity [15].

The last scenario is the oral transmission of Chagas disease, often leading to outbreaks of acute Chagas disease (ACD). The Amazon basin is the region where most of such outbreaks have been reported. Actually, Carlos Chagas himself first described ACD in 1909 when a 2-year-old girl from Lassance, Minas Gerais presented with fever, hepatosplenomegaly and Romaña's sign [8]. ACD outbreaks have occurred in both preserved and human disturbed areas [13], but with exposure of humans to the sylvatic cycle as a common factor. Most ACD outbreaks and cases are related to the consumption of food contaminated with trypomastigote forms as reported with the ingestion of fruits from palm trees such as acaí, bacaba, or the ingestion of wild meat [16]. In fact, ACD cases may have increased in the last decade [16]. It was reported an increase of notified suspected cases in Pará state in Brazil from 2010 to 2016, that may be related to the increase of production to acaí juice during those years, but also related to the increase in its consumption since the majority of patients indicated a daily consumption of this juice [16]. Consistently with these findings, juice contaminated with *T. cruzi* was able to experimentally infect mice [17, 18], despite the juice being previously frozen or refrigerated.

In Brazil, *T. cruzi* infection got more attention only after 1969 when ACD cases were reported in Belém, State of Pará [12]. However, sporadic human infections have been reported before this date in the Amazon basin. In a serological study by indirect inmunoflourescence test in a county of 15 villages in the Brazilian Amazon found 0.83% (212/25, 451) of prevalence for *T. cruzi* infection. Children ≤ 10 years had positive serological results indicating recent transmission and acute infection [12]. In Peru, the first ACD case was reported in 1919 in the southeastern Amazonian department of Madre de Dios. Only a few ACD cases have been reported since, although multiple cases have been reported in recent years in Amazonian regions such as Pasco and Loreto [19]. In Ecuador, Chagas disease was first diagnosed in 1929 in Guayas, a coastal province. The first human cases in the Amazon region of Ecuador were yet reported in 1991 in Napo and Sucumbíos provinces [20]. Some studies later demonstrated the exposure and active transmission of *T. cruzi* in more Amazonian provinces such as Orellana, Pastaza, Morona Santiago [20]. Similar to Brazil, ACD cases were also reported in children ranging from 1 to 5 years old in the Ecuadorian Amazon region [21]. In Colombia, the *T. cruzi* haplotype Ia has been isolated among ACD cases from the Amazonian departments of Caquetá and Putumayo, including children and servicemen who were in contact with the forest [22].

Indigenous communities in the Amazon basin are also affected by *T. cruzi* infection. During 2006 to 2010, six ACD cases were reported among children from the Aguaruna, Huambisa and Kandoshi communities in the Peruvian Amazon basin. ACD was attributed to either the biting of infected triatomines found inside the dwellings or oral transmission by drinking "masato" (a fermented beverage made with yuca) or "chapo" (a ripe banana beverage) since no traveling outside the community or blood transfusion was recorded [19]. ACD cases in indigenous communities in Ecuador Amazon region have been reported since 1987; a seroepidemiological study found a 6.0% (61/1011) of positive individuals from 15 of the 18 studied communities. Likewise, the prevalence increased with age, individuals older than 50 showed 18.8% of seroprevalence [21].

3. *Trypanosoma cruzi* reservoir hosts in the wild

The first *T. cruzi* infection in Amazonian wildlife was reported in 1924 among squirrel monkeys (*Saimiri sciureus*) [23]. In mammalian hosts, *T. cruzi* infection could develop depending on factors such as the host health status, the transmission route, the parasite population and co-infection with other parasites. However,

reservoir hosts should support the maintenance and dispersion of the parasite in nature [9], and therefore be capable of infecting vectors. Vectors can become infected depending on the parasitemia in the host's blood, which varies among host species and individuals [9]. This is corroborated by previous studies that showed differences in *T. cruzi* infection rates between species of the same genus. For instance, experimental *T. cruzi* infection in the rodent *Thrychomys fosteri* was more severe with high and long-lasting parasitemia compared to *Thrychomys laurentius* [24].

Trypanosoma cruzi can infect different mammal species from all forest strata and canopy levels. **Table 1** lists wild animal species reported positive to *T. cruzi* infection in the Amazon basin. A higher proportion of positive blood culture and serology by immunofluorescence antibody test (IFAT) was observed in mammals from the Amazon compared to specimens from other biomes (Atlantic forest, Caatinga, Cerrado, Pampa and Pantanal) [9]. This study also suggested that 8% of positive animals would be sufficient to maintain the *T. cruzi* cycle in any biome. Different mammalian orders seem to participate in the transmission cycle of *T. cruzi*, but Primates, Didelphimorphia, Chiroptera and Carnivora apparently are the primary taxa due to their high parasitemias and Discrete Typing Units (DTUs) diversity [9].

TcI, TcIII and TcIV are the reported DTUs circulating in the Amazon basin [25]. A very low prevalence of TcIII and TcIV (0.8%) infection was found in 714 *T. cruzi* isolates from five different biomes in Brazil. TcIV was more prevalent than TcIII, and no specific association between genotypes and animal species or geographical distribution was suggested [26]. However, these findings may be biased by the methodology used in this study or the temporal distribution of these DTUs in nature.

The main taxa for the transmission of *T. cruzi* in the Amazon basin are reported below:

3.1 Didelphimorphia

This order includes the hosts most frequently infected by *T. cruzi* in all biomes of Brazil, presenting high rates of positive hemoculture [9, 26]. Didelphimorphia species in the Amazon basin seems to be predominantly infected by TcIII and/or TcIV across different biomes. This order includes the most frequent hosts infected by TcIII, TcIV or mixed trypanosome/genotypes compared to other mammalian taxa [26].

It is suggested that marsupials could act as generalist species due to the diversity of Trypanosoma spp. found simultaneously infecting a single host [9]. For instance, a mixed, triple infection with *Trypanosoma cascavelli* (reported in reptiles), *Trypanosoma dionisii* (reported in bats) and Trypanosoma sp. was reported in *Monodelphis americana* [27]. Other species such as *Philander opossum* show a high prevalence of *T. cruzi* positive hemoculture implying its ability to infect vectors. *Philander opossum* was also the most abundant species present across different areas of the Amazon basin regardless of the season and the degree of land disturbance [13].

Didelphis marsupialis is the specie from this order most commonly reported with *T. cruzi* infection. However, it has been suggested that its presence in a specific area may not be directly related to the endemicity of *T. cruzi*. Low prevalence of the parasite was found in an area despite their great abundance of this mammal, and it may not be a critical specie for the maintenance of the *T. cruzi* sylvatic cycle in the Amazon basin [13]. *Trypanosoma cruzi* infection of *D. marsupialis* could take place orally by ingestion of small mammals or triatomines. However, host-to-host infection could also occur through direct contact with infective metacyclic trypomastigotes released by the scent glands of infected marsupials. Scent glands have demonstrated being suitable for epimastigotes and their differentiation into infective metacyclic forms [10]. Didelphis can also adapt to peridomestic areas, and

Order/Genus	Specie	Methods				References
		H	I	P	DTU	
Artiodactyla						
Sus	*Sus scrofa*		x			[9]
Cingulata						
Cyclopes	*Cyclopes didactylus*	x			Zymodeme 1	[46]
Dasypus	*Dasypus novemcinctus*	x		x	TcIV	[9, 28, 46, 47]
Tamandua	*Tamandua tetradactyla*	x		x	TcI	[9, 48]
Didelphimorphia						
Caluromys	*Caluromys philander*		x			[9]
	Caluromys sp.	x	x	x	TcI	[9, 46, 47]
Didelphis	*Didelphis marsupialis*	x	x	x	TcI, TcII, TcI+ T. rangeli, TcI+ TcII, TcI+ TcIII	[9, 13, 46, 47, 49]
	Didelphis albiventris	x		x		[49]
	Didelphis sp.		x			[9]
Gracilinanus	*Gracilinanus sp.*	x	x	x	TcI	[9, 49]
Marmosa	*Marmosa murina*	x	x	x	TcI	[9, 13]
	Marmosa cinerea	x				[47]
	Marmosa sp.		x			[9]
Marmosops	*Marmosops parvidens*		x			[9]
	Marmosops sp.	x	x	x	TcI	[9]
Metachirus	*Metachirus nudicaudatus*	x			Zymodeme 1	[46]
Micoureus	*Micoureus demerarae*	x	x	x	TcI	[9, 13]
Monodelphis	*Monodelphis domestica*		x			[9]
	Monodelphis brevicaudata	x			Zymodeme 3	[46]
	Monodelphis sp.	x	x	x	TcI, TcI + TcIV	[9]
Philander	*Philander opossum*	x	x	x	TcI, TcI+ TcII	[9, 13, 46, 47, 49]
	Philander sp.	x	x	x	TcI, TcI+ TcIII/TcIV, TcI + T. Rangeli	[9]
Primates						
Alouatta	*Alouatta belzubul*	x	x	x	TcI+ TcIV	[9]
	Alouatta caraya	x	x	x	TcI+ TcIV	[9]

Order/Genus	Specie	Methods				References
		H	I	P	DTU	
Cebuella	*Cebuella pygmaea*	x				[50]
Callicebus	*Callicebus cupreus*			x		[33]
Cebus	*Cebus albifrons*			x		[33]
	Cebus apella			x		[33]
Lagothrix	*Lagothrix poeppigii*			x		[33]
Saguinus	*Saguinus midas niger*	x			Zymodeme 1	[46]
	Saguinus bicolor	x		x	TcI, TcI + T.rangeli	[32]
Saimiri	*Saimiri boliviensis*			x		[33]
	Saimiri sciureus	x		x		[30, 33]
	Saimiri ustus	x				[30]
Sanguinus	*Sanguinus imperator imperator*	x				[50]
	Sanguinus fuscicollis weddelli	x				[50]
Sapajus	*Sapajus libidinosus*	x	x	x	TcI, TcI + T.rangeli	[9]
	Sapajus macrocephalus			x		[33]
Rodentia						
Cuniculus	*Cuniculus paca*	x		x		[28, 47]
Akodon	*Akodon lindberghi*	x		x	TcI	[9]
Cerradomys	*Cerradomys sp.*		x			[9]
Coendou	*Coendou prehensilis*	x	x	x	TcI + T.rangeli	[9]
	Coendou sp.	x			Zymodeme 1	[46, 47]
Dasyprocta	*Dasyprocta prymnolopha*		x			[9]
	Dasyprocta sp.	x		x		[28, 47]
Echymys	*Echymys chrysurus*	x			Zymodeme 1	[46]
Rodentia						
Holochilus	*Holochilus sp.*		x			[9]
Hylaeamys	*Hylaeamys megacephalus*	x	x	x	TcI	[9]
Nectomys	*Nectomys squamipes*					[51]
Oryzomys	*Oryzomys capito*	x				[47]
	Oryzomys sp.		x			[9]

Order/Genus	Specie	Methods				References
		H	I	P	DTU	
Oxymycterus	*Oxymycterus sp.*		x			[9]
	Proechimys roberti		x			[9]
Proechimys	*Proechimys gr. cuvieri*	x	x	x	TcI	[9]
	Proechimys gr. guianensis	x	x			[9, 47]
	Proechimys sp.	x	x	x	TcI+ TcIII/TcIV	[9]
Rattus	*Rattus rattus*	x			Zymodeme 1	[46]
Sciurus	*Sciurus sp.*	x			Zymodeme 1	[46]
Carnivora						
Nasua	*Nasua nasua*	x		x		[28, 47]
Tayra	*Tayra barbara*					[48]
Chiroptera						
Anoura	*Anoura caudifer*	x		x	TcI	[9]
Artibeus	*Artibeus lituratus*	x		x	TcI, TcIV	[9]
	Artibeus cf. fimbriatus	x		x	TcI	[9]
	Artibeus planirostris	x		x	TcI	[9]
Carollia	*Carollia perspicillata*	x		x	TcI	[9]
	Carollia cf. beikeith	x		x	TcI	[9]
	Carollia brevicauda	x		x	TcI	[9]
Choeroniscus	*Choeroniscus minor*					[52]
Chiroptera						
Dermanura	*Dermanura cinereus*	x		x	TcI	[9]
Desmodus	*Desmodus rotundus*			x		[35]
Diaemus	*Diaemus youngi*			x		[35]
Glossophaga	*Glossophaga soricina*	x		x	TcI, TcIV	[9, 52]
Lasiurus	*Lasiurus blossevillii*	x		x	TcI	[9]
Lonchophylla	*Lonchophylla thomasi*	x		x	TcI	[9]
	Lonchophylla mordax					[52]
Molossus	*Molossus major*					[52]
	Molossus ater					[52]

Order/Genus	Specie	Methods				References
		H	I	P	DTU	
Mycronycteris	*Mycronycteris megalotis*					[52]
Noctilio	*Noctilio labialis*					[52]
Phyllostomus	*Phyllostomus discolor*	x		x	TcI	[9]
	Phyllostomus hastatus	x		x	TcIV	[9, 35, 52]
	Phylostomus alongatus					[52]
Plathyrhinus	*Plathyrhinus infuscus*	x		x	TcI	[9]
Saccopterix	*Saccopterix bilineata*					[52]
Tonatia	*Tonatia saurophila*	x		x	TcI	[9]
Trachops	*Trachops cirrhosus*	x		x	TcI	[9, 35]
Uroderma	*Uroderma bilobatum*	x		x	TcI	[9]
Vampyressa	*Vampyressa sp.*	x		x	TcI	[9]

H = hemoculture, I = IFAT, P=PCR.

Table 1.
Free-ranging wild animal's species reported positive to Trypanosoma cruzi infection in the Amazon Basin.

T. cruzi-infected specimens have been found in backyards of houses from different villages in the Brazilian Amazon [12].

Histopathological lesions observed in infected marsupials resemble those presented by Chagas disease patients. Myocarditis with mononuclear infiltrates, cell lysis and inflammatory infiltrates in skeletal muscles, esophagus and small and large intestines are other lesions found in natural infection [12].

3.2 Carnivores

Carnivores are likely to be infected by the oral route, and those whose diet includes insects or flesh present the highest infection rates [9]. The most reported carnivore infected with *T. cruzi* is the coati (*Nasua nasua*) [28]. Coatis are terrestrial but construct their nests in the high canopy level of large trees, which then serve as habitat for triatomines and other insects. Two-thirds of 34 Triatoma and Rhodnius triatomines found in 24 coati nests in the Pantanal state in Brazil were positive to *T. cruzi*. The 62.5% (8/8) of 8 nests infested with triatomines were *T. cruzi*-positive. The triatomines blood meal sources identified by precipitation of the stomach contents included coati and also rodents, birds and marsupials. Moreover, rodents and birds were observed visiting these nests [29].

3.3 Non-human primates

Different species of trypanosomatids have been reported in wild non-human primates from the Amazon. The *T. cruzi* prevalence rates reported in the Brazilian

Amazon range from 10.3% (17/165) using parasitological methods in captured free-ranging non-human primates to 45.5% (45/99) by IFAT in captive and semi-captive primates [30, 31]. However, the *T. cruzi* prevalence in wild non-human primates of the Amazon basin seems to be lower than the prevalence of *T. rangeli*, with the latter showing lasting parasitemias [32]. Several studies corroborate this observation, such as 35.2% versus 10.3% in 165 wild squirrel monkeys captured in the Brazilian Amazon [30], and 75.0% versus 10.4% cultures among 96 tamarins captured in the Amazonas state in Brazil [32]. In a study conducted in the Peruvian Amazon, free-ranging non-human primates had a high prevalence of trypanosomatids (64.3% vs. 27.9%) and *T. cruzi* (8.7% vs. 3.3%) by PCR, compared to captive primates, suggesting that parasite transmission occurs more actively in the sylvatic cycle. Pitheciidae had the highest trypanosomatid prevalence (20/22, 90.9%) and Cebidae had the highest *T. cruzi* prevalence (15/117, 12.8%) [33]. The difference in the prevalence is not well understood but may be related to the route of infection. While *T. cruzi* might be transmitted through either contact with infective forms in the triatomine feces or orally, *T. rangeli* may be primarily transmitted during biting by infected triatomines [32].

There is scarce evidence regarding the physiopathology of *T. cruzi* natural infection in free-ranging non-human primates. Electrocardiography abnormalities were found in a few *T. cruzi*-infected tamarins aged 7 months to 4 years old from an Atlantic Forest reserve in Brazil with infection time spanning 6 months to almost 5 years. It was estimated that infected individuals were 18 times more likely to show detectable electrocardiogram (ECG) abnormalities than those uninfected. Likewise, infected non-human primates were prone to have higher levels of cardiac injury markers such as MBi, a cardiac lesion marker and total protein in serum. Although these findings did not suggest a general health problem, left ventricular hypertrophy was present in some of the infected tamarins similar to the chronic form of Chagas disease in humans [34]. These studies probably underestimate the clinical burden since non-human primates with severe cardiac lesions probably do not survive in the wild and are therefore not included. Despite this limitation, it is important to consider the impact of the physiopathology of natural infection, particularly for at risk species. For instance, *T. cruzi* infection has been reported in *Saguinus bicolor*, a critical endangered monkey in the Amazon basin [32].

3.4 Quiroptera

It was suggested that bats could be bio-accumulator hosts and dispersers of trypanosomatids because of their ability to fly and the great diversity of Trypanosoma species found in bats specially compared to other animal taxon [9]. Out of 1219 Brazilian bats from 76 genera and 94 species, 14% were positive to Trypanosoma sp. by hemoculture and 5% of them were *T. cruzi* positive in single or mixed infections. Although bat is able to be infected with other DTUs most bats were infected with TcI, and TcII was not detected. The highest infection rate was found in the Amazon basin compared to other biomes, suggesting that unaltered areas nurture a high parasite diversity [9]. In the Peruvian Amazon, a 4.1% *T. cruzi* infection prevalence in bats was reported, in both hematophagous (2.7%; 2/73) and non-hematophagous species (6.2%; 3/48) [35]. Interestingly, *T. cruzi* DNA was detected in the salivary glands of *Diaemus youngi*, an hematophagous bat [35]. This highlights the importance of studying the transmission mechanisms of *T. cruzi* in bats and their public health implications for the Amazon basin.

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4. Wild meat consumption as a potential risk of oral transmission

Trypanosoma cruzi hosts encompass more than 100 wild and domestic mammalian species, which in turn belong to eight different taxonomic orders distributed in all phytogeography regions of the Neotropics [36]. Since subsistence hunting of wild mammals for consumption is one of the main sources of animal protein in the Amazon Basin, wild meat constitute a potential source for human infection.

In the Amazon region, wild meat represents an important component of household food security, income and a key social and cultural driver. Wild meat is still a key element in Amazon peoples' diet and accounts for a high percentage of daily protein intake. It is estimated that the wild meat consumption rate in rural settlements of the Amazon Basin is 172 g per person per day [37]. A study found that 39% of households in Latin America harvested and consumed wild meat, and dependence was highest among the poorest households [38]. Estimates of the annual wild meat harvest in the Northern Peruvian Amazon are 113,000 animals (1680 tons), and 89,224 tons of meat per year in the Brazilian Amazon [39, 40]. Furthermore, hunting pressure has increased in recent years due to various causes, such as the growth of human populations, access to remaining forests, commercialization of wild meat, increasing use of efficient modern hunting techniques and erosion of traditional hunting institutions due to rapid cultural changes [41].

Notwithstanding its positive nutritional contributions, some serious health concerns may be associated with wild meat consumption in the Amazon basin. Emerging infectious diseases worldwide are increasing over time and are dominated by zoonoses (60%), of which the majority (72%) originates in wildlife [42]. A study conducted in the Peruvian Amazon estimated an annually consumption of 45 animals infected with *T. cruzi*, translating to 0.75 infected animals typically consumed by a large extended family, suggesting recurrent infection opportunities [28].

A few studies from Argentina and Brazil report Chagas disease transmission through consumption of raw, poorly cooked meat, blood or contact with carcasses of wildlife among children [43]. However, some of these studies did not rule out the possibility of vectorial transmission and others have not found proof of exposure to infected triatomines. What it is certainly known is that frequently hunted animals in the Amazon have been reported as *T. cruzi* reservoirs [28, 33], and carnivores might tend to have higher infection rates than non-carnivores suggesting accumulation through wild meat intake. Thus, the consumption of wild meat may be an important risk for human *T. cruzi* infection in the Amazon Basin, which may happen particularly during cleaning of wild meat or contamination of cooking utensils [13]. Other associated risk factors in rural Amazon societies are poor hygienic conditions, unavailability of clean water, inadequate medical care and insufficient knowledge about local diseases [44]. Nevertheless, further studies addressing the relationship between wild meat consumption and Chagas disease are required to better understand the risk of infection in Amazonian communities. Prompt diagnosis, notification of cases and epidemiological studies to assess the risk factors that trigger ACD outbreaks are greatly needed.

5. Domestic animals as reservoir hosts of *Trypanosoma cruzi* in the Amazon basin

Domestic animals should be included in epidemiological studies since they are good sentinels of disease transmission in a geographic area. In the Amazon, several studies report *T. cruzi*-positive domestic animals such as pigs and dogs

[45]. TcI is the primary DTU reported infecting dogs in the Brazilian Amazon [13]. Although a higher prevalence of *T. cruzi* was found in pigs compared to dogs in some anthropogenic disturbed areas, dogs present longer parasitemias. Extended parasitemia suggests that dogs are better able to infect vectors, thereby acting as *T. cruzi* reservoirs. These domestic animals may become infected by different routes, mainly the biting of the infected triatomines since bugs and dogs converge near human dwellings [13]. Other routes of transmission would be the ingestion of the infected bugs or food contaminated with triatomine feces and predation of infected small mammals.

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Conflicts of interest

The authors declare that they have no conflicts of interest regarding the publication of this chapter.

Nomenclature

ACD = acute Chagas disease
DTUs = discrete typing units
ECG = electrocardiogram
IFAT = immunofluorescence antibody test
MBi = cardiac lesion marker
PCR = polymerase chain reaction
TP = total protein

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