Deforestation, Urbanization, and Mosquito and Tick-Borne Diseases

Subjects: Entomology | Public, Environmental & Occupational Health | Ecology

Contributor: Diana Ortiz

Central America is a unique geographical region that connects North and South America, enclosed by the Caribbean Sea to the East, and the Pacific Ocean to the West. This region, encompassing Belize, Costa Rica, Guatemala, El Salvador, Honduras, Panama, and Nicaragua, is highly vulnerable to the emergence or resurgence of mosquito-borne and tick-borne diseases due to a combination of key ecological and socioeconomic determinants acting together, often in a synergistic fashion. Of particular interest are the effects of land use changes, such as deforestation-driven urbanization and forest degradation, on the incidence and prevalence of these diseases, which are not well understood. In recent years, parts of Central America have experienced social and economic improvements; however, the region still faces major challenges in developing effective strategies and significant investments in public health infrastructure to prevent and control these diseases.

arbovirus   malaria   deforestation   urbanization   Rickettsiales   Culicidae   Ixodidae

Central America

1. Introduction

Vector-borne diseases (VBDs) remain an important public health problem worldwide, particularly in tropical and subtropical regions, and they are becoming more prevalent in recent years. Arthropod vectors are associated with the transmission of some of the most significant infectious diseases affecting both animals and humans [1][2][3]. The global burden of VBDs is significant, accounting for more than 17% of infectious diseases in humans with more than three billion people currently inhabiting endemic areas and at risk of exposure to these pathogens [4]. Most people affected by these diseases live in developing countries under conditions that favor a greater burden of disease, especially in poor and marginalized populations, including rural inhabitants, Indigenous populations, women living in poverty, the elderly, and children [5][6]. Together, these diseases produce significant mortality and morbidity, causing millions of deaths every year, long-term disabilities, and life-long sequelae [5][7][8][9].

Emerging and resurging diseases are defined as recently evolved or newly discovered pathogens that have demonstrated increased incidence in host populations in the past 20 years or poses a future threat. These pathogens are characterized by their expanding geographic spread, increasing public health impact, changes in their clinical presentation, or novel infection occurrence in humans [10][11]. Some of these pathogens are also characterized by their resurgence after long periods of decline in infection incidence. About 60% of these diseases are zoonotic in origin and, to various degrees, dependent on animal reservoirs for survival and maintenance [10][11]

[12][13][14]. In many countries, the incidence of these diseases has declined to low levels due mostly to effective prevention and control programs; however, some were never satisfactorily controlled throughout their endemic regions. Many are currently increasing in incidence and spreading beyond their previously known geographical ranges. Some of them have reappeared only in limited regions while others have become major global problems [9][12][15].

Most emerging zoonotic VBDs are transmitted by ticks (Family *Ixodidae*) and mosquitoes (*Culicidae*) and caused by RNA viruses (Families *Flaviviridae*, *Bunyaviridae*, and *Togaviridae*) and Rickettsiaceae bacteria [12][16]. The vectorial capacity of mosquitoes and ticks is enhanced by their high environmental adaptability, which includes high reproductive outputs under suitable conditions and high capacity to invade ecologically disturbed environments, especially peridomestic habitats where human and domesticated animal hosts are readily available [16][17][18]. In terms of human morbidity and mortality, malaria, dengue, Rickettsial fevers, and Lyme disease are some of the most important of these resurgent infections [2][15][16][19].

The relationship between arthropod vectors and the pathogens they transmit is particularly sensitive to anthropogenically driven global changes. Factors behind the dramatic emergence and resurgence of VBDs are complex, vary geographically and temporarily, and often have an additive effect on disease ecology and epidemiology [1][2][12][16][19]. Currently recognized anthropogenic drivers of VBD emergence and resurgence include demographic changes (e.g., global population movements and growth, unplanned and uncontrolled urbanization), socioeconomic changes (e.g., modern transportation and commerce, human encroachment on natural disease foci), illegal activities (e.g., illegal logging and cattle ranching, illegal drug trafficking), accelerated exploitation of natural resources (e.g., changes in land use, forest degradation, reduction in biodiversity, agricultural practices), changes in host susceptibility and pathogen adaptation (e.g., increased movement of humans and animals, pathogen genetic variability), degradation of public health infrastructure (e.g., lack of effective vector control, disease surveillance, and prevention programs), and climate change (e.g., changes in regional temperature and rainfall patterns lead to alterations in vector dynamics) [1][2][12][16][19][20][21][22].

There is increasing evidence that anthropogenic land use changes can directly and indirectly influence vector-borne pathogen transmission dynamics [10][23]. Although land use changes, such as urbanization as a result of forest degradation and deforestation, have been associated with increased disease transmission, their direction, extent, specific mechanisms, and persistence are not clearly understood. Often the effects of these factors on VBD emergence and resurgence are interdependent, synergistic, and difficult to study.

Central America is the region that links North and South America comprising most of the narrow isthmus that separates the Pacific Ocean from the Caribbean Sea. It consists of the countries of Belize, Costa Rica, Guatemala, El Salvador, Honduras, Panama, and Nicaragua, and is inhabited by about 44.5 million people. Generally, this region is characterized by a diversity of ecosystems, physical geographies, sociocultural and socioeconomic structures, and public health profiles [24]. Over the past decades, this region has sustained dramatic land use changes, including cattle ranching, large-scale commercial plantations, illegal airstrips, mining, road construction, tourism infrastructure, illegal timber extraction, and housing construction. These land use changes have

significantly accelerated the rates of deforestation and urbanization in the region [25][26][27][28][29][30]. Despite the high prevalence of several VBDs in Central America, the effects of anthropogenic-driven deforestation and urbanization on the transmission dynamics of these diseases are not well understood. The presence and interactions of numerous physical and socioeconomic factors in the region amplifies its vulnerability to the emergence and resurgence of several VBDs [6].

2. Mosquito-Borne and Tick-Borne Diseases in Central America

The global emergence and resurgence of VBDs in the last three decades are closely linked to demographic, economic, and societal changes. The decay in public health infrastructure required to prevent and control these diseases and the unprecedented population growth, primarily in rapidly growing cities, are factors that have facilitated VBD transmission and their geographic spread [2][5]. In Central America, the most important VBDs affecting humans and animals include Chagas disease, leishmaniasis, dengue fever, malaria, Zika fever, chikungunya fever, West Nile fever, rickettsial diseases, Eastern equine encephalitis, Saint Louis encephalitis, and Venezuelan equine encephalitis (**Table 1**) [6][31].

Table 1. Description of the most important mosquito-borne and tick-borne diseases in Central America.

Disease	Causative Agents	Distribution of Infections in Humans	Confirmed or Suspected Mosquitoes and/or Tick Vectors	Confirmed or Suspected Non-Human Vertebrate Hosts
West Nile fever	West Nile virus (*Flavivirus*)	Clinical, serosurveys (CR, N)	*Culex quinquefasciatus*, *Cx. mollis/Cx. inflictus* (G)	Equines, non-human primates, wild birds, sentinel chickens (CR, B, ES, G)
Saint Louis encephalitis	Saint Louis encephalitis virus (*Flavivirus*)	Clinical, serosurveys (P, B, G, H)	*Sabethes chloropterus*, *Trichoposopon* spp., *Wyeomyia* spp., *Haemagogus lucifer*, *Deinocerites pseudes*, *Mansonia dyari*, *Culex nigripalpus* (P, CR, G)	Wild rodents, wild birds, sentinel rodents, sentinel chickens, non-human primates,

Disease	Causative Agents	Distribution of Infections in Humans	Confirmed or Suspected Mosquitoes and/or Tick Vectors	Confirmed or Suspected Non-Human Vertebrate Hosts
				sloths, equines, pigs (P, CR, B, H, G)
Venezuelan equine encephalitis	Venezuelan equine encephalitis virus (*Alphavirus*)	Clinical, serosurveys (all countries)	*Psorophora confinnis, Culex nigripalpus, * Cx. (Melanoconion) taeniopus, other Cx. (Melanoconion)* spp., *Mansonia titillans, Ps. cilipes, Aedes taeniorhynchus*, and *Deinocerites pseudes* (P, CR, B, G)	Equines, wild rodent, opossum, birds, and bats (CR, N, H, ES, G)
Eastern equine encephalitis	Madariaga virus (*Alphavirus*)	Clinical, serosurveys (P)	*Culex (Mel.) taeniopus* (P)	Horses, bats, wild lizards, wild birds (P, CR, B)
Yellow fever	Yellow fever virus (*Flavivirus*)	Clinical (all countries)	*Aedes aegypti, Haemagogus janthinomys, Hg. leucocelaenus, Hg. lucifer, Hg. equinus, Hg. spegazzinii*, and *Sa. chloropterus, Hg. mesodentatus* (P, CR, N, G)	Non-human primates, marsupials (P, CR, N, B, H, G)
Zika fever	Zika virus (*Flavivirus*)	Clinical and serological (all countries)	** *Aedes aegypti, Ae. albopictus* (all countries)	Unknown
Chikungunya fever	Chikungunya virus (*Alphavirus*)	Clinical and serological (all countries)	** *Aedes aegypti, Ae. albopictus* (all countries)	Unknown
Dengue fever	Dengue viruses 1–4 (*Flavivirus*)	Clinical and serological (all countries)	** *Aedes aegypti, Ae. albopictus* (suspected) (all countries)	Bats, non-human primates (CR)
Mayaro fever	*Mayaro virus (Alphavirus)*	Clinical and serological	*Haemagogus janthinomys, Psorophora ferox, Culex* (*Mel.*) *vomerifer* (P)	Non-human

Disease	Causative Agents	Distribution of Infections in Humans	Confirmed or Suspected Mosquitoes and/or Tick Vectors	Confirmed or Suspected Non-Human Vertebrate Hosts
		(P, CR, G)		primates (P, CR, H, G)
Malaria	*Plasmodium vivax, P. falciparum*	Clinical and serological (all countries)	* *Anopheles albimanus, An. darlingi, An. punctimacula*, other *Anopheles* spp. (all countries)	Unknown
	P. malariae	Clinical and serological (P, CR, B, ES, G)	Unknown	Unknown
Rickettsiosis	*Rickettsia* spp. (species causing spotted fevers was not identified)	Clinical, serosurveys (all countries)	*Amblyomma mixtum* (G)	Wild rabbits, dogs, coyote (P, CR)
	R. rickettsii (Rocky Mountain spotted fever)	Clinical (P, CR)	* *A. mixtum, Rhipicephalus sanguineus* s.l., *A. varium, Dermacentor nitens, Haemaphysalis leporispalustris* (P, CR)	Dog, horse (P, CR)
	R. akari (rickettsialpox)	Serosurvey (CR)	Unknown	Unknown
	R. parkeri	Unknown	* *A. maculatum* (B)	Unknown
	R. parkeri strain Atlantic Forest	Unknown	* *A. ovale* (B) [32]	Unknown
	R. africae	Unknown	*A. ovale* (N)	Unknown
Ehrlichiosis [34]	*Ehrlichia chaffeensis* (monocytic ehrlichiosis) [33]	Clinical (CR)	*Amblyoma mixtum, Amblyomma* sp., *Dermacentor nitens, Rhipicephalus microplus* (P)	Unknown
	E. ewingii (monocytic ehrlichiosis)	Unknown	[33] *R. microplus* (P) [35][36]	Unknown
	E. canis (granulocytic ehrlichiosis)	Clinical (P, CR)	[37] ** *R. sanguineus* s.l. (all countries)	Dogs (all countries)

Several studies have been conducted in Central America to understand WNV transmission involving equines and mosquitoes. For instance, studies were conducted in Guatemala to determine WNV transmission dynamics and seroepidemiology [38][39][40]. Of the seven departments selected for monitoring by sentinel chickens in the country, one transmission focus was identified in the eastern city of Puerto Barrios. Annual transmission at that site was detected between the months of May and October of 2005–2008. Additionally, great-tailed grackles (*Quiscalus mexicanus* [Gmelin, 1788]) have been identified as primary amplifying hosts in the region [38][40]. Environmental factors, such as high temperatures and low rainfall, are strongly associated with chicken seroconversions since these factors influence mosquito population density and viral infection kinetics in the vector in both rural and urban

Disease [38]	Causative Agents [41]	Distribution of Infections in Humans	Confirmed or Suspected Mosquitoes and/or Tick Vectors	Confirmed or Suspected Non-Human Vertebrate Hosts
Anaplasmosis	*Anaplasma phagocytophilum* (human granulocytic anaplasmosis)	Unknown	*Rhipicephalus sanguineus* s.l., *R. microplus* (P, CR)	Dogs, bovines, equines, deer (CR, N, G)
Borreliosis [43]	*Borrelia burgdorferi* s.l. (Lyme disease)	Clinical (CR)	*Ixodes* c.f. *boliviensis* (P)	Dogs (CR)
	Borrelia sp. (tick-borne relapsing fever)	Clinical (P, G) [38][39][44][45][46]	*Ornithodoros talaje, O. rudis* (P) [43][45][46]	Armadillos, opossums (P)

* Main vector. ** Suspected vectors; pathogen has not been detected/isolated in all countries from these vectors, but they are present and considered the main vectors worldwide. Country abbreviation (Belize = B; Costa Rica = CR; El Salvador = ES; Guatemala = G; Honduras = H; Nicaragua = N; Panama =P).

transmission of WNV in Central America is largely unknown. A more recent study by Piche-Ovares et al. (2021) during the rainy and dry seasons reported additional serological evidence of neutralizing antibodies against WNV in equines, humans, sentinel chickens, a local wild bird, and one seroconversion event in a horse [47]. However, the study did not find molecular evidence of active virus circulation in wild birds and mosquitoes. Although human cases of neurological WNV infection have not yet been recorded in Costa Rica, evidence suggests that the virus is widely distributed throughout the region [47].

A limited number of studies have examined the incrimination of mosquito species in WNV transmission in Central America. For instance, a study by Morales-Betoulle et al. (2013) at a WNV transmission foci in Guatemala isolated the virus from *Culex quinquefasciatus* Say, 1823 and *Culex mollis* Dyar and Knab, 1906/*Cx. inflictus* Theobald, 1901 mosquitoes; however, no isolates were obtained from the most abundant species, *Cx. nigripalpus* Theobald, 1901 [38]. Laboratory vector competence using Central American WNV strains and mosquitoes have found evidence of moderate-to-strong competency for *Cx. quinquefasciatus* and *Cx. nigripalpus* in Honduras and Guatemala [48][49].

One of the most critical issues in Central America is the lack of data on the extent of WNV human disease burden in the region since it can be very challenging to diagnose these infections. Human WNV cases are often misdiagnosed or underdiagnosed due to several factors, including serological cross-reactivity with other flaviviruses circulating in the region, virus genome diversity, low transient viremia, and the lack of laboratory testing capacity [50]. Presently, the diagnosis of WNV infection is mostly based on serological methods since molecular identification of virus RNA is often unreliable due to the short-term transient viremia and low viral load at the time of the onset of symptoms [51]. As seen in other areas where the WNV circulates, the serological diagnosis of WNV disease in Central America is also problematic due to the active co-circulation of dengue virus, Zika virus, and other flaviviruses that will likely produce cross-reactivity in serological assays [51]. Lastly, laboratory capacity to test for WNV and other VBDs varies widely among Central American countries, ranging from some laboratory capacity and epidemiological surveillance systems to a lack of critical resources, such as appropriate testing technologies, reagents, facilities, epidemiological surveillance systems, and technical expertise [52].

One of the possible reasons for the lack of evidence for human and equine WNV neurological disease and high avian mortality in Central America is that birds infected with more virulent strains could not start their migration process to South America while only birds infected with less virulent viral strains are able to migrate [53]. Another hypothesis that seeks to explain the relatively low human, equines, and avian mortality in Central America focuses on pre-existing neutralizing antibodies against other flaviviruses, such as dengue virus (DENV) and Saint Louis encephalitis virus (SLEV), which might offer partial protection from WNV disease [36][54][55]. Finally, environmental and host factors, such as temperature, vector competence, and host susceptibility, may influence the genetic selection of less virulent variants [54]. Although WNV disease has not been reported in most of Central America, all the components that could support virus circulation are present throughout the region maintaining the risk for future outbreaks in the region.

2.1.2. Saint Louis Encephalitis

Saint Louis encephalitis virus (SLEV) (genus *Flavivirus*, family *Flaviviridae*) was first identified in St. Louis, Missouri, USA in 1933. SLEV can be classified into eight genotypes, with genotypes I and II circulating mainly in the US and associated with outbreaks of human encephalitis [56], while genotypes III–VIII have been found only in Central and South America. In Central America, SLEV circulation was first documented in 1957 in Buena Vista, Panama, when the virus was isolated from a pool of *Sabethes chloropterus* (von Humbolt, 1819) [57]. Subsequently, serological evidence and isolates of SLEV were also detected in Maje Island, Darien Province, and Panama Province in humans, wild birds (local and migratory), several wild mammals (rodents, non-human primates, sloths), various sentinel animals, and several mosquito species, including *Haemagogus lucifer* (Howard, Dyar and Knab, 1913), *Deinocerites pseudes* Dyar and Knab, 1909, *Mansonia dyari* Belkin, Heinemann and Page, 1970, *Cx. nigripalpus*, *Trichoprosopon* spp., and *Wyeomyia* spp. [57][58][59][60]. In the Pacific coast of Guatemala, the virus was also isolated from *Cx. nigripalpus* [61].

More recently, two studies in Costa Rica reported serological evidence of SLEV infection in free range and captive non-human primates and sloths. For example, a study by Chavez et al. (2021) found homotypic and heterotypic neutralization reactivity to SLEV of 47.6% and 5.9%, respectively, in mantled howler monkeys (*Alouatta palliata* [Gray, 1849]) and spider monkeys (*Ateles geoffroyi* Kuhl, 1820), while another study found a seropositivity of 42% in Hoffman's two-toed sloths (*C. hoffmanni*) and brown-throated sloths (*B. variegatus*) [44][45]. Moreover, a study conducted between 2017 and 2018 in northwestern and southeastern Costa Rica found neutralizing SLEV antibodies in humans, local and migratory birds, and equines [47]. One seroconversion event to SLE, between the rainy and the dry seasons, was also detected in a horse from the province of Guanacaste [47].

2.1.3. Venezuelan Equine Encephalitis

Venezuelan equine encephalitis virus (VEEV) (genus *Alphavirus*, family *Togaviridae*) is a serocomplex of six antigenic subtypes (I–VI). Within subtype I, there are five antigenic variants (AB–F), with antigenic variants IAB and IC associated with epizootic/epidemic activity in equines and humans, while variants ID, IE, and IF and subtypes II–IV are associated with enzootic forest cycles. These enzootic viruses circulate in sylvatic rodent populations within tropical and subtropical forests or swamp habitats continuously transmitted by *Cx.*

(Melanoconion) mosquitoes [62][63]. Most enzootic variants are avirulent in equines, but some strains could cause clinical disease in both humans and equines with a similar clinical presentation to epizootic strains infections [64].

The first VEEV epizootic involving humans and equines in Central America took place between 1969–1970 in Honduras, Guatemala, Nicaragua, El Salvador, and Costa Rica, as part of a major intercontinental epizootic that spread from Central America to northern South America and south Texas, U.S. [65][66]. The virus strain responsible for this epizootic was identified VEEV subtype IB (later reclassified as IAB) and, at the time, was commonly isolated from equines, humans, and several mosquito species, including *Psorophora confinnis* (Lynch Arribalzaga, 1891), *Cx. nigripalpus*, *Cx. (Melanoconion)* sp., *Ma. titillans* (Walker, 1848), *Aedes taeniorhynchus* (Wiedemann, 1821), and *De. pseudes* [65][67][68][69]. Following this epizootic, numerous studies linked the origin of the VEEV epizootic subtype IAB to incompletely innactivated vaccines [70]. Moreover, evidence also suggest that key mutations in enzootic VEEV strains could potentially mediate the emergence of novel epizootic strains [71].

In recent decades, only two enzootic VEEV subtypes have been reported in Central America. The VEEV subtype IE appears to be widely distributed throughout the region while the VEEV subtype ID has been detected only in Panama [41][43][71][72][73]. Recent data suggest that humans infected with these two enzootic subtypes could develop sufficient viremia to potentially infect both Central American enzootic and epizootic VEEV vectors [71][72]. Moreover, experimental competency studies suggest the potential transmission of VEEV subtype ID in Central America by *Ae. aegypti* (Linnaeus, 1762) and *Ae. albopictus* (Skuse, 1894), increasing the possibility that urban transmission cycles could take place in the region [74][75]. It is possible that febrile illness linked to endemic VEEV infections in Latin America are more widespread than previously observed but are vastly unrecognized or misdiagnosed due to the presence of similar dengue-like febrile illnesses and lack of effective public health surveillance systems [71][72].

The first reported human case of enzootic VEEV ID virus infection in Panama took place in the early 1960s near Panama City [76]. Recently, an increase in human VEEV cases in Panama have intensified ecological and epidemiological studies in the region, with several studies reporting the active and stable circulation in humans of both VEEV subtypes ID and IE throughout the country. For example, recent serosurveys have found neutralizing VEEV antibodies levels between 8.5% and 78% across several communities in the Darien region [41][72][73]. Moreover, subtype ID outbreaks in Panama also show higher case fatality rates than those reported during previous subtype IAB epidemics [72].

Field studies in Panama, Costa Rica, Guatemala, Belize, and Honduras have also detected the circulation of enzootic VEEV ID and IE viruses in a variety of vertebrates, including wild rodents (*Zygodontomys brevicauda* [Allen and Chapman, 1893]), *Transadinomys (=Oryzomys) bolivaris* Allen, 1901, *Proechimys semispinosus* [Tomes, 1860], *Melanomys caliginosus* [Tomes, 1860]; *Oryzomys* spp.), opossums (*Didelphis marsupialis* Linnaeus, 1758, and *Marmosa robinsoni* Bangs, 1898; *Philander* spp.), sloths (*Bradypus* spp. and *Choloepus* spp.), sentinel guinea pigs and hamsters, equines, wild and domestic birds, and bats [41][43][73][77][78][79][80][81][82][83]. Moreover, entomological surveys in Central America have identified *Cx. (Melanoconion)* spp., particularly *Cx. (Mel.) taeniopus* Dyar and Knab, 1907, *Cx. (Mel.) ocossa* Dyar and Knab, 1919, and *Cx. (Mel.) panocossa* Dyar, 1923; formerly known as *Cx. (Mel.) aikenii* (Aiken and Rowland, 1906), *Cx. (Mel.)*

vomerifer Komp, 1932, and *Cx. (Mel.) erraticus* (Dyar and Knab, 1906) as the most important enzootic VEEV vectors in the region [71][84].

2.2. Malaria in Central America

Malaria, a febrile disease caused by *Plasmodium* spp. parasites transmitted by *Anopheles* spp. mosquitoes, causes the highest morbidity and mortality compared to any other VBD worldwide [85]. While less than 1% of the 2019 global malaria burden was recorded in the Americas, the region remains endemic for the disease. In Central America, there were nearly 20,000 autochthonous malaria cases recorded in 2019, with cases reported in five out of seven countries within the region: Nicaragua (13,200)), Guatemala (2100), Panama (1600), Honduras (330), and Costa Rica (91) [85]. Almost all these cases were caused by *P. vivax* (Grassi and Feletti, 1890) (74%) and *P. falciparum* (Welch, 1897) (23%), which are transmitted mainly by *An. albimanus* Wiedemann, 1820, *An. pseudopuntipennis* Theobald, 1901, *An. darlingi* Root, 1896, *An. marajoara*-Galvao and Damasceno, 1942, *An. aquasalis* Curry, 1932, *An. albitarsis* Lynch Arribalzaga, 1878, and *An. vestitipennis* Dyar and Knab, 1906, reflecting a remarkable diversity of competent vectors with diverse ecologies and bionomics [85][86][87][88][89][90][91].

For broader context, these 2019 regional case estimates represent more than a 50% reduction from 2010 estimates, when nearly 40,000 cases were recorded, and over 80% reduction from 2000 estimates when more than 340,000 cases were reported [92]. Despite some challenges that persist, current case numbers highlight the remarkable progress and numerous successes achieved by malaria control and elimination programs in the region [93]. For example, El Salvador was recently certified as malaria free while Belize, Costa Rica, Guatemala, Honduras, and Panama have the potential to eliminate malaria within the next five years [94].

While it is important to reflect on this encouraging progress and celebrate recent malaria control successes across Central America, it is equally important for the region not to let its collective guard down. With multiple competent malaria vectors naturally occurring throughout its geography [86], Central America remains vulnerable to the re-introduction of malaria across its entire range. Furthermore, the influx of people from malaria endemic regions of the Americas, both travelers and migrants, as well as international travelers from other malaria endemic regions, creates a low but constant risk of *Plasmodium* parasite re-introduction into areas with greatly reduced local transmission [95][96][97][98]. This risk is exacerbated by the prospects of a changing climate, deforestation, changing land-use patterns, and increasing drug and insecticide resistance [99][100].

2.3. Tick-Borne Diseases in Central America

In Central America, recent studies on the ecology of tick-borne diseases are limited for most countries, and outbreaks and case reports in humans are mostly sporadic and infrequent. However, there is evidence of possible zoonotic human pathogens in ticks and/or vertebrate animals, such as *Anaplasma phagocytophilum* (Foggie, 1949), *Ehrlichia canis* (Donatien and Lestoquard, 1935), *E. chaffeensis* Anderson et al., 1992, *E. ewingii* Anderson et al., 1992, *Rickettsia rickettsii* (Wolbach, 1919), *R. parkeri* Lackman et al., 1965; including the strain Atlantic Rainforest), *R. akari* Huebner et al., 1946, *R. africae* Kelly et al., 1996, *Borrelia burgdorferi* s.l. Johnson et al., 1984, and *Borrelia* sp. (causing tick-borne relapsing fever), as well as several other microorganisms that may infect

ticks and/or vertebrate animals for which pathogenicity is unknown, or that are not known to cause human disease [101][102][103][104][105][106][107][108][109][110][111][112][113][114][115][116][117][118][119][120][121][122][123][124][125][126][127][128][129][130][131][132][133][134]. In humans, the most common tick-borne diseases reported in Central America in the past few decades are spotted fever group rickettsioses and ehrlichioses, followed by isolated reports of probable Lyme disease in which the bacteria were not directly detected or identified [120][122][135][136][137][138][139][140][141]. In addition, there are records of tick-borne relapsing fever and serological evidence of exposure to *R. akari* [105][118][142], while human anaplasmosis and *R. parkeri* spotted fevers have yet to be confirmed in the region. In most cases of outbreak descriptions of human tick-borne diseases, the main vectors and vertebrate reservoirs implicated in local transmission cycles have not been clearly identified.

2.3.1. Rickettsioses

Bacteria of the genus *Rickettsia* (Rickettsiales: family *Rickettsiaceae*) are responsible for various clinical rickettsioses in humans; in the Americas, *R. rickettsii* spotted fever is the most relevant in terms of morbidity and mortality [143]. Tick-borne rickettsioses have been known to occur in Central America since the 1950s [122][144]. The first cases were identified and confirmed by isolation of *R. rickettsii* from humans and ticks in Panama and later in Costa Rica [122][141]. Ecological investigations to identify possible vertebrate hosts and tick vectors have been carried out in these countries throughout the decades. For instance, *Amblyomma mixtum* Koch, 1844 (previously referred to as *A. cajennense*) has been implicated as a probable vector of *R. rickettsii* to humans in Panama and probably Costa Rica [122][141]. This agrees with the information available about transmission of rickettsiae in South America, where several tick species belonging to the "*A. cajennense* species group" are considered vectors of *R. rickettsii* [145][146]. Moreover, the brown dog tick, *Rhipicephalus sanguineus* s.l. (Latreille, 1806), has also been investigated as a possible vector of urban human cases of *R. rickettsii* infection in Panama [122][147]. As for other spotted fever group rickettsiae, there are reports of human disease and outbreaks in Guatemala, Honduras, Nicaragua, although the species was not identified in these cases [148][149][150]. In addition, there are historical records in which antibodies against *R. akari* (or an antigenically similar species) have been detected in humans in Central America, but there are no records of direct detection of the bacterium in ticks or human cases of rickettsial pox [142]. *Rickettsia akari* transmission is usually associated with hematophagous mites, but it has been detected in humans, dogs, and ticks in neighboring Yucatan, Mexico [151][152]. Recently, DNA of *R. africae* was detected in *A. ovale* Koch, 1844 ticks from Nicaragua, but there are no human cases of African tick-bite fever confirmed to this date [124].

2.3.2. Ehrlichiosis and Anaplasmosis

The most relevant species of *Ehrlichia* and *Anaplasma* (Rickettsiales: family *Anaplasmataceae*) in terms of morbidity in humans are *E. chaffeensis* and *A. phagocytophilum*, which cause human monocytic ehrlichiosis and human granulocytic anaplasmosis, respectively [153][154]. Different species of *Ehrlichia* and *Anaplasma* are known to occur in Central America, especially in domestic animals (e.g., *E. canis*, *E. chaffeensis*, *Ehrlichia* sp. H7, *A. platys* (Dumler et al., 2001), *A. phagocytophilum*), while *E. ewingii* have been detected only through DNA in ticks [101][104][106][107][108][109][111][112][113][114][116][123][125][127][155]. In humans, there are several reports of possible ehrlichiosis/anaplasmosis diagnosed by observation of morulae in stained blood smears, by indirect antigen or

antibody detection, without isolation, or by molecular identification of the species [120][136][137][139]. In 2015, *E. chaffeensis* was confirmed in humans by polymerase chain reaction (PCR) and DNA sequencing in Costa Rica [115]. In addition, *E. canis* DNA has also been reported in humans in Costa Rica and in one case in Panama, which was the only one associated with severe disease [121][128]. *Anaplasma phagocytophilum* DNA has been detected repeatedly in ticks and domestic animals, but this species has not been confirmed in human infections [104][106][109][119][125][127]. In areas of North America where human ehrlichiosis and anaplasmosis are common, the principal vectors are *Amblyomma americanum* (Linnaeus, 1758) and *Ixodes* spp. (*I. scapularis* Say, 1821; *I. pacificus* Cooley and Kohls, 1943; and others), respectively, but these ticks are not found in Central America or South America, where the possible vectors are still being determined [156][157][158]. As for *E. canis*, studies have confirmed that *R. sanguineus* s.l. is the vector within the dog population in Central America, in correspondence to what is known for other regions [119][156][159].

3. Impact of Deforestation, Urbanization, and Changing Land Use Patterns on the Ecology of Mosquito and Tick-Borne Diseases in Central America

3.1. Impact on Mosquito-Borne Arboviral Diseases

Major knowledge gaps persist on the enzootic transmission cycles of most arboviral diseases endemic to Central America, including information on geographic distribution, vertebrate reservoir species, mosquito vectors, and human disease risk. Furthermore, little is known about the potential effects of increasing deforestation, forest fragmentation, and urbanization on the ecology and epidemiology of these diseases in the region. While dengue fever has received the most attention throughout Central America [84][160], research on other arboviral diseases in the region have been conducted mostly in Panama and Costa Rica, the countries with highest economic development and significant public health investments in the region [30]. Arboviral diseases present in Central America are of increasing public health concern due to their recent emergence or resurgence and most are considered neglected [6][71][161][162].

Changes in land use can significantly affect mosquito population dynamics, oviposition, abundance, and host-seeking behaviors. Numerous studies have shown that modifications in land use could result in the loss of hosts, predators, and mosquito habitats, which may affect vector population dynamics, abundance, oviposition, and host-seeking behaviors. These environmental modifications could drive mosquito vectors to search for new blood-feeding sources and alternative breeding habitats [3][23][163], promote higher host contact rates, and initiate disease spillover events, introducing new infections into susceptible human populations [164][165][166]. Other factors, such as human migration and urbanization, can significantly impact the distribution and occurrence of arboviruses by driving their emergence or resurgence. Moreover, different sociodemographic factors associated with urbanization, such as social inequality, health care capacity, food safety, population density, and inadequate infrastructure, could be associated with human disease outbreaks [165][167].

Recent studies in the Amazon region have focused on the relationship between deforestation, vector mosquito abundance, and arbovirus outbreaks [163][166][168]. Forest fragments and growing agricultural areas show higher abundance, richness, and diversity of mosquito species. Conversely, mosquito abundance and richness decreased in the urban environment [168][169]. Consequently, anthropophilic species, such as *Ae. aegypti*, could become very efficient vectors in urban areas compared to *Culex* spp. and *Ae. albopictus*, which feed on a broader range of hosts [167]. Moreover, several species that serve as vectors of multiple human pathogens appear to benefit from deforestation, including species found in Central America, such as *An. darlingi, Ae. aegypti*, and *Cx. quinquefasciatus* [164].

Recent studies in central Panama found that mosquito diversity peaks in pristine forest habitats, such as old-growth forests, while the abundance of colonist mosquito vectors (e.g., disturbed-areas specialists) increased significantly in highly disturbed forest sites [170][171]. These differences in mosquito abundances and diversity across various levels of forest disturbance could be attributed to changes in ecological conditions that maybe affecting the quality and availability of larval breeding sites. All together, these results suggest that forest disturbance could drive VBD emergence and increase risk for disease transmission in recently disturbed tropical regions due to the abundance of colonist-vector species. These mosquito vectors tend to be opportunistic feeders, targeting hosts that are readily available [172]. Moreover, a study by Bayles et al. (2020) in Costa Rica found that areas with a high proportion of anthropogenic-altered landscapes, especially in areas with a high degree of agricultural intensification, have the highest transmission risk for VBDs, such as ZIKV, compared to protected areas [173].

The relationship between yellow fever and deforestation was initially established in the first half of the 20th century when ecological observations were made regarding the ability of *Haemagogus* mosquitoes to survive in forests with some degree of deforestation pressure [174]. In recent years, the number of cases of sylvatic yellow fever cases in Brazil have increased significantly in epizootic or transition areas and have been linked to large-scale deforestation and forest fragmentation within urbanized settings [175][176]. The main vectors of YFV in the Americas, *Haemagogus* spp. and *Sabethes* spp., share similar ecological and bionomic characteristics, including acrodendrophilous behavior and opportunistic feeding habits [177]. Studies on these vectors have shown that they are more abundant in sites with lower forest cover suggesting that forest fragmentation could be a critical factor in determining their presence [178]. For example, *Sa. chloropterus* was recently found in deforested areas adjacent to a primary forest in Costa Rica [179], indicating that deforestation could increase microhabitats for mosquito colonization, such as phytotelma and tree holes, typically used by some enzootic yellow fever vectors [180][181][182]. Other studies suggest that deforestation could be pushing YFV vectors to adapt their feeding habits due to pressure from habitat disturbance. For instance, *Hg. janthinomys* Dyar, 1921 and other YFV vectors in Brazil were frequently observed descending to ground level in the presence of humans conducting wood extraction activities or when the non-human primate population number is small [183]. Moreover, others have found that their feeding behavior is more prolonged and aggressive at ground level than in the forest canopy [184]. *Haemagogus* spp. and *Sabethes* spp. are also eclectic feeders able to shift their host seeking among various wild or domestic animals and human hosts according to their local availability, frequently moving vertically from the top of trees to feed at ground level. However, it is not clear if this eclectic feeding behavior is innate or if it has been driven by an increase

in deforestation in regions where this species occurs [185][177][186][187]. Although there is no current evidence of active YFV transmission by *Ae. aegypti* or *Ae. albopictus* [188], experimental studies have determined that local domestic and peridomestic mosquito populations may be competent vectors for YFV strains circulating in South America [189].

The recent reemergence and dispersion of yellow fever in Brazil, potentially driven forest fragmentation near urbanized areas [176], raises the possibility of its resurgence elsewhere in the continent, including parts of Central America, where these sylvatic vectors are also present and similar ecological disturbances are taking place. The urbanization of yellow fever is one of the biggest infectious disease threats in Latin America [190]. The establishment of urban cycles, via *Ae. aegypti and Ae. albopictus*, could be catastrophic since these vectors are widely distributed throughout the region [183]. Most people living in urban areas of Latin America are not vaccinated, which could lead to high disease incidence that could further spread transmission [191]. It has been estimated that the proportion of infected people during an epizootic could be up to 29% under these conditions [192].

Deforestation can also affect non-human primate reservoirs of YFV and other arboviruses in the Americas. Recent studies have found a strong association between non-human primate diversity, their mobility patterns through forests, and the presence of human yellow fever cases [193][194]. The importance of howler monkeys (*Alouatta* spp.) as main reservoir host for several arboviruses, such as YFV, and their behavior must be further explored. In Costa Rica, *Alouatta* monkeys spend most of their day (77%) in an inactive state while a smaller proportion of their time is spent moving through the forest, feeding, or engaging in social behavior. In contrast, capuchin monkeys (*Cebus* spp.) spend most of the time (70–80%) day foraging or conducting other active behaviors [195]. The low activity budget of *Alouatta* monkeys could make them more susceptible to mosquito bites and YFV transmission, given that host movement has an important influence on the R0 in vector-borne disease systems [196]. Furthermore, *Alouatta* spp. can adjust their behavior to accommodate different feeding strategies as their forest habitat changes. These monkeys are found in both modified and undisturbed habitats throughout their distribution in Mexico and Central America. Their ability to adapt to changing environments is evidenced in their continued presence in regions where white-faced capuchins and spider monkeys no longer exist [197]. However, recent studies indicate that this behavior may not be spatially consistent. A recent study by Schreirer et al. (2021) in Costa Rica found that *A. palliata* do not appear to adjust their activity or spatial cohesion patterns in response to anthropogenic edge effects due to forest fragmentation, suggesting that these monkeys exhibit less behavioral flexibility than *A. palliata* at some other sites [198]. The high susceptibility of *A. palliata* to YFV infection and movement within disturbed or fragmented habitats could increase the risk of arbovirus transportation and exposure to humans in urbanized forest edges [199][200][201][202].

In Latin America, the relationship between DENV and deforestation has not been clearly established or evidence is scarce. For instance, studies conducted in the Brazilian Amazon have found no association [203][204]. Studies conducted in other endemic DENV regions suggest that land use changes following deforestation (e.g., agriculture, settlements, or road construction) have been identified as significant dengue fever risk factors [164][205]. An increase in human population densities benefits DENV and its vectors through the establishment of artificial breeding habitats (i.e., water storage) and more frequent contact with susceptible populations, thus increasing the risk for

virus transmission in rural and urban settings [206]. Other factors, such as proximity to paved roads and house clustering, could also promote breeding habitats [207]. The expansion of urban centers and bordering rural areas closer to the forest edge increases the chance of *Ae. aegypti* dispersal and colonization. For example, a study in the Peruvian Amazon by Guagliardo et al. (2014) found that the geographic spread of *Ae. aegypti* is driven by human transportation networks along rivers and highways in proximity to the city [208]. In this region, urban development and the availability of oviposition sites appear to contribute to the colonization of *Ae. aegypti* along roads. Moreover, unintentional transport of mosquitoes on boats disperses their populations over long distances into rural, riverine communities [208].

Forest fragmentation driven by land use changes could also facilitate movement of adult mosquito vectors between communities. For instance, a mark-release-recapture study by Russell et al. (2005) reported that released *Ae. aegypti* exhibit nonrandom patterns of dispersal with larger proportion of mosquitoes being recaptured along a corridor with heavy shading from trees and vegetation [209]. In Central America, several studies have been conducted using several vegetation indexes to evaluate correlations between vegetation coverage and dengue incidence. For example, a study by Fuller at al. (2009) was able to explain up to 83% of the variability in weekly cases of dengue fever and dengue hemorrhagic fever in Costa Rica between 2003 and 2007 based on vegetation indexes [210]. In another study, high dengue fever incidence correlated spatially with high temperature, low altitude, and a high vegetation index [211]. Moreover, ovitrap egg counts are also associated with vegetation indexes, which are dependent on temperature and rainfall, well known factors affecting vector abundance [212][213][214]. Interestingly, other studies conducted in Central America revealed that dengue cases may be directly associated with tree cover and non-forested areas and inversely associated with built areas, probably because more larval habitats are available in larger, tree-covered outdoor areas with vegetation during rainfall episodes [215][216][217].

In Central America, there are increasing concerns regarding the expansion of *Ae. albopictus* throughout the region. This important arbovirus vector species was initially detected in El Salvador, Honduras, and Guatemala in 1995; later in Panama and Nicaragua in 2002–2003; and Costa Rica and Belize between 2007 and 2009 [218][219][220][221] [222]. During the last decade, its distribution in Central America has expanded which is evidenced by surveillance and field study reports [223][224][225][226], as well as by population genetic studies in Panama and Costa Rica [227]. Recent studies in Costa Rica demonstrate the adaptive capacity of this species to changes in land use and expansion of urbanized areas. For example, a study by Calderon-Arguedas et al. (2019) detected all four DENV serotypes in both *Ae. albopictus* female and male adults, and larvae associated with commercial pineapple farming, suggesting local horizontal and vertical transmission of DENV in the region [228]. Moreover, during these studies, most adult *Ae. albopictus* were collected within forest galleries bordering pineapple fields, which may indicate that forest edge habitats could serve as ecological refuges for this species in DENV endemic areas [229] [228].

The movement of pathogens from sylvatic to amplified human transmission cycles in rural and urban settings (e.g., disease spillover) have been commonly reported in the literature; however, important questions on specific mechanisms still linger [230]. These events are typically associated with human and animal populations near forest edges, leading occasionally to urban cycles involving peridomestic vectors [168][231]. Arboviral disease spillover

events in Central America are possible considering that these viruses share several ecological characteristics, including vectoring by anthropophilic mosquitoes in urban and rural transmission cycles, use of humans as main reservoirs, and their occurrence in habitats associated with expanding agriculture and urbanization near forest edges [232][233][234]. Several studies in Central America suggest the potential for disease spillover of sylvatic arbovirus transmission cycles into rural and urbanized areas. For example, in Panama, evidence suggests that human cases of VEEV infections are associated with spillover infections from an enzootic cycle involving sylvatic rodents and *Cx. (Melanoconion)* spp. mosquitoes [41]. Recent clusters of human cases have occurred in Darien and Panama Provinces near rainforest and swamp habitats. In addition, entry of humans into forest galleries appears to be a risk factor for VEEV transmission [41][72]. Other studies suggest frequent occurrence of spillover events associated with other sylvatic arboviruses in Central America, such as MADV and MAYV [41][77][161][162][235].

Conversely, spillback (e.g., reverse zoonosis) involves the movement of pathogens from urban/rural transmission cycles to sylvatic cycles between wild non-human primates and forest mosquitoes [236]. In the Americas, spillback events have been reported in several countries, including Argentina, Brazil, Colombia, and French Guyana, involving DENV, ZIKV, YFV, CHIKV, neotropical wild primates, and known mosquito vectors [232][230][236][237]. A recent study carried out in Costa Rica found evidence of SLEV, WNV, and DENV seroprevalence in the primate species *A. palliata*, *A. geoffroyi*, and *S. oerstedii* (Reinhardt, 1872). Based on the collection of seropositive samples coinciding temporarily and spatially with peaks of infections in human populations, their study concluded that DENV exposure in these monkeys occurred through bidirectional human–wildlife contact or bridging vectors [44].

The ecological mechanisms by which forest disturbance triggers disease spillback events are still poorly understood, which makes it difficult to predict disease risk scenarios for future outbreaks in human-altered forest habitats and the potential establishment of sylvatic transmission cycles [230][236]. In Central America, where arbovirus-susceptible wild primates and known mosquito vectors are present (e.g., *Haemagogus* spp., *Sabethes* spp.), the occurrence of any one or a combination of epidemiological factors that could drive transmission is highly plausible, considering the high level of deforestation, extensive land use changes, and accelerating development of human settlements near forest edges. It is also possible that immunity of monkeys against active sylvatic arboviruses, such as YFV, could inhibit infections by DENV and ZIKV and, thus, evade the emergence of sylvatic cycles [236]. However, considering that YFV epizootics have not been reported in Central America in decades, cross-immunity to flaviviruses in non-human primates in the region may not be sufficient to suppress other arboviral infections.

Several mosquito species distributed throughout Central America could serve as bridging vectors and initiate spillback events due to their bionomic plasticity. For example, *Ae. albopictus* is found predominantly in urban areas, but also spreads into rural, semi-rural, and forest areas, which could potentially drive arboviruses, such as ZIKV, DENV, and CHIKV, into sylvatic transmission cycles [230][236]. Moreover, sylvatic vectors like *Haemagogus* spp. could also serve as bridging vectors since they can be opportunistic feeders, frequently imbibing on humans and monkeys, in environmentally disturbed regions along forest edges [238][177][187][239]. These mosquito vectors tend to be opportunistic feeders, targeting hosts that are immediately available [172]. This evidence demonstrates that

disease spillover is not a random process but may be the result of forest degradation increasing the likelihood of contact between humans and mosquito vectors in forest-altered sites [170][171].

Both spillover and spillback events occur due to increasing human activities near adjacent forest areas and where humans closely interact with wild animals and their pathogens, facilitating the "jump" or "shift" of new pathogens between different host species [166]. Despite our current knowledge of these diseases, it is still difficult to predict the risk for future outbreaks since the ecological mechanisms that drive spillovers and spillbacks in human-modified forest habitats are not well defined. Considering the accelerated rate of deforestation, unplanned urbanization and agricultural expansion near forest edges, high endemicity of multiple arboviruses, and close human contact with wildlife and vectors taking place in Central America, there is a tangible possibility that spillback and spillover events may be more frequent than reported and may surge in the future. Therefore, it is critical that more intense epidemiological and ecological monitoring systems are established in the region to help predict future epizootics and epidemics.

3.2. Impact on Malaria

In recent decades, compelling evidence linking deforestation and land use changes with malaria transmission and anopheline vector ecology in Central America have been demonstrated [99][240][241][242]. Deforestation and land use changes can have profound anthropogenic environmental effects on malaria transmission. In general, evidence shows that, in the Americas, deforestation leads to an increased potential for malaria transmission, particularly in communities where the vector *An. darlingi* is present [243][244][245][246][247]. Working specifically in the Darien Province of Panama, Loaiza et al. (2017) correlated increased malaria incidence rates with extensive changes in landscape, including deforestation, and an associated expansion of *An. darlingi* habitat. These findings were recently supported when *An. darlingi* was implicated as an important vector of *P. vivax* in the Darien region [170][248]. Studies in other parts of Latin America have found that the human-biting activity of *An. darlingi* is more intense in areas associated with deforestation and road development [249], while a study by Loaiza et al. (2008) found that the Central American malaria vectors, *An. vestitipennis* and *An. neivai* Howard, Dyar and Knab, 1913 are closely associated with specific forms of vegetation and land-use practices in Panama [87].

Other studies have suggested that agricultural land use changes and environmental pollution can significantly alter natural malaria vector habitats and even affect mosquito diversity driving disease prevalence and more dominant vector populations. For instance, a study by Chapin and Wasserstrom (1981) showed that, as early as the 1970s, expanding the acreage used for cotton cultivation, and the associated increases in pesticide use in Guatemala, Nicaragua, and El Salvador, correlated with both the emergence of DDT resistance in local malaria vectors and rapid increases in annual malaria case incidence rates, in some cases three times greater than previously recorded [250]. Similar findings were noted in Belize, where malathion use in sugarcane cultivation was associated with malathion resistance in local *An. albimanus* populations [251]. Another compelling case study from Belize highlights how phosphorous runoff from sugarcane cultivation in proximity to marshlands increases the amount of dense cattail (*Typha domingensis*) marsh habitat favored by *An. vestitipennis*, the most efficient malaria vector in Northern Belize, and is associated with higher larval densities [252][253]. Interestingly, a strategy to reduce this cattail habitat

through environmental management (e.g., mowing and burning) to control *An. vestitipennis* populations in the area was marginally successful, with one important caveat: while the habitat management strategies significantly reduced *An. vestitipennis* larval populations, the resulting altered marshland habitats proved suitable for another important local vector species, *An. albimanus*, whose populations increased [254]. Changes in landscape structure, in the form forest cover percentage and forest density, could also lead to dominant vector populations, such as *An. darlingi*, increasing the risk for malaria transmission [255].

Another aspect of environmental change, like urbanization, is the global expansion of invasive malaria vectors, like *An. stephensi* Liston, 1901, and its potential implications for malaria control and elimination throughout Central America. Often described as a highly competent malaria vector able to breed in human-made containers, *An. stephensi* is known to establish and sustain outbreaks of urban malaria in previously malaria-free regions [256][257] [258]. While the arrival and establishment of *An. stephensi* in Central America is probably less likely than in Africa or other parts of Asia [256], it is nonetheless realistic to consider the possibility. Regional receptivity to the vector and its potential to impact local malaria control and elimination initiatives is poorly understood but potentially worrisome given the rapid urbanization of population centers, the high abundance of peridomestic habitats, its invasive potential, and intense human migration from malaria-endemic regions through Central America [259][260].

Although the risk for malaria transmission in Central America has been steadily declining in recent years, the intensification of human migration and illegal drug trafficking throughout the region, which are driving deforestation and land use changes, have the potential to reignite the resurgence of malaria in the region [241]. Currently, the La Mosquitia tropical forest region, located between Honduras and Nicaragua, is one of the most malaria endemic locations in Central America [99]. Although this region is part of the protected MBC, it continues to be threatened by illegal drug smuggling, cattle ranching, logging, and land grabbing [22][261][262]. The recent increase in malaria cases in Panama, Costa Rica, and Nicaragua, especially of the more severe form, *P. falciparum*, raises concerns about the possibility of eradicating malaria in those countries in the next few years. This recent resurgence has been linked to imported cases from foreign migrants passing through the region [263]. While numerous findings highlight the effects of land use and micro and macro habitat changes have on of malaria vectors and disease risk across Central America, it is also evident that more research into these complex relationships and interactions is desperately needed. An improved understanding of anopheline vector ecologies will help inform on how malaria transmission dynamics might change over time and guide future malaria control and elimination activities in the region.

3.3. Impact on Tick-Borne Diseases

Studies in different areas of the world have demonstrated that deforestation practices and forest fragmentation could both decrease and modify biodiversity, which may lead to a greater abundance of fewer vertebrate species and an increased prevalence of their associated ticks that can thrive in modified habitats [264][265][266][267][268][269] [270]. In addition, some domestic animals, such as dogs and horses, may act as bridges for ticks and zoonotic pathogens from wildlife to humans, resulting in disease spillover events into urban environments [271][272][273][274]. It is also important to consider environmental factors, such as temperature, precipitation and humidity, critical to host

and tick survival, which could also change in modified environments. Each host, vector, and pathogen has its own requirements, which must be met to ensure their establishment and survival [267][275][276][277][278][279].

Zoonotic pathogens may resurge depending on the conditions present, such as availability of humans and other domestic animals, tick vectors, and environmental conditions. When conditions are optimal and disease vectors and suitable vertebrate reservoir hosts become abundant, pathogen transmission risk may increase. Decreased contact between ticks and vertebrate hosts not relevant in pathogen transmission can also increase disease risk, which could cause a dilution effect. However, the mechanisms of this effect depend on scale and overall context, including local land use changes, climate/microclimate conditions, host communities, and their interactions [268][270]. Therefore, if humans encounter tick vectors in these deforested or disturbed areas, there may be a greater risk of zoonotic disease transmission.

In general, the scientific literature concerning tick-borne diseases in Central America focuses on the distribution or presence of tick species and their associations with vertebrate hosts and reports of human infections by specific pathogens (for examples see [103][104][106][108][111][113][114][117][119][123][124][125][127][129][131][132][133][134][148][155][280][277][281][282][283][284][285][286][287]). However, very few studies in the region have focused on the influence of land use changes on pathogen and vector ecology. Except for recent studies in Panama [278][288][289], no other published reports in the scientific literature have directly and specifically investigated the effects of forest modification, deforestation, or land use changes on tick species, or on pathogen transmission dynamics.

Among the few studies conducted in the region, one conducted in the Chiriquí province of Panama aimed to identify environmental predictors of tick burdens on dogs, as well as environmental predictors of pathogens in these ticks, including vegetation cover and land use change [278]. Although the most relevant predictor of tick prevalence and abundance was elevation, a decrease in vegetation cover linked to increased urbanization, was also associated with the highest tick prevalence and abundance. This is probably due to the close relationship of *R. sanguineus* s.l., the most common dog tick in the region, with human dwellings [278]. In areas with higher vegetation cover and less urbanization, other tick species became relevant, including *A. ovale* and *I.* c.f. *boliviensis*. Therefore, deforestation linked to increased urbanization appears to hinder survival and establishment of ticks, such as *A. ovale* and *I.* cf. *boliviensis*, while benefitting *R. sanguineus* s.l. [278]. In areas where *R. sanguineus* s.l. may act as a vector of zoonotic pathogens, decreases in forest cover and urbanization may increase transmission risk to humans.

Another study in Panama investigated tick diversity in a gradient of decreasing disturbance (low trees and shrubs, secondary forest, secondary forest crossed by a creek, and secondary–primary transition forests also crossed by a creek) and increasing forest cover along the 17 km Oleoducto trail in Soberania National Park [288]. Results showed that the most disturbed site had fewer tick–host interactions, compared with the other sites, and showed low tick diversity and few potential hosts [288]. Notably, this site included more *A. mixtum* ticks, which are common in diverse environments, including disturbed landscapes. Moreover, secondary and transition forests had a higher diversity of tick species and tick-host interactions, including a high abundance and diversity of birds and small mammals as well as several medium and large sized mammals that could serve as hosts for different tick species

[288]. Therefore, this study suggests that a decrease in tick and host diversity may be a consequence of deforestation and forest disturbances in Central America. If these tick populations and their small mammal hosts are relevant in zoonotic transmission, deforestation in the region may also increase the risk for human infections due to diversity loss [268][270].

Additionally in Panama, a recent study investigated both vertebrate and tick communities in forest fragments, specifically in forested islands and peninsulas in the Barro Colorado Nature Monument, which were formed as a result of of damming the Chagres River about a century ago [289]. Its main findings indicate that tick species richness and abundance in this area increases according to the availability of vertebrate host species richness and wildlife biomass, which is higher in larger forest patches. In addition, tick species that have a broad range of vertebrate hosts as adults (e.g., generalists) increase in abundance when host diversity and specialist tick species is low [289]. Therefore, when human activities cause forest fragmentation, there is a decrease in wildlife biodiversity, while smaller sized tick hosts and generalist tick species may become more abundant. When these include possible reservoirs and vectors of pathogens, the risk of transmission between vertebrate species, including humans, can also increase [270].

Other studies in Panama have documented that most local species of ticks are host- specific as adults or at least they are associated with taxonomically related vertebrate species [290]. Therefore, a notable change in host diversity driven by deforestation and other land use changes could result in significant changes in local tick populations and disease transmission dynamics. For instance, the most common ticks on bovines in Central America are *Rhipicephalus microplus* (Canestrini, 1888) and *Amblyomma mixtum* (formerly cited as *Boophilus microplus* and *A. cajennense*, respectively), whereas horses are usually parasitized by *Dermacentor nitens* Neumann, 1897 (=*Anocentor nitens*) and *A. mixtum* [101][104][129][275][277][282][291][292][293]. Depending on the specific area, other frequently found species include *A. maculatum* Koch, 1844, *A.* cf. *oblongoguttatum* Koch, 1844, and/or *A. parvum* Aragão, 1908, among others [282][292][293]. Therefore, deforestation associated with cattle ranching may increase local populations of these ticks driven by changes in host abundance if other environmental conditions are suitable for ticks to complete their development. The case of *A. mixtum* is of particular interest since this species is considered a generalist biter frequently feeding on humans in areas of Central America [146][294][277][281][284][285][295]. This tick species has been identified as a vector of *R. rickettsii* in Costa Rica and Panama [107][277][285][296]. Moreover, there is molecular evidence of other pathogens detected in *A. mixtum* in Central America, including *E. chaffeensis* [107]. *Amblyomma maculatum* is the main vector of *R. parkeri*, which has been reported in Belize [287][297]. It is also relevant to note that there are additional reports of bovines and equines infected by *A. phagocytophilum* and detections of *E. chaffeensis*, *E. ewingii*, and/or *A. phagocyophilum* DNA in unidentified *Amblyomma* sp., *D. nitens*, and *R. microplus* collected from horses and cows in Guatemala and Panama [104][106]. Furthermore, *R. microplus* and *D. nitens* can also parasitize white tail deer (*Odocoileus virginianus* [Zimmermann, 1780]) in areas where they coexist with cattle and horses [281][298][299]. In countries like Brazil, infection of *R. rickettsii* in humans has been associated with a shift from predominantly rural to a more urban transmission in Rio de Janeiro where ticks like *D. nitens*, *R. microplus*, and *A. sculptum* Berlese, 1888 are common on horses and cattle [271]. In this area, *A. sculptum* is also common on wild animals and it is considered a generalist biter and the most important vector of *R. rickettsii*. In Central America, an increase in the abundance of cattle,

horses, and *A. mixtum* (and other generalist ticks) in areas where there is already local transmission of zoonotic pathogens may result in an increased risk of human exposure to these potential vectors and pathogen infection.

Rhipicephalus sanguineus s.l. is the most common tick on domestic dogs in Central America, although it is not indigenous to this region [111][119][277][282][300][301][302]. Despite its marked preference toward dogs, this tick also bites humans and can do so frequently in rural and urban areas in Central America [294][295]. Considering that these ticks are usually found in urban environments or areas that have been disturbed, deforestation and other land use changes leading to human settlements and increased dog populations could result in the establishment of this species where it was not present before human activity, as it has been observed in Panama [278]. In addition to being the main vector of *E. canis* in the region, *R. sanguineus* s.l. may be implicated in the transmission of *A. phagocytophilum*, given reports of infection in dogs in Costa Rica and Nicaragua and the detection of its DNA in this tick species in Costa Rica and Panama [109][119][125][127][277]. Moreover, *R. sanguineus* s.l. has been identified as responsible for urban outbreaks of severe human rickettsiosis by *R. rickettsii* in Mexico, and it may have been associated with a human case in Panama [147][303]. Considering the presence of these bacteria in Central America, an increase in dog populations in this region may facilitate contact with pathogen transmission cycles in wildlife and their ticks. In Rio de Janeiro, Brazil, recent investigations close to the Pedra Branca State Park have found *R. rickettsii* in *R. sanguineus* ticks as well as a higher exposure of dogs to *Rickettsia* spp. in recently urbanized areas, compared to rural and non-endemic areas [304]. This supports the hypothesis that interactions of dogs with wildlife and their ticks may eventually lead to the establishment of urban transmission cycles among dogs and *R. sanguineus* s.l. vectors and the possibility of a higher risk to humans mediated by contact with *R. sanguineus* s.l.

Amblyomma ovale is another tick species in Central America that can be found feeding on dogs in rural areas, urban periphery, and nearby human-disturbed forests areas or frequently accessed forest habitat (e.g., for hunting) [111][124][277][281][282][285][300][301][305][306]. Although it is not frequently found in highly urbanized areas, this species readily bites humans in rural landscapes of Central America and it is the main vector of *R. parkeri* (strain Atlantic rainforest) in other parts of Latin America, including Colombia, Brazil, and Argentina [294][277][295][297][306][307][308][309]. The presence of this strain of *R. parkeri* and *R. africae* in *A. ovale* has been recently reported in Belize and Nicaragua, respectively [124][286]. Moreover, several studies in the Americas, such as the U.S. and Brazil, have found that people who own or hunt with dogs that are in contact with wildlife and forest areas are more exposed to wildlife ticks, including vectors of zoonotic pathogens, that could also parasitize dogs [272][273]. Therefore, a consequence of increased human settlements in proximity to forest habitats and incursions into nearby forests, along with their domestic dogs for company, herding, hunting, and other activities, may increase human and dog exposure to *A. ovale* and other ticks and wildlife pathogens in disturbed and forest areas of Central America.

Land use changes leading to human settlements may also increase contact of humans with argasid ticks and the pathogens that they carry. Tick infestations in domestic environments and human bites by *Ornithodoros* spp. in Central America can occur due to bats, rodents, birds, or other small animals accessing the interior of buildings or seeking refuge close to buildings [118][280][294][310][311][312]. In Panama, *O. talaje* and *O. rudis* were implicated as vectors of an unidentified *Borrelia* sp. that causes tick-borne relapsing fever in the area, but other common species (e.g., *O. puertoricensis* [Fox, 1947]) may also be competent vectors of *Borrelia* spp. [118][312]. In other regions of the

world where cases of tick-borne relapsing fever occur, infection usually takes place within houses or buildings infested with argasid ticks [118][313]. In Central America, the expansion of human settlements, especially informal and unplanned housing, could offer adequate refuge for argasid ticks and small animals, such as rodents and bats, placing humans in close proximity to these ticks and increasing the risk for pathogen transmission.

There are other potential tick-borne pathogens in Central America that have been directly or indirectly documented in wild animals or ticks, but not in humans. For example, DNA of *A. phagocytophilum* and *B. burgdorferi* s.l. was recently detected in *I. tapirus* Kohls, 1956 and in *I.* cf. *boliviensis*, respectively, in Panama [133]. Although there is evidence of possible exposure to *B. burgdorferi* s.l. in dogs and humans in Central America, the bacterium involved has not been clearly identified [127][135][140][314]. However, species such as *I.* c.f. *boliviensis* have the potential to transmit enzootic pathogens and may parasitize domestic animals, including dogs and even humans [125][294][281] [300][301][315]. This generalist feeding behavior may allow them to act as bridge vectors in peridomestic environments, especially near disturbed forests and rural settings near forests. The transmission cycles of *A. phagocytophilum* and *B. burgdorferi* s.s. typically involve wild animals, such as white tail deer and rodents, and tick populations that may increase in abundance due to forest disturbances, conservation measures, and reforestation practices [264][266][269][316][317].

References

1. Bos, R. New Approaches to Disease Vector Control in the Context of Sustainable Development. Cad. Saúde Públ. 1992, 8, 240–248.

2. Gubler, D.J. The Global Threat of Emergent/Re-Emergent Vector-Borne Diseases. Vector Biol. Ecol. Control 2010, 39–62.

3. Burkett-Cadena, N.; Vittor, A.Y. Deforestation and Vector-Borne Disease: Forest Conversion Favors Important Mosquito Vectors of Human Pathogens. Basic Appl. Ecol. 2018, 26, 101–110.

4. WHO (World Health Organization). Vector-Borne Diseases Factsheet. Available online: https://www.who.int/news-room/fact-sheets/detail/vector-borne-diseases (accessed on 5 October 2021).

5. Wilke, A.B.B.; Beier, J.C.; Benelli, G. Complexity of the Relationship between Global Warming and Urbanization—An Obscure Future for Predicting Increases in Vector-Borne Infectious Diseases. Curr. Opin. Insect Sci. 2019, 35, 1–9.

6. Hotez, P.J.; Damania, A.; Bottazzi, M.E. Central Latin America: Two Decades of Challenges in Neglected Tropical Disease Control. PLoS Negl. Trop. Dis. 2020, 14, e0007962.

7. Oliveira, W.K. Increase in Reported Prevalence of Microcephaly in Infants Born to Women Living in Areas with Confirmed Zika Virus Transmission During the First Trimester of Pregnancy—Brazil, 2015. MMWR Morb. Mortal. Wkl. Rep. 2019, 65.

8. Lorenz, C.; Azevedo, T.S.; Virginio, F.; Aguiar, B.S.; Chiaravalloti-Neto, F.; Suesdek, L. Impact of Environmental Factors on Neglected Emerging Arboviral Diseases. PLoS Negl. Trop. Dis. 2017, 11, e0005959.

9. Rosenberg, R. Vital Signs: Trends in Reported Vectorborne Disease Cases—United States and Territories, 2004–2016. MMWR Morb. Mortal. Wkl. Rep. 2019, 67, 496–501.

10. Morse, S.S. Factors in the Emergence of Infectious Diseases. Emerg. Infect. Dis. 1995, 1, 7–15.

11. Institute of Medicine. Microbial Threats to Health: Emergence, Detection, and Response. In Microbial Threats to Health; National Academies Press (US): Washington, DC, USA, 2003.

12. Jones, K.E.; Patel, N.G.; Levy, M.A.; Storeygard, A.; Balk, D.; Gittleman, J.L.; Daszak, P. Global Trends in Emerging Infectious Diseases. Nature 2008, 451, 990–993.

13. Hassell, J.M.; Begon, M.; Ward, M.J.; Fèvre, E.M. Urbanization and Disease Emergence: Dynamics at the Wildlife–Livestock–Human Interface. Trends Ecol. Evol. 2017, 32, 55–67.

14. Petersen, E.; Petrosillo, N.; Koopmans, M.; Beeching, N.; di Caro, A.; Gkrania-Klotsas, E.; Kantele, A.; Kohlmann, R.; Koopmans, M.; Lim, P.-L.; et al. Emerging Infections—An Increasingly Important Topic: Review by the Emerging Infections Task Force. Clin. Microbiol. Infect. 2018, 24, 369–375.

15. Gratz, N.G. Emerging and Resurging Vector-Borne Diseases. Annu. Rev. Entomol 1999, 44, 51–75.

16. Swei, A.; Couper, L.I.; Coffey, L.L.; Kapan, D.; Bennett, S. Patterns, Drivers, and Challenges of Vector-Borne Disease Emergence. Vector Borne Zoonotic Dis. 2020, 20, 159–170.

17. Juliano, S.A.; Philip Lounibos, L. Ecology of Invasive Mosquitoes: Effects on Resident Species and on Human Health. Ecol. Lett. 2005, 8, 558–574.

18. Medlock, J.M.; Hansford, K.M.; Schaffner, F.; Versteirt, V.; Hendrickx, G.; Zeller, H.; van Bortel, W. A Review of the Invasive Mosquitoes in Europe: Ecology, Public Health Risks, and Control Options. Vector Borne Zoonotic Dis. 2012, 12, 435–447.

19. Özer, N. Emerging Vector-Borne Diseases in a Changing Environment. Turk. J. Biol. 2005, 29, 125–135.

20. Dantas-Torres, F. Climate Change, Biodiversity, Ticks and Tick-Borne Diseases: The Butterfly Effect. Int. J. Parasitol. Parasites Wildl. 2015, 4, 452–461.

21. Sheela, A.M.; Ghermandi, A.; Vineetha, P.; Sheeja, R.V.; Justus, J.; Ajayakrishna, K. Assessment of Relation of Land Use Characteristics with Vector-Borne Diseases in Tropical Areas. Land Use Policy 2017, 63, 369–380.

22. Devine, J.A.; Wrathall, D.; Aguilar-González, B.; Benessaiah, K.; Tellman, B.; Ghaffari, Z.; Ponstingel, D. Narco-Degradation: Cocaine Trafficking's Environmental Impacts in Central America's Protected Areas. World Dev. 2021, 144, 105474.

23. Gottdenker, N.L.; Streicker, D.G.; Faust, C.L.; Carroll, C.R. Anthropogenic Land Use Change and Infectious Diseases: A Review of the Evidence. EcoHealth 2014, 11, 619–632.

24. Berglee, R. World Regional Geography: People, Places, and Globalization v2.0; FlatWorld: Boston, MA, USA, 2017.

25. Myers, N.; Tucker, R. Deforestation in Central America: Spanish Legacy and North American Consumers. Environ. Hist. Rev. 1987, 11, 55–71.

26. De Clerck, F.A.J.; Chazdon, R.; Holl, K.D.; Milder, J.C.; Finegan, B.; Martinez-Salinas, A.; Imbach, P.; Canet, L.; Ramos, Z. Biodiversity Conservation in Human-Modified Landscapes of Mesoamerica: Past, Present and Future. Biol. Conserv. 2010, 143, 2301–2313.

27. Kim, D.-H.; Sexton, J.O.; Townshend, J.R. Accelerated Deforestation in the Humid Tropics from the 1990s to the 2000s. Geophys. Res. Lett. 2015, 42, 3495–3501.

28. Douglass Biodiversity and Deforestation in Central America. Available online: https://storymaps.arcgis.com/stories/b21e1154ae354c9d8629626a5f80ee88 (accessed on 20 September 2021).

29. Tellman, B.; Sesnie, S.E.; Magliocca, N.R.; Nielsen, E.A.; Devine, J.A.; McSweeney, K.; Jain, M.; Wrathall, D.J.; Dávila, A.; Benessaiah, K.; et al. Illicit Drivers of Land Use Change: Narcotrafficking and Forest Loss in Central America. Glob. Environ. Chang. 2020, 63, 102092.

30. Maria, A.; Acero, J.L.; Aguilera, A.I.; Garcia Lozano, M. Central America Urbanization Review: Making Cities Work for Central America; World Bank: Washington, DC, USA, 2017; ISBN 978-1-4648-0985-9.

31. OECD. Health at a Glance: Latin America and the Caribbean 2020. Health at a Glance: Latin America and the Caribbean 2020; OECD Publishing: Paris, France, 2020.

32. Kramer, L.D.; Ciota, A.T.; Kilpatrick, A.M. Introduction, Spread, and Establishment of West Nile Virus in the Americas. J. Med. Entomol. 2019, 56, 1448–1455.

33. Cruz, L.; Cardenas, V.M.; Abarca, M.; Rodriguez, T.; Reyna, R.F.; Serpas, M.V.; Fontaine, R.E.; Beasley, D.W.C.; da Rosa, A.P.A.T.; WEAVER, S.C.; et al. Serological Evidence of West Nile Virus Activity in El Salvador. Am. J. Trop. Med. Hyg. 2005, 72, 612–615.

34. OIE (Organization for Animal Health). Update on West Nile Virus (WNV) in Belize—Vol. 2, No. 11 (18 March 2004)—PAHO/WHO|Pan American Health Organization. Emerg. Infect. Dis. Wkl. Updat. 2004, 2. Available online: https://www.paho.org/en/documents/update-west-nile-virus-wnv-belize-vol-2-no-11-18-march-2004 (accessed on 15 November 2021).

35. Gubler, D.J. The Continuing Spread of West Nile Virus in the Western Hemisphere. Clin. Infect. Dis. Off. Pub. Infect. Dis. Soc. Am. 2007, 45, 1039–1046.

36. Komar, N.; Clark, G.G. West Nile Virus Activity in Latin America and the Caribbean. Rev. Panam. Salud Publica 2006, 19, 112–117.

37. Monge Maillo, B.; López-Vélez, R.; Norman, F.; de Ory, F.; Sanchez-Seco, M.P.; Giovanni Fedele, C. Importation of West Nile Virus Infection from Nicaragua to Spain. Emerg. Infect. Dis. 2008, 14, 1171–1173.

38. Morales-Betoulle, M.E.; Komar, N.; Panella, N.A.; Álvarez, D.; López, M.R.; Betoulle, J.; Sosa, S.M.; Muller, M.L.; Kilpatrick, A.M.; Lanciotti, R.S.; et al. West Nile Virus Ecology in a Tropical Ecosystem in Guatemala. Am. J. Trop. Med. Hyg. 2013, 88, 116–126.

39. Morales-Betoulle, M.E.; Morales, H.; Blitvich, B.J.; Powers, A.M.; Davis, E.A.; Klein, R.; Cordón-Rosales, C. West Nile Virus in Horses, Guatemala. Emerg. Infect. Dis. 2006, 12, 1038–1039.

40. Kading, R.C.; Reiche, A.S.; Morales-Betoulle, M.E.; Komar, N. Host Selection of Potential West Nile Virus Vectors in Puerto Barrios, Guatemala, 2007. Am. J. Trop. Med. Hyg. 2013, 88, 108–115.

41. Carrera, J.-P.; Bagamian, K.H.; da Rosa, A.P.T.; Wang, E.; Beltran, D.; Gundaker, N.D.; Armien, B.; Arroyo, G.; Sosa, N.; Pascale, J.M.; et al. Human and Equine Infection with Alphaviruses and Flaviviruses in Panamá during 2010: A Cross-Sectional Study of Household Contacts during an Encephalitis Outbreak. Am. J. Trop. Med. Hyg. 2018, 98, 1798–1804.

42. Hobson-Peters, J.; Arévalo, C.; Cheah, W.Y.; Blitvich, B.J.; Tan, C.S.; Sandis, A.; Araya, L.N.; Hernández, J.L.; Toye, P.; Hall, R.A. Detection of Antibodies to West Nile Virus in Horses, Costa Rica, 2004. Vector Borne Zoonotic Dis. 2011, 11, 1081–1084.

43. Jiménez, C.; Romero, M.; Piche, M.; Baldi, M.; Alfaro, A.; Chaves, A.; Morales, J.; León, B.; Hutter, S.; Corrales-Aguilar, E. Arboviral Encephalitis in Costa Rican Horses: 2009–2016. Int. J. Infect. Dis. 2016, 53, 153.

44. Chaves, A.; Piche-Ovares, M.; Ibarra-Cerdeña, C.N.; Corrales-Aguilar, E.; Suzán, G.; Moreira-Soto, A.; Gutiérrez-Espeleta, G.A. Serosurvey of Nonhuman Primates in Costa Rica at the Human–Wildlife Interface Reveals High Exposure to Flaviviruses. Insects 2021, 12, 554.

45. Medlin, S.; Deardorff, E.R.; Hanley, C.S.; Vergneau-Grosset, C.; Siudak-Campfield, A.; Dallwig, R.; da Rosa, A.T.; Tesh, R.B.; Martin, M.P.; Weaver, S.C.; et al. Serosurvey of Selected Arboviral Pathogens in Free-Ranging, Two-Toed Sloths (Choloepus hoffmanni) and Three-Toed Sloths (Bradypus variegatus) in Costa Rica, 2005–2007. J. Wildl. Dis. 2016, 52, 883–892.

46. Dolz, G.; Chaves, A.; Gutiérrez-Espeleta, G.A.; Ortiz-Malavasi, E.; Bernal-Valle, S.; Herrero, M.V. Detection of Antibodies against Flavivirus over Time in Wild Non-Human Primates from the Lowlands of Costa Rica. PLoS ONE 2019, 14, e0219271.

47. Piche-Ovares, M.; Romero-Vega, M.; Vargas-Gonzalez, D.; Barrantes-Murillo, D.; Soto-Garita, C.; Francisco-Llamas, J.; Alfaro-Alarcon, A.; Jimenez, C.; Corrales-Aguilar, E. Serosurvey in Two Rural Areas Evidences Recent and Previously Undetected WNV and SLEV circulation in Costa Rica. medRxiv 2021.

48. Mores, C.N.; Turell, M.J.; Dohm, D.J.; Blow, J.A.; Carranza, M.T.; Quintana, M. Experimental Transmission of West Nile Virus by Culex nigripalpus from Honduras. Vector Borne Zoonotic Dis. 2007, 7, 279–284.

49. Kent, R.J.; Crabtree, M.B.; Miller, B.R. Transmission of West Nile Virus by Culex quinquefasciatus Say Infected with Culex Flavivirus Izabal. PLoS Negl. Trop. Dis. 2010, 4, e671.

50. Barzon, L.; Pacenti, M.; Ulbert, S.; Palù, G. Latest Developments and Challenges in the Diagnosis of Human West Nile Virus Infection. Expert Rev. Anti-Infect. Ther. 2015, 13, 327–342.

51. Lustig, Y.; Sofer, D.; Bucris, E.D.; Mendelson, E. Surveillance and Diagnosis of West Nile Virus in the Face of Flavivirus Cross-Reactivity. Front. Microbiol. 2018, 9, 1–10.

52. Moreira, J.; Barros, J.; Lapouble, O.; Lacerda, M.V.G.; Felger, I.; Brasil, P.; Dittrich, S.; Siqueira, A.M. When Fever Is Not Malaria in Latin America: A Systematic Review. BMC Med. 2020, 18, 1294.

53. Rappole, J.H.; Derrickson, S.R.; Hubálek, Z. Migratory Birds and Spread of West Nile Virus in the Western Hemisphere. Emerg. Infect. Dis. 2000, 6, 319–328.

54. Maharaj, P.D.; Bosco-Lauth, A.M.; Langevin, S.A.; Anishchenko, M.; Bowen, R.A.; Reisen, W.K.; Brault, A.C. West Nile and St. Louis Encephalitis Viral Genetic Determinants of Avian Host Competence. PLoS Negl. Trop. Dis. 2018, 12, e0006302.

55. Tesh, R.B.; Travassos da Rosa, A.P.; Guzman, H.; Araujo, T.P.; Xiao, S.Y. Immunization with Heterologous Flaviviruses Protective against Fatal West Nile Encephalitis. Emerg. Infect. Dis. 2002, 8, 245–251.

56. Diaz, L.A.; Goñi, S.E.; Iserte, J.A.; Quaglia, A.I.; Singh, A.; Logue, C.H.; Powers, A.M.; Contigiani, M.S. Exploring Genomic, Geographic and Virulence Interactions among Epidemic and Non-Epidemic St. Louis Encephalitis Virus (Flavivirus) Strains. PLoS ONE 2015, 10, e0136316.

57. Galindo, P.; Peralta, P.H.; Mackenzie, R.B.; Beye, H. Saint Louis Encephalitis in Panama: A Review and Progress Report. Am. J. Trop. Med. Hyg. 1964, 13, 455.

58. de Rodaniche, E.; Galindo, P. St. Louis Encephalitis in Panama. III. Investigation of Local Mammals and Birds as Possible Reservoir Hosts. Am. J. Trop. Med. Hyg. 1961, 10, 390–392.

59. Galindo, P.; Adames, A.; Peralta, P.; Johnson, C.; Read, R. Impacto de La Hidroeléctrica de Bayano En La Transmisión de Arbovirus. Rev. Méd. Panamá 1983, 8, 89–134.

60. Dutary, B.E.; Peralta, P.H.; Petersen, J.L. Estudios Biológicos Del Virus de La Encefalitis de San Luis En Mage, Bayano. Rev. Méd. Panamá 1984, 200–211.

61. Cupp, E.W.; Scherer, W.F.; Lok, J.B.; Brenner, R.J.; Dziem, G.M.; Ordonez, J.V. Entomological Studies at an Enzootic Venezuelan Equine Encephalitis Virus Focus in Guatemala, 1977–1980. Am. J. Trop. Med. Hyg. 1986, 35, 851–859.

62. Weaver, S.C.; Ferro, C.; Barrera, R.; Boshell, J.; Navarro, J.C. Venezuelan equine encephalitis. Ann. Rev. Entomol. 2004, 49, 141–174.

63. Guzmán-Terán, C.; Calderón-Rangel, A.; Rodriguez-Morales, A.; Mattar, S. Venezuelan Equine Encephalitis Virus: The Problem Is Not Over for Tropical America. Ann. Clin. Microbiol. Antimicrob. 2020, 19, 19.

64. Oberste, M.S.; Schmura, S.M.; Weaver, S.C.; Smith, J.F. Geographic Distribution of Venezuelan Equine Encephalitis Virus Subtype IE Genotypes in Central America and Mexico. Am. J. Trop. Med. Hyg. 1999, 60, 630–634.

65. Franck, P.T.; Johnson, K.M. An Outbreak of Venezuelan Equine Encephalomyelitis in Central America. Evidence for Exogenous Source of a Virulent Virus Subtype. Am. J. Epidemiol. 1971, 94, 487–495.

66. de la Hoz Restrepo, F. Encefalitis Equina Venezolana. Rev. MVZ Córdoba 2000, 5, 18–22.

67. Sudia, W.D.; Lord, R.D.; Newhouse, V.F.; Miller, D.L.; Kissling, R.E. Vector-Host Studies of an Epizootic of Venezuelan Equine Encephalomyelitis in Guatemala, 1969. Am. J. Epidemiol. 1971, 93, 137–143.

68. Martin, D.; Eddy, G.A.; Sudia, W.D.; Reeves, W.C.; Newhouse, V.F.; Johnson, K. An Epidemiologic Study of Venezuelan Equine Encephalomyelitis in Costa Rica, 1970. Am. J. Epidemiol. 1972, 95, 565–578.

69. Lord, R.D. Encefalitis Equina Venezolana Su Historia y Distribución Geográfica. Bol. Oficina Sanit. Panam. 1973, 1973, 530–541.

70. Weaver, S.C.; Kinney, R.M.; Kang, W.; Pfeffer, M.; Marriott, K. Genetic Evidence for the Origins of Venezuelan Equine Encephalitis Virus Subtype IAB Outbreaks. Am. J. Trop. Med. Hyg. 1999, 60, 630–634.

71. Aguilar, P.V.; Estrada-Franco, J.G.; Navarro-Lopez, R.; Ferro, C.; Haddow, A.D.; Weaver, S.C. Endemic Venezuelan Equine Encephalitis in the Americas: Hidden under the Dengue Umbrella. Future Virol. 2011, 6, 721–740.

72. Quiroz, E.; Aguilar, P.V.; Cisneros, J.; Tesh, R.B.; Weaver, S.C. Venezuelan Equine Encephalitis in Panama: Fatal Endemic Disease and Genetic Diversity of Etiologic Viral Strains. PLoS Negl. Trop. Dis. 2009, 3, e472.

73. Vittor, A.Y.; Armien, B.; Gonzalez, P.; Carrera, J.P.; Dominguez, C.; Valderrama, A.; Glass, G.E.; Beltran, D.; Cisneros, J.; Wang, E.; et al. Epidemiology of Emergent Madariaga Encephalitis in a Region with Endemic Venezuelan Equine Encephalitis: Initial Host Studies and Human Cross-Sectional Study in Darien, Panama. PLoS Negl. Trop. Dis. 2016, 10, e0004554.

74. Turell, M.J.; Beaman, J.R. Experimental Transmission of Venezuelan Equine Encephalomyelitis Virus by a Strain of Aedes albopictus (Diptera: Culicidae) from New Orleans, Louisiana. J. Med. Entomol. 1992, 29, 802–805.

75. Ortiz, D.I.; Kang, W.; Weaver, S.C. Susceptibility of Ae. aegypti (Diptera: Culicidae) to Infection with Epidemic (Subtype IC) and Enzootic (Subtypes ID, IIIC, IIID) Venezuelan Equine Encephalitis Complex Alphaviruses. J. Med. Entomol. 2008, 45, 1117–1125.

76. Johnson, K.M.; Shelokov, A.; Peralta, P.H.; Dammin, G.J.; Young, N.A. Recovery of Venezuelan Equine Encephalomyelitis Virus in Panamá: A Fatal Case in Man. Am. J. Trop. Med. Hyg. 1968, 17, 432–440.

77. Galindo, P.; Srihongse, S.; de Rodaniche, E.; Grayson, M.A. An Ecological Survey for Arboviruses in Almirante, Panama, 1959–1962. Am. J. Trop. Med. Hyg. 1966, 15, 385–400.

78. Grayson, M.A.; Galindo, P. Epidemiologic Studies of Venezuelan Equine Encephalitis Virus in Almirante, Panama. Am. J. Epidemiol. 1968, 88, 80–86.

79. Grayson, M.A.; Galindo, P. Ecology of Venezuelan Equine Encephalitis Virus in Panama. J. Am. Vet. Med. Assoc. 1969, 155, 2141–2145.

80. Seymour, C.; Dickerman, R.W.; Martin, M.S. Venezuelan Encephalitis Virus Infection in Neotropical Bats: I. Natural Infection in a Guatemalan Enzootic Focus. Am. J. Trop. Med. Hyg. 1978, 27, 290–296.

81. Scherer, W.F.; Dickerman, R.W.; Cupp, E.W.; Ordonez, J.V. Ecologic Observations of Venezuelan Encephalitis Virus in Vertebrates and Isolations of Nepuyo and Patois Viruses from Sentinel Hamsters at Pacific and Atlantic Habitats in Guatemala, 1968–1980. Am. J. Trop. Med. Hyg. 1985, 34, 790–798.

82. León, B.; Jiménez, C.; González, R.; Ramirez-Carvajal, L. First Complete Coding Sequence of a Venezuelan Equine Encephalitis Virus Strain Isolated from an Equine Encephalitis Case in Costa Rica. Microbiol. Resour. Announc. 2019, 8.

83. León, B.; Käsbohrer, A.; Hutter, S.E.; Baldi, M.; Firth, C.L.; Romero-Zúñiga, J.J.; Jiménez, C. National Seroprevalence and Risk Factors for Eastern Equine Encephalitis and Venezuelan Equine Encephalitis in Costa Rica. J. Equine Vet. Sci. 2020, 92, 103140.

84. Torres, R.; Samudio, R.; Carrera, J.-P.; Young, J.; Márquez, R.; Hurtado, L.; Weaver, S.; Chaves, L.F.; Tesh, R.; Cáceres, L. Enzootic Mosquito Vector Species at Equine Encephalitis Transmission Foci in the República de Panamá. PLoS ONE 2017, 12, e0185491.

85. WHO (World Health Organization). World Malaria Report: 20 Years of Global Progress and Challenges; WHO: Geneva, Switzerland, 2020.

86. Sinka, M.E.; Bangs, M.J.; Manguin, S.; Rubio-Palis, Y.; Chareonviriyaphap, T.; Coetzee, M.; Mbogo, C.M.; Hemingway, J.; Patil, A.P.; Temperley, W.H.; et al. A Global Map of Dominant Malaria Vectors. Parasites Vectors 2012, 5, 1–11.

87. Loaiza, J.R.; Bermingham, E.; Scott, M.E.; Rovira, J.R.; Conn, J.E. Species Composition and Distribution of Adult Anopheles (Diptera: Culicidae) in Panama. J. Med. Entomol. 2008, 45, 841–851.

88. Hiwat, H.; Bretas, G. Ecology of Anopheles darlingi Root with Respect to Vector Importance: A Review. Parasites Vectors 2011, 4, 1–13.

89. Warren, M.; Mason, J.; Hobbs, J. Natural Infections of Anopheles albimanus with Plasmodium in a Small Malaria Focus. Am. J. Trop. Med. Hyg. 1975, 24, 545–546.

90. Roberts, D.R.; Paris, J.F.; Manguin, S.; Harbach, R.E.; Woodruff, R.; Rejmankova, E.; Polanco, J.; Wullschleger, B.; Legters, L.J. Predictions of Malaria Vector Distribution in Belize Based on Multispectral Satellite Data. Am. J. Trop. Med. Hyg. 1996, 54, 304–308.

91. Escobar, D.; Ascencio, K.; Ortiz, A.; Palma, A.; Fontecha, G. Distribution and Phylogenetic Diversity of Anopheles Species in Malaria Endemic Areas of Honduras in an Elimination Setting. Parasites Vectors 2020, 13, 1–12.

92. Malaria Atlas Project Trends in Global Malaria Burden. Available online: https://www.malariaatlas.org/ (accessed on 30 September 2021).

93. PAHO (Pan American Health Organization). El Salvador Certified as Malaria-Free by WHO. Available online: https://www.paho.org/en/news/25-2-2021-salvador-certified-malaria-free-who (accessed on 4 October 2021).

94. WHO (World Health Organization). Zeroing in on Malaria Elimination: Final Report of the E-2020 Initiative; WHO: Geneva, Switzerland, 2021.

95. WHO (World Health Organization). Global Technical Strategy for Malaria 2016–2030; WHO: Geneva, Switzerland, 2016.

96. WHO (World Health Organization). A Framework for Malaria Elimination Global Malaria Programme; WHO: Geneva, Switzerland, 2017.

97. PAHO (Pan American Health Organization). Epidemiological Update: Malaria in the Americas in the Context of COVID-19 Pandemic. 2020. Available online: https://www.paho.org/en/documents/epidemiological-update-malaria-10-june-2020 (accessed on 4 October 2021).

98. Ferreira, M.U.; Castro, M.C. Malaria Situation in Latin America and the Caribbean: Residual and Resurgent Transmission and Challenges for Control and Elimination. Methods Mol. Biol. 2019, 2013, 57–70.

99. Herrera, S.; Quiñones, M.L.; Quintero, J.P.; Corredor, V.; Fuller, D.O.; Mateus, J.C.; Calzada, J.E.; Gutierrez, J.B.; Llanos, A.; Soto, E.; et al. Prospects for Malaria Elimination in Non-Amazonian Regions of Latin America. Acta Trop. 2012, 121, 315–323.

100. Caminade, C.; Kovats, S.; Rocklov, J.; Tompkins, A.M.; Morse, A.P.; Colón-González, F.J.; Stenlund, H.; Martens, P.; Lloyd, S.J. Impact of Climate Change on Global Malaria Distribution. Proc. Natl. Acad. Sci. USA 2014, 111, 3286–3291.

101. Payne, R.C.; Scott, J.M. Anaplasmosis and Babesiosis in El Salvador. Trop. Anim. Health Prod. 1982, 14, 75–80.

102. Guglielmone, A.A. Epidemiology of Babesiosis and Anaplasmosis in South and Central America. Vet. Parasitol. 1995, 57, 109–119.

103. van Andel, J.M.; Dwinger, R.H.; Alvarez, J.A. Etude Du Taux d'infection Du Bétail et Des Tiques Associées Par Anaplasma et Babesia Sur La Côte Sud Du Guatemala. Rev. Élev. Méd. Vét. Pays Trop. 1997, 50, 284–292.

104. Teglas, M.; Matern, E.; Lein, S.; Foley, P.; Mahan, S.M.; Foley, J. Ticks and Tick-Borne Disease in Guatemalan Cattle and Horses. Vet. Parasitol. 2005, 131, 119–127.

105. Heerdink, G.; Petit, P.L.C.; Hofwegen, H.; van Genderen, P.J.J. Een Patiënt Met Koorts Na Een Bezoek Aan de Tropen: "Tick-Borne Relapsing Fever" Ontdekt in Een Dikkedruppelpreparaat . Ned. Tijdschr. Geneeskd. 2006, 150, 2386–2389.

106. Eremeeva, M.E.; Karpathy, S.E.; Levin, M.L.; Caballero, C.M.; Bermudez, S.; Dasch, G.A.; Motta, J.A. Spotted Fever Rickettsiae, Ehrlichia and Anaplasma, in Ticks from Peridomestic Environments in Panama. Clin. Microbiol. Infect. 2009, 15, 12–14.

107. Bermúdez, S.E.; Eremeeva, M.E.; Karpathy, S.E.; Samudio, F.; Zambrano, M.L.; Zaldivar, Y.; Motta, J.A.; Dasch, G.A. Detection and Identification of Rickettsial Agents in Ticks from Domestic Mammals in Eastern Panama. J. Med. Entomol. 2009, 46, 856–861.

108. Shebish, E.; Vemulapalli, R.; Oseto, C. Prevalence and Molecular Detection of Anaplasma marginale, Babesia bovis and Babesia bigemina in Cattle from Puntarenas Province, Costa Rica. Vet. Parasitol. 2012, 188, 164–167.

109. Dolz, G.; Abrego, L.; Romero, L.E.; Campos-Calderon, L.; Bouza-Mora, L.; Jiménez-Rocha, A.E. Ehrlichiosis y Anaplasmosis en Costa Rica. Acta Méd. Costarric. 2013, 55, 34–40.

110. Mehrkens, L.R.; Shender, L.A.; Yabsley, M.J.; Shock, B.C.; Chinchilla, F.A.; Suarez, J.; Gilardi, K.V.K. White-Nosed Coatis (Nasua Narica) Are a Potential Reservoir of Trypanosoma cruzi and

Other Potentially Zoonotic Pathogens in Monteverde, Costa Rica. J. Wildl. Dis. 2013, 49, 1014–1018.

111. Rojas, A.; Rojas, D.; Montenegro, V.; Gutiérrez, R.; Yasur-Landau, D.; Baneth, G. Vector-Borne Pathogens in Dogs from Costa Rica: First Molecular Description of Babesia vogeli and Hepatozoon canis Infections with a High Prevalence of Monocytic Ehrlichiosis and the Manifestations of Co-Infection. Vet. Parasitol. 2014, 199, 121–128.

112. Santamaria, A.; Calzada, J.E.; Saldaña, A.; Yabsley, M.J.; Gottdenker, N.L. Molecular Diagnosis and Species Identification of Ehrlichia and Anaplasma Infections in Dogs from Panama, Central America. Vector Borne Zoonotic Dis. 2014, 14, 368–370.

113. Wei, L.; Kelly, P.; Ackerson, K.; El-Mahallawy, H.S.; Kaltenboeck, B.; Wang, C. Molecular Detection of Dirofilaria immitis, Hepatozoon canis, Babesia spp., Anaplasma platys and Ehrlichia canis in Dogs on Costa Rica. Acta Parasitol. 2014, 60, 21–25.

114. Wei, L.; Kelly, P.; Ackerson, K.; Zhang, J.; El-Mahallawy, H.S.; Kaltenboeck, B.; Wang, C. First Report of Babesia gibsoni in Central America and Survey for Vector-Borne Infections in Dogs from Nicaragua. Parasites Vectors 2014, 7, 1–6.

115. Rojas, N.; Castillo, D.; Marin, P. Molecular Detection of Ehrlichia chaffeensis in Humans, Costa Rica. Emerg. Infect. Dis. 2015, 21, 532–534.

116. O'Nion, V.L.; Montilla, H.J.; Qurollo, B.A.; Maggi, R.G.; Hegarty, B.C.; Tornquist, S.J.; Breitschwerdt, E.B. Potentially Novel Ehrlichia Species in Horses, Nicaragua. Emerg. Infect. Dis. 2015, 21, 335–338.

117. Posada-Guzmán, M.F.; Dolz, G.; Romero-Zúñiga, J.J.; Jiménez-Rocha, A.E. Detection of Babesia caballi and Theileria equi in Blood from Equines from Four Indigenous Communities in Costa Rica. Vet. Med. Int. 2015, 2015.

118. Lopez, J.E.; Krishnavahjala, A.; Garcia, M.N.; Bermudez, S. Tick-Borne Relapsing Fever Spirochetes in the Americas. Vet. Sci. 2016, 3, 16.

119. Campos-Calderón, L.; Ábrego-Sánchez, L.; Solórzano-Morales, A.; Alberti, A.; Tore, G.; Zobba, R.; Jiménez-Rocha, A.E.; Dolz, G. Molecular Detection and Identification of Rickettsiales Pathogens in Dog Ticks from Costa Rica. Ticks Tick-Borne Dis. 2016, 7, 1198–1202.

120. Chikeka, I.; Matute, A.J.; Dumler, J.S.; Woods, C.W.; Mayorga, O.; Reller, M.E. Use of Peptide-Based Enzyme-Linked Immunosorbent Assay Followed by Immunofluorescence Assay to Document Ehrlichia chaffeensis as a Cause of Febrile Illness in Nicaragua. J. Clin. Microbiol. 2016, 54, 1581–1585.

121. Bouza-Mora, L.; Dolz, G.; Solórzano-Morales, A.; Romero-Zuñiga, J.J.; Salazar-Sánchez, L.; Labruna, M.B.; Aguiar, D.M. Novel Genotype of Ehrlichia canis Detected in Samples of Human Blood Bank Donors in Costa Rica. Ticks Tick-Borne Dis. 2017, 8, 36–40.

122. Bermúdez, C.S.; Troyo, A. A Review of the Genus Rickettsia in Central America. Res. Rep. Trop. Med. 2018, 9, 103–112.

123. Rimbaud Giambruno, E.; Mayorga-Escobar, M.I.; González, D.; Sequeira Valle, E.J.; Torres, H.; Montoya, C.; Ramírez, S. Estudio Epidemiológico de La Prevalencia y Evolución de Hemoparásitos En Burros Del Norte de Nicaragua. Calera 2018, 18, 26–28.

124. Vogel, H.; Foley, J.; Fiorello, C.V. Rickettsia africae and Novel Rickettsial Strain in Amblyomma spp. Ticks, Nicaragua, 2013. Emerg. Infect. Dis. 2018, 24, 385–387.

125. Springer, A.; Montenegro, V.M.; Schicht, S.; Pantchev, N.; Strube, C. Seroprevalence and Current Infections of Canine Vector-Borne Diseases in Nicaragua. Parasites Vectors 2018, 11, 1–9.

126. Springer, A.; Montenegro, V.M.; Schicht, S.; Wölfel, S.; Schaper, S.R.; Chitimia-Dobler, L.; Siebert, S.; Strube, C. Detection of Rickettsia monacensis and Rickettsia amblyommatis in Ticks Collected from Dogs in Costa Rica and Nicaragua. Ticks Tick-Borne Dis. 2018, 9, 1565–1572.

127. Springer, A.; Montenegro, V.M.; Schicht, S.; Globokar Vrohvec, M.; Pantchev, N.; Balzer, J.; Strube, C. Seroprevalence and Current Infections of Canine Vector-Borne Diseases in Costa Rica. Front. Vet. Sci. 2019, 6, 164.

128. Daza, C.; Osorio, J.; Santamaria, A.; Suárez, J.; Hurtado, A.; Bermúdez Castillero, S.E. Caracterización Del Primer Caso de Infección Humana Por Ehrlichia canis En Panamá. Rev. Méd. Panamá 2018, 38, 63–68.

129. Tyrrell, J.D.; Qurollo, B.A.; Tornquist, S.J.; Schlaich, K.G.; Kelsey, J.; Chandrashekar, R.; Breitschwerdt, E.B. Molecular Identification of Vector-Borne Organisms in Ehrlichia Seropositive Nicaraguan Horses and First Report of Rickettsia felis Infection in the Horse. Acta Trop. 2019, 200, 105170.

130. Panti-May, J.A.; Rodríguez-Vivas, R.I. Canine Babesiosis: A Literature Review of Prevalence, Distribution, and Diagnosis in Latin America and the Caribbean. Vet. Parasitol. Reg. Studies Rep. 2020, 21, 100417.

131. Bader, J.; Ramos, R.A.N.; Otranto, D.; Dantas-Torres, F. Vector-Borne Pathogens in Dogs from Guatemala, Central America. Vet. Parasitol. Reg. Stud. Rep. 2020, 22, 100468.

132. Romero, L.; Costa, F.B.; Labruna, M.B. Ticks and Tick-Borne Rickettsia in El Salvador. Exp. Appl. Acarol. 2021, 83, 545–554.

133. Bermúdez, C.S.E.; Félix, M.L.; Domínguez, A.L.; Kadoch, N.; Muñoz-Leal, S.; Venzal, J.M. Molecular Screening for Tick-Borne Bacteria and Hematozoa in Ixodes cf. boliviensis and Ixodes tapirus (Ixodida: Ixodidae) from Western Highlands of Panama. Curr. Res. Parasitol. Vector-Borne Dis. 2021, 1, 100034.

134. Bermúdez, S.; Martínez-Mandiche, J.; Domínguez, L.; Gonzalez, C.; Chavarria, O.; Moreno, A.; Góndola, J.; Correa, N.; Rodríguez, I.; Castillo, B.; et al. Diversity of Rickettsia in Ticks Collected from Wild Animals in Panama. Ticks Tick-Borne Diseases 2021, 12, 101723.

135. Su, H. Enfermedad de Lyme. Presentación de Un Caso. Rev. Med. Hond. 2004, 72, 193–197.

136. Rojas-Solano, J.R.; Villalobos-Vindas, J. Caso Clínico Ehrliquiosis Granulocitotrópica Humana. Acta Méd. Costarric. 2007, 49, 121–123.

137. de Mezerville, V.H.; Cuadra, J.I.P. Choque Séptico Por Ehrliquiosis. Acta Méd. Costarric. 2007, 49, 118–120.

138. Boza-Cordero, R. Enfermedad de Lyme (Borreliosis de Lyme) En Costa Rica. Acta Med. Costarric. 2011, 53, 34–36.

139. Brenes Valverde, D.; Brenes Martínez, S.; Quirós Rojas, I. Ehrliquiosis: Reporte de 2 Casos. Rev. Méd. Costa Rica Centroam. 2011, 598, 315–318.

140. Villalobos-Zúñiga, M.-A.; Somogyi, T. Acute Lyme Disease in Costa Rica. Description of the First Autochthonous Case. Acta Méd. Costarric. 2012, 54, 55–58.

141. Hun, L. Rickettsiosis En Costa Rica. Acta Med. Costarric. 2013, 55, 25–28.

142. Peacock, M.G.; Ormsbee, R.A.; Johnson, K.M. Rickettsioses of Central America. Am. J. Trop. Med. Hyg. 1971, 20, 941–949.

143. Eremeeva, M.E.; Dasch, G.A. Challenges Posed by Tick-Borne Rickettsiae: Eco-Epidemiology and Public Health Implications. Front. Public Health 2015, 3, 55.

144. de Rodaniche, E.C.; Rodaniche, A. Spotted Fever in Panama; Isolation of the Etiologic Agent from a Fatal Case. Am. J. Trop. Med. Hyg. 1950, s1-30, 511–517.

145. Labruna, M.B. Ecology of Rickettsia in South America. Ann. N. Y. Acad. Sci. 2009, 1166, 156–166.

146. Nava, S.; Beati, L.; Labruna, M.B.; Cáceres, A.G.; Mangold, A.J.; Guglielmone, A.A. Reassessment of the Taxonomic Status of Amblyomma cajennense, (Fabricius, 1787) with the Description of Three New Species, Amblyomma tonelliae n. sp., Amblyomma interandium n. sp. and Amblyomma patinoi n. sp., and Reinstatement of Amblyomma mixtum, and Amblyomma sculptum (Ixodida: Ixodidae). Ticks Tick-Borne Dis. 2014, 5, 252–276.

147. Martínez-Caballero, A.; Moreno, B.; González, C.; Martínez, G.; Adames, M.; Pachar, J.V.; Varela-Petrucelli, J.B.; Martínez-Mandiche, J.; Suárez, J.A.; Domínguez, L.; et al. Descriptions of Two New Cases of Rocky Mountain Spotted Fever in Panama, and Coincident Infection with Rickettsia rickettsii in Rhipicephalus sanguineus s.l. in an Urban Locality of Panama City, Panama. Epidemiol. Infect. 2018, 146, 875–878.

148. Eremeeva, M.E.; Berganza, E.; Suarez, G.; Gobern, L.; Dueger, E.; Castillo, L.; Reyes, L.; Wikswo, M.E.; Abramowicz, K.F.; Dasch, G.A.; et al. Investigation of an Outbreak of Rickettsial Febrile Illness in Guatemala, 2007. Int. J. Infect. Dis. 2013, 17, e304–e311.

149. Chen, L.H.; Wilson, M.E. Tick-Borne Rickettsiosis in Traveler Returning from Honduras. Emerg. Infect. Dis. 2009, 15, 1321–1323.

150. Reller, M.E.; Chikeka, I.; Miles, J.J.; Dumler, J.S.; Woods, C.W.; Mayorga, O.; Matute, A.J. First Identification and Description of Rickettsioses and Q Fever as Causes of Acute Febrile Illness in Nicaragua. PLoS Negl. Trop. Dis. 2016, 10, e0005185.

151. Zavala-Castro, J.E.; Zavala-Velázquez, J.E.; Peniche-Lara, G.F.; Uicab, J.E.S. Human Rickettsialpox, Southeastern Mexico. Emerg. Infect. Dis. 2009, 15, 1665–1667.

152. Zavala-Castro, J.E.; Zavala-Velázquez, J.E.; García, M.D.R.; León, J.J.A.; Dzul-Rosado, K.R. A Dog Naturally Infected with Rickettsia akari in Yucatan, México. Vector Borne Zoonotic Dis. 2009, 9, 345–347.

153. Rar, V.; Golovljova, I. Anaplasma, Ehrlichia, and "Candidatus Neoehrlichia" Bacteria: Pathogenicity, Biodiversity, and Molecular Genetic Characteristics, a Review. Infect. Genet. Evol. 2011, 11, 1842–1861.

154. Pruneau, L.; Moumène, A.; Meyer, D.F.; Marcelino, I.; Lefrançois, T.; Vachiéry, N. Understanding Anaplasmataceae Pathogenesis Using "Omics" Approaches. Front. Cell. Infect. Microbiol. 2014, 4, 86.

155. Conrad, J.; Norman, J.; Rodriguez, A.; Dennis, P.M.; Arguedas, R.; Jimenez, C.; Hope, J.G.; Yabsley, M.J.; Hernandez, S.M. Demographic and Pathogens of Domestic, Free-Roaming Pets and the Implications for Wild Carnivores and Human Health in the San Luis Region of Costa Rica. Vet. Sci. 2021, 8, 65.

156. Little, S.E. Ehrlichiosis. In Arthropod Borne Diseases; Springer: Cham, Switzerland, 2017; pp. 205–213.

157. Rodriguez-Morales, A.J.; Bonilla-Aldana, D.K.; Idarraga-Bedoya, S.E.; Garcia-Bustos, J.J.; Cardona-Ospina, J.A.; Faccini-Martínez, Á.A. Epidemiology of Zoonotic Tick-Borne Diseases in Latin America: Are We Just Seeing the Tip of the Iceberg? F1000Research 2019, 7, 1988.

158. Rochlin, I.; Toledo, A. Emerging Tick-Borne Pathogens of Public Health Importance: A Mini-Review. J. Med. Microbiol. 2020, 69, 781–791.

159. Barrantes-González, A.V.; Jiménez-Rocha, A.E.; Romero-Zuñiga, J.J.; Dolz, G. Serology, Molecular Detection and Risk Factors of Ehrlichia canis Infection in Dogs in Costa Rica. Ticks Tick-Borne Dis. 2016, 7, 1245–1251.

160. Ramos-Castañeda, J.; dos Santos, F.B.; Martínez-Vega, R.; de Araujo, J.M.G.; Joint, G.; Sarti, E. Dengue in Latin America: Systematic Review of Molecular Epidemiological Trends. PLoS Negl. Trop. Dis. 2017, 11, e0005224.

161. Carrera, J.-P.; Forrester, N.; Wang, E.; Vittor, A.Y.; Haddow, A.D.; López-Vergès, S.; Abadía, I.; Castaño, E.; Sosa, N.; Báez, C.; et al. Eastern Equine Encephalitis in Latin America. N. Engl. J. Med. 2013, 369, 732–744.

162. Mota, M.T.d.O.; Ribeiro, M.R.; Vedovello, D.; Nogueira, M.L. Mayaro Virus: A Neglected Arbovirus of the Americas. Future Virol. 2015, 10, 1109–1122.

163. Vieira, C.J.D.S.P.; Thies, S.F.; da Silva, D.J.F.; Kubiszeski, J.R.; Barreto, E.S.; Monteiro, H.A.O.; Mondini, A.; São Bernardo, C.S.; Bronzoni, R.V.M. Ecological Aspects of Potential Arbovirus Vectors (Diptera: Culicidae) in an Urban Landscape of Southern Amazon, Brazil. Acta Trop. 2020, 202, 105276.

164. Wilke, A.; Benelli, G.; Beier, J.C. Anthropogenic Changes and Associated Impacts on Vector-borne Diseases. Trends Parasitol. 2021, 37, 1027–1030.

165. Weaver, S.C. Urbanization and Geographic Expansion of Zoonotic Arboviral Diseases: Mechanisms and Potential Strategies for Prevention. Trends Microbiol. 2013, 21, 360–363.

166. Ellwanger, J.H.; Kulmann-Leal, B.; Kaminski, V.L.; Valverde-Villegas, J.M.; da Veiga, A.B.G.; Spilki, F.R.; Fearnside, P.M.; Caesar, L.; Giatti, L.L.; Wallau, G.L.; et al. Beyond Diversity Loss and Climate Change: Impacts of Amazon Deforestation on Infectious Diseases and Public Health. An. Acad. Bras. Ciênc. 2020, 92, 20191375.

167. Lwande, O.W.; Obanda, V.; Lindström, A.; Ahlm, C.; Evander, M.; Näslund, J.; Bucht, G. Globe-Trotting Aedes aegypti and Aedes albopictus: Risk Factors for Arbovirus Pandemics. Vector Borne Zoonotic Dis. 2020, 20, 71–81.

168. da Silva Pessoa Vieira, C.J.; Bernardo, C.S.S.; da Silva, D.J.F.; Kubiszeski, J.R.; Barreto, E.S.; de Oliveira Monteiro, H.A.; Canale, G.R.; Peres, C.A.; Massey, A.L.; Levi, T.; et al. Land-Use Effects on Mosquito Biodiversity and Potential Arbovirus Emergence in the Southern Amazon, Brazil. Transbound. Emerg. Dis. 2021.

169. Wimberly, M.C.; Davis, J.K.; Evans, M.V.; Hess, A.; Newberry, P.M.; Solano-Asamoah, N.; Murdock, C.C. Land Cover Affects Microclimate and Temperature Suitability for Arbovirus Transmission in an Urban Landscape. PLoS Negl. Trop. Dis. 2020, 14, e0008614.

170. Loaiza, J.R.; Dutari, L.C.; Rovira, J.R.; Sanjur, O.I.; Laporta, G.Z.; Pecor, J.; Foley, D.H.; Eastwood, G.; Kramer, L.D.; Radtke, M.; et al. Disturbance and Mosquito Diversity in the Lowland Tropical Rainforest of Central Panama. Sci. Rep. 2017, 7, 1–13.

171. Loaiza, J.R.; Rovira, J.R.; Sanjur, O.I.; Zepeda, J.A.; Pecor, J.E.; Foley, D.H.; Dutari, L.; Radtke, M.; Pongsiri, M.J.; Molinar, O.S.; et al. Forest Disturbance and Vector Transmitted Diseases in the

Lowland Tropical Rainforest of Central Panama. Trop. Med. Int. Health 2019, 24, 849–861.

172. Laporta, G.Z.; de Prado, P.I.K.L.; Kraenkel, R.A.; Coutinho, R.M.; Sallum, M.A.M. Biodiversity Can Help Prevent Malaria Outbreaks in Tropical Forests. PLoS Negl. Trop. Dis. 2013, 7, e2139.

173. Bayles, B.R.; Rusk, A.; Christofferson, R.; Agar, G.; Pineda, M.A.; Chen, B.; Dagy, K.; Kelly, E.; Hummel, T.; Kuwada, K.; et al. Spatiotemporal Dynamics of Vector-Borne Disease Risk across Human Land-Use Gradients: Examining the Role of Agriculture, Indigenous Territories, and Protected Areas in Costa Rica. Lancet Glob. Health 2020, 8, S32.

174. Bates, M. Observations on the Distribution of Diurnal Mosquitoes in a Tropical Forest. Ecology 1944, 25, 159–170.

175. Pessanha, J. Febre Amarela: Uma Visão Do Cenário Atual. Rev. Med. Minas Gerais 2009, 19, 97–102.

176. Ilacqua, R.C.; Medeiros-Sousa, A.R.; Ramos, D.G.; Obara, M.T.; Ceretti-Junior, W.; Mucci, L.F.; Marrelli, M.T.; Laporta, G.Z. Reemergence of Yellow Fever in Brazil: The Role of Distinct Landscape Fragmentation Thresholds. J. Environ. Public Health 2021, 2021, 8230789.

177. Alencar, J.; Lorosa, E.S.; Dégallier, N.; Serra-Freire, N.M.; Pacheco, J.B.; Guimarães, A.É. Feeding Patterns of Haemagogus janthinomys (Diptera: Culicidae) in Different Regions of Brazil. J. Med. Entomol. 2005, 42, 981–985.

178. Alencar, J.; de Mello, C.F.; Morone, F.; Albuquerque, H.G.; Serra-Freire, N.M.; Gleiser, R.M.; Silva, S.O.F.; Guimarães, A.É. Distribution of Haemagogus and Sabethes Species in Relation to Forest Cover and Climatic Factors in the Chapada Dos Guimarães National Park, State of Mato Grosso, Brazil. J. Am. Mosq. Control Assoc. 2018, 34, 85–92.

179. Chaverri, L.G.; Dillenbeck, C.; Lewis, D.; Rivera, C.; Romero, L.M.; Chaves, L.F. Mosquito Species (Diptera: Culicidae) Diversity from Ovitraps in a Mesoamerican Tropical Rainforest. J. Med. Entomol. 2018, 55, 646–653.

180. Galindo, P.; Trapido, H. Forest Canopy Mosquitoes Associated with the Appearance of Sylvan Yellow Fever in Costa Rica, 1951. Am. J. Trop. Med. Hyg. 1955, 4, 543–549.

181. Yanoviak, S.P.; Paredes, J.E.R.; Lounibos, L.P.; Weaver, S.C. Deforestation Alters Phytotelm Habitat Availability and Mosquito Production in the Peruvian Amazon. Ecol. Appl.: Publ. Ecol. Soc. Am. 2006, 16, 1854–1864.

182. Santos, M.; Collado Mariscal, L.; Henríquez, B.; Garzón, J.; González, P.; Carrera, J.P.; Tello, J.; Koo, S.; Pascale, J.M.; Burkett-Cadena, N.; et al. Implementation of Bamboo and Monkey-Pot Traps for the Sampling Cavity-Breeding Mosquitoes in Darién, Panama. Acta Trop. 2020, 205, 105352.

183. Vasconcelos, P.; Travassos da Rosa, A.; Pinheiro, F.; Dégallier, N.; Travassos da Rosa, J. Febare Amarela CONCE I T 0. In Doenças Infecciosas e Parasitárias: Enfoque Amazônico; de Leão, R., Ed.; Cejup/UEPA/Instituto Evandro Chagas: Belém, Brazil, 1997.

184. Pajot, F.; Geoffroy, B.; Chippaux, J. Ecologie d'Haemagogus janthinomys Dyar (DIptera, Culicidae) En Guyane Française. Premières Données. Cahiers-ORSTOM. Entomol. Méd. Parasitol. 1985, 23, 209–216.

185. Monath, T.P.; Vasconcelos, P.F.C. Yellow Fever. J. Clin. Virol. 2015, 64, 160–173.

186. Gomes, A.d.C.; Torres, M.A.N.; de Paula, M.B.; Fernandes, A.; Marassá, A.M.; Consales, C.A.; Fonseca, D.F. Ecologia de Haemagogus e Sabethes (Diptera: Culicidae) Em Áreas Epizoóticas Do Vírus Da Febre Amarela, Rio Grande Do Sul, Brasil. Epidemiol. Serv. Saúde 2010, 19, 101–113.

187. Pinto, C.S.; Confalonieri, U.E.C.; Mascarenhas, B.M. Ecology of Haemagogus sp. and Sabethes sp. (Diptera: Culicidae) in Relation to the Microclimates of the Caxiuanã National Forest, Pará, Brazil. Mem. Inst. Oswaldo Cruz 2009, 104, 592–598.

188. Silva, N.I.O.; Sacchetto, L.; de Rezende, I.M.; Trindade, G.D.S.; Labeaud, A.D.; de Thoisy, B.; Drumond, B.P. Recent Sylvatic Yellow Fever Virus Transmission in Brazil: The News from an Old Disease. Virol. J. 2020, 17, 1–12.

189. Couto-Lima, D.; Madec, Y.; Bersot, M.I.; Campos, S.S.; Motta, M.A.; dos Santos, F.B.; Vazeille, M.; Vasconcelos, P.F.d.C.; Lourenço-de-Oliveira, R.; Failloux, A.-B. Potential Risk of Re-Emergence of Urban Transmission of Yellow Fever Virus in Brazil Facilitated by Competent Aedes Populations. Sci. Rep. 2017, 7, 1–12.

190. Massad, E.; Amaku, M.; Coutinho, F.A.B.; Struchiner, C.J.; Lopez, L.F.; Coelho, G.; Wilder-Smith, A.; Burattini, M.N. The Risk of Urban Yellow Fever Resurgence in Aedes-Infested American Cities. Epidemiol. Infect. 2018, 146, 1219–1225.

191. Da Costa Vasconcelos, P.F. Yellow Fever in Brazil: Thoughts and Hypotheses on the Emergence in Previously Free Areas. Rev. Saúde Pública 2010, 44, 1144–1149.

192. Codeço, C.T.; Luz, P.M.; Struchiner, C.J. Risk Assessment of Yellow Fever Urbanization in Rio de Janeiro, Brazil. Trans. R. Soc. Trop. Med. Hyg. 2004, 98, 702–710.

193. Moreno, E.; Spinola, S.; Tengan, C.; Brasil, R.; Siciliano, M.; Coimbra, T.; Silveira, V.; Rocco, I.; Bisordi, I.; Souza, R.; et al. Yellow Fever Epizootics in Non-Human Primates, São Paulo State, Brazil, 2008–2009. Rev. Inst. Med. Trop. Sao Paulo 2013, 55, 45–50.

194. Hamrick, P.N.; Aldighieri, S.; Machado, G.; Leonel, D.G.; Vilca, L.M.; Uriona, S.; Schneider, M.C. Geographic Patterns and Environmental Factors Associated with Human Yellow Fever Presence in the Americas. PLoS Negl. Trop. Dis. 2017, 11, e0005897.

195. Graham, K.; Bulloch, M.; Lewis, T. Foraging Behaviour of Three Primate Species in a Costa Rican Coastal Lowland Tropical Wet Forest. Biodivers. J. 2013, 4, 327–334.

196. Cosner, C. Models for the Effects of Host Movement in Vector-Borne Disease Systems. Math. Biosci. 2015, 270, 192–197.

197. Clarke, M.R.; Collins, D.A.; Zucker, E.L. Responses to Deforestation in a Group of Mantled Howlers (Alouatta palliata) in Costa Rica. Int. J. Primatol. 2002, 23, 365–381.

198. Schreier, A.L.; Bolt, L.M.; Russell, D.G.; Readyhough, T.S.; Jacobson, Z.S.; Merrigan-Johnson, C.; Coggeshall, E.M.C. Mantled Howler Monkeys (Alouatta palliata) in a Costa Rican Forest Fragment Do Not Modify Activity Budgets or Spatial Cohesion in Response to Anthropogenic Edges. Folia Primatol. 2021, 92, 49–57.

199. Arroyo-Rodríguez, V.; Dias, P.A.D. Effects of Habitat Fragmentation and Disturbance on Howler Monkeys: A Review. Am. J. Primatol. 2010, 72, 1–16.

200. Estrada, A.; Coates-Estrada, R. Tropical Rain Forest Fragmentation and Wild Populations of Primates at Los Tuxtlas, Mexico. Int. J. Primatol. 1996, 17, 759–783.

201. McCann, C.; Williams-Guillén, K.; Koontz, F.; Espinoza, A.A.R.; Sánchez, J.C.M.; Koontz, C. Shade Coffee Plantations as Wildlife Refuge for Mantled Howler Monkeys (Alouatta palliata) in Nicaragua. Primates Fragm. 2003, 321–341.

202. Villatoro-Paz, F.; Sáenz-Méndez, J. La Fragmentación Del Hábitat: Impactos Sobre La Dinámica Huésped-Parásito de La Avifauna En Paisajes Agropecuarios de Esparza, Costa Rica. Zeledonia 2005, 9, 3–9.

203. Bauch, S.C.; Birkenbach, A.M.; Pattanayak, S.K.; Sills, E.O. Public Health Impacts of Ecosystem Change in the Brazilian Amazon. Proc. Natl. Acad. Sci. USA 2015, 112, 7414–7419.

204. Kalbus, A.; de Souza Sampaio, V.; Boenecke, J.; Reintjes, R. Exploring the Influence of Deforestation on Dengue Fever Incidence in the Brazilian Amazonas State. PLoS ONE 2021, 16, e0242685.

205. Young, K.I.; Mundis, S.; Widen, S.G.; Wood, T.G.; Tesh, R.B.; Cardosa, J.; Vasilakis, N.; Perera, D.; Hanley, K.A. Abundance and Distribution of Sylvatic Dengue Virus Vectors in Three Different Land Cover Types in Sarawak, Malaysian Borneo. Parasites Vectors 2017, 10, 1–14.

206. Calderón-Arguedas, O.; Troyo, A.; Solano, M.E.; Avendaño, A.; Beier, J.C. Urban Mosquito Species (Diptera: Culicidae) of Dengue Endemic Communities in the Greater Puntarenas Area, Costa Rica. Rev. Biol. Trop. 2009, 57, 1223–1234.

207. Madewell, Z.J.; Sosa, S.; Brouwer, K.C.; Juárez, J.G.; Romero, C.; Lenhart, A.; Cordón-Rosales, C. Associations between Household Environmental Factors and Immature Mosquito Abundance in Quetzaltenango, Guatemala. BMC Public Health 2019, 19, 1–11.

208. Guagliardo, S.A.; Barboza, J.L.; Morrison, A.C.; Astete, H.; Vazquez-Prokopec, G.; Kitron, U. Patterns of Geographic Expansion of Aedes aegypti in the Peruvian Amazon. PLoS Negl. Trop. Dis. 2014, 8, e3033.

209. Hemme, R.R.; Thomas, C.L.; Chadee, D.D.; Severson, D.W. Influence of Urban Landscapes on Population Dynamics in a Short-Distance Migrant Mosquito: Evidence for the Dengue Vector Aedes aegypti. PLoS Negl. Trop. Dis. 2010, 4, e634.

210. Fuller, D.O.; Troyo, A.; Beier, J.C. El Niño Southern Oscillation and Vegetation Dynamics as Predictors of Dengue Fever Cases in Costa Rica. Environ. Res. Lett. 2009, 4, 014011.

211. Mena, N.; Troyo, A.; Bonilla-Carrión, R.; Calderón-Arguedas, Ó. Factors Associated with Incidence of Dengue in Costa Rica | Factores Asociados Con La Incidencia de Dengue En Costa Rica. Rev. Panam. Salud Publica/Pan Am. J. Public Health 2011, 29.

212. Campbell-Lendrum, D.; Manga, L.; Bagayoko, M.; Sommerfeld, J. Climate Change and Vector-Borne Diseases: What Are the Implications for Public Health Research and Policy? Philos. Trans. R. Soc. B: Biol. Sci. 2015, 370, 1–8.

213. Kramer, L.D. Complexity of Virus–Vector Interactions. Curr. Opin. Virol. 2016, 21, 81–86.

214. Chaves, L.F.; Valerín Cordero, J.A.; Delgado, G.; Aguilar-Avendaño, C.; Maynes, E.; Gutiérrez Alvarado, J.M.; Ramírez Rojas, M.; Romero, L.M.; Marín Rodríguez, R. Modeling the Association between Aedes aegypti Ovitrap Egg Counts, Multi-Scale Remotely Sensed Environmental Data and Arboviral Cases at Puntarenas, Costa Rica (2017–2018). Curr. Res. Parasitol. Vector-Borne Dis. 2021, 1, 100014.

215. Fuller, D.O.; Troyo, A.; Calderón-Arguedas, O.; Beier, J.C. Dengue Vector (Aedes aegypti) Larval Habitats in an Urban Environment of Costa Rica Analysed with ASTER and QuickBird Imagery. Int. J. Remote Sens. 2010, 31, 3–11.

216. Troyo, A.; Fuller, D.O.; Calderón-Arguedas, O.; Solano, M.E.; Beier, J.C. Urban Structure and Dengue Incidence in Puntarenas, Costa Rica. Singap. J. Trop. Geogr. 2009, 30, 265–282.

217. Joyce, A.L.; Alvarez, F.S.; Hernandez, E. Forest Coverage and Socioeconomic Factors Associated with Dengue in El Salvador, 2011–2013. Vector Borne Zoonotic Dis. 2021, 21, 602–613.

218. Benedict, M.Q.; Levine, R.S.; Hawley, W.A.; Lounibos, L.P. Spread of The Tiger: Global Risk of Invasion by The Mosquito Aedes albopictus. Vector Borne Zoonotic Dis. 2007, 7, 76–85.

219. Lugo, E.D.C.; Moreno, G.; Zachariah, M.A.; López, M.M.; López, J.D.; Delgado, M.A.; Valle, S.I.; Espinoza, P.M.; Salgado, M.J.; Pérez, R.; et al. Identification of Aedes albopictus in Urban Nicaragua. J. Am. Mosq. Control Assoc. 2005, 21, 325–327.

220. Marín, R.; Marquetti, M.d.C.; Álvarez, Y.; Gutiérrez, J.M.; González, R. Especies de Mosquitos (Diptera: Culicidae) y Sus Sitios de Cría En La Región Huetar Atlántica, Costa Rica. Rev. Bioméd. 2009, 20, 15–23.

221. Ogata, K.; Lopez Samayoa, A. Discovery of Aedes albopictus in Guatemala. J. Am. Mosq. Control Assoc. 1996, 12, 503–506.

222. Ortega-Morales, A.I.; Mis-Avila, P.; Domínguez-Galera, M.; Canul-Amaro, G.; Esparza-Aguilar, J.; Carlos-Azueta, J.; Badillo-Perry, S.; Marin, P.; Polanco, J.; Fernández-Salas, I. First Record of Stegomyia albopicta

223. Belli, A.; Arostegui, J.; Garcia, J.; Aguilar, C.; Lugo, E.; Lopez, D.; Valle, S.; Lopez, M.; Harris, E.; Coloma, J. Introduction and Establishment of Aedes albopictus (Diptera: Culicidae) in Managua, Nicaragua. J. Med. Entomol. 2015, 52, 713–718.

224. Marín-Rodríguez, R.; Calderón-Arguedas, O.; Díaz-Ríos, M.; Duarte-Solano, G.; Valle Arguedas, J.J.; Troyo-Rodríguez, A. Primer Hallazgo de Aedes albopictus Skuse En El Gran Área Metropolitana de Costa Rica. Rev. Costarric. Salud Pública 2014, 23, 1–4.

225. Rojas-Araya, D.; Marín-Rodríguez, R.; Gutiérrez-Alvarado, M.; Romero-Vega, L.M.; Calderón-Arguedas, O.; Troyo, A. Nuevos Registros de Aedes albopictus (Skuse) En Cinco Localidades de Costa Rica. Rev. Bioméd. 2017, 28, 79–88.

226. Wagman, J.; Grieco, J.P.; King, R.; Briceño, I.; Bautista, K.; Polanco, J.; Pecor, J.; Achee, N.L. First Record and Demonstration of a Southward Expansion of Aedes albopictus into Orange Walk Town, Belize, Central America. J. Am. Mosq. Control Assoc. 2013, 29, 380–382.

227. Futami, K.; Valderrama, A.; Baldi, M.; Minakawa, N.; Marín Rodríguez, R.; Chaves, L.F. New and Common Haplotypes Shape Genetic Diversity in Asian Tiger Mosquito Populations from Costa Rica and Panamá. J. Econ. Entomol. 2015, 108, 761–768.

228. Calderón-Arguedas, Ó.; Moreira-Soto, R.D.; Vicente-Santos, A.; Corrales-Aguilar, E.; Rojas-Araya, D.; Troyo, A.; Calderón-Arguedas, Ó.; Moreira-Soto, R.D.; Vicente-Santos, A.; Corrales-Aguilar, E.; et al. Papel Potencial de Aedes albopictus Skuse En La Transmisión de Virus Dengue (DENV) En Una Zona de Actividad Piñera de Costa Rica. Rev. Bioméd. 2019, 30, 33–41.

229. Calderón-Arguedas, O.; Troyo, A.; Moreira-Soto, R.D.; Marín, R.; Taylor, L. Dengue Viruses in Aedes albopictus Skuse from a Pineapple Plantation in Costa Rica. J. Vector Ecol. 2015, 40, 184–186.

230. Hanley, K.A.; Monath, T.P.; Weaver, S.C.; Rossi, S.L.; Richman, R.L.; Vasilakis, N. Fever versus Fever: The Role of Host and Vector Susceptibility and Interspecific Competition in Shaping the Current and Future Distributions of the Sylvatic Cycles of Dengue Virus and Yellow Fever Virus. Infect. Genet. Evol. 2013, 19, 292–311.

231. Hubálek, Z.; Rudolf, I.; Nowotny, N. Arboviruses Pathogenic for Domestic and Wild Animals. Adv. Virus Res. 2014, 89, 201–275.

232. Vasilakis, N.; Weaver, S.C. Flavivirus Transmission Focusing on Zika. Curr. Opin. Virol. 2017, 22, 30–35.

233. Rezza, G.; Weaver, S.C. Chikungunya as a Paradigm for Emerging Viral Diseases: Evaluating Disease Impact and Hurdles to Vaccine Development. PLoS Negl. Trop. Dis. 2019, 13, e0006919.

234. Vasilakis, N.; Weaver, S.C. Chapter 1—The History and Evolution of Human Dengue Emergence. Adv. Virus Res. 2008, 72, 1–76.

235. Pinheiro, F.; LeDuc, J. The Arboviruses: Epidemiology and Ecology. In The Arboviruses: Epidemiology and Ecology; Monath, T.P., Ed.; CRC Press: Boca Raton, FL, USA, 2019; Volume 3, pp. 137–150.

236. Figueiredo, L.T.M. Human Urban Arboviruses Can Infect Wild Animals and Jump to Sylvatic Maintenance Cycles in South America. Front. Cell. Infect. Microbiol. 2019, 9, 259.

237. Althouse, B.M.; Guerbois, M.; Cummings, D.A.T.; Diop, O.M.; Faye, O.; Faye, A.; Diallo, D.; Sadio, B.D.; Sow, A.; Faye, O.; et al. Role of Monkeys in the Sylvatic Cycle of Chikungunya Virus in Senegal. Nat. Commun. 2018, 9, 1–10.

238. Trapido, H.; Galindo, P. The Epidemiology of Yellow Fever in Middle America. Exp. Parasitol. 1956, 5, 285–323.

239. Hendy, A.; Hernandez-Acosta, E.; Valério, D.; Mendonça, C.; Costa, E.R.; Júnior, J.T.A.; Assunção, F.P.; Scarpassa, V.M.; Gordo, M.; Fé, N.F.; et al. The Vertical Stratification of Potential Bridge Vectors of Mosquito-Borne Viruses in a Central Amazonian Forest Bordering Manaus, Brazil. Sci. Rep. 2020, 10, 1–13.

240. Yasuoka, J.; Levins, R. Impact of Deforestation and Agricultural Development on Anopheline Ecology and Malaria Epidemiology. Am. J. Trop. Med. Hyg. 2007, 76, 450–460.

241. Carter, K.H.; Singh, P.; Mujica, O.J.; Escalada, R.P.; Ade, M.P.; Castellanos, L.G.; Espinal, M.A. Malaria in the Americas: Trends from 1959 to 2011. Am. J. Trop. Med. Hyg. 2015, 92, 302–316.

242. Alvarez Pineda, M.; Bayles, B.; Dagy, K.; Kelly, E.; Martin, S.; Faerron, C. Spatial Dynamics of Vector-Borne Disease Risk in Costa Rica: Examining the Role of Anthropogenic Landscapes, Indigenous Territories, and Protected Areas in a Biodiversity Hotspot. In Proceedings of the American Geophysical Union, Fall Meeting 2019, San Francisco, CA, USA, 9–13 December 2019; Volume 2019.

243. MacDonald, A.J.; Mordecai, E.A. Amazon Deforestation Drives Malaria Transmission, and Malaria Burden Reduces Forest Clearing. Proc. Natl. Acad. Sci. USA 2019, 116, 22212–22218.

244. Laporta, G.Z.; Ilacqua, R.C.; Bergo, E.S.; Chaves, L.S.M.; Rodovalho, S.R.; Moresco, G.G.; Figueira, E.A.G.; Massad, E.; de Oliveira, T.M.P.; Bickersmith, S.A.; et al. Malaria Transmission in Landscapes with Varying Deforestation Levels and Timelines in the Amazon: A Longitudinal Spatiotemporal Study. Sci. Rep. 2021, 11, 1–14.

245. Bourke, B.P.; Conn, J.E.; de Oliveira, T.M.P.; Chaves, L.S.M.; Bergo, E.S.; Laporta, G.Z.; Sallum, M.A.M. Exploring Malaria Vector Diversity on the Amazon Frontier. Malar. J. 2018, 17, 1–17.

246. Sallum, M.A.M.; Conn, J.E.; Bergo, E.S.; Laporta, G.Z.; Chaves, L.S.M.; Bickersmith, S.A.; de Oliveira, T.M.P.; Figueira, E.A.G.; Moresco, G.; Olívêr, L.; et al. Vector Competence, Vectorial Capacity of Nyssorhynchus darlingi and the Basic Reproduction Number of Plasmodium vivax in Agricultural Settlements in the Amazonian Region of Brazil. Malar. J. 2019, 18, 1–15.

247. Guerra, C.A.; Snow, R.W.; Hay, S.I. A Global Assessment of Closed Forests, Deforestation and Malaria Risk. Ann. Trop. Med. Parasitol. 2006, 100, 189–204.

248. Torres-Cosme, R.; Rigg, C.; Santamaría, A.M.; Vásquez, V.; Victoria, C.; Ramirez, J.L.; Calzada, J.E.; Carrera, L.C. Natural Malaria Infection in Anophelines Vectors and Their Incrimination in Local Malaria Transmission in Darién, Panama. PLoS ONE 2021, 16, e0250059.

249. Vittor, A.Y.; Gilman, R.H.; Tielsch, J.; Glass, G.; Shields, T.; Lozano, W.S.; Pinedo-Cancino, V.; Patz, J.A. The Effect of Deforestation on the Human-Biting Rate of Anopheles darlingi, the Primary Vector of Falciparum Malaria in the Peruvian Amazon. Am. J. Trop. Med. Hyg. 2006, 74, 3–11.

250. Chapin, G.; Wasserstrom, R. Agricultural Production and Malaria Resurgence in Central America and India. Nature 1981, 293, 181–185.

251. Dusfour, I.; Achee, N.L.; Briceno, I.; King, R.; Grieco, J.P. Comparative Data on the Insecticide Resistance of Anopheles albimanus in Relation to Agricultural Practices in Northern Belize, CA. J. Pest Sci. 2010, 83, 41–46.

252. Grieco, J.P.; Johnson, S.; Achee, N.L.; Masuoka, P.; Pope, K.; Rejmánková, E.; Vanzie, E.; Andre, R.; Roberts, D. Distribution of Anopheles albimanus, Anopheles vestitipennis, and Anopheles crucians Associated with Land Use in Northern Belize. J. Entomol. 2006, 43, 614–622.

253. Hakre, S.; Masuoka, P.; Vanzie, E.; Roberts, D.R. Spatial Correlations of Mapped Malaria Rates with Environmental Factors in Belize, Central America. Int. J. Health Geogr. 2004, 3, 1–12.

254. Grieco, J.P.; Vogtsberger, R.C.; Achee, N.L.; Vanzie, E.; Andre, R.G.; Roberts, D.R.; Rejmankova, E. Evaluation of Habitat Management Strategies for the Reduction of Malaria Vectors in Northern Belize. J. Vector Ecol. J. Soc. Vector Ecol. 2005, 30, 235–243.

255. Chaves, L.S.M.; Bergo, E.S.; Conn, J.E.; Laporta, G.Z.; Prist, P.R.; Sallum, M.A.M. Anthropogenic Landscape Decreases Mosquito Biodiversity and Drives Malaria Vector Proliferation in the Amazon Rainforest. PLoS ONE 2021, 16, e0245087.

256. Sinka, M.E.; Pironon, S.; Massey, N.C.; Longbottom, J.; Hemingway, J.; Moyes, C.L.; Willis, K.J. A New Malaria Vector in Africa: Predicting the Expansion Range of Anopheles stephensi and Identifying the Urban Populations at Risk. Proc. Natl. Acad. Sci. USA 2020, 117, 24900–24908.

257. WHO (World Health Organization). Vector Alert: Anopheles stephensi Invasion and Spread. Available online: https://www.who.int/publications/i/item/WHO-HTM-GMP-2019.09 (accessed on 5 October 2021).

258. Seyfarth, M.; Khaireh, B.A.; Abdi, A.A.; Bouh, S.M.; Faulde, M.K. Five Years Following First Detection of Anopheles stephensi (Diptera: Culicidae) in Djibouti, Horn of Africa: Populations Established—Malaria Emerging. Parasitol. Res. 2019, 118, 725–732.

259. Cáceres, L.; Rovira, J.; Torres, R.; García, A.; Calzada, J.; Cruz, M.D. La Characterization of Plasmodium vivax Malaria Transmission at the Border of Panamá and Costa Rica. Biomédica 2012, 32, 557–569.

260. Surendran, S.N.; Sivabalakrishnan, K.; Sivasingham, A.; Jayadas, T.T.P.; Karvannan, K.; Santhirasegaram, S.; Gajapathy, K.; Senthilnanthanan, M.; Karunaratne, S.P.; Ramasamy, R. Anthropogenic Factors Driving Recent Range Expansion of the Malaria Vector Anopheles stephensi. Front. Public Health 2019, 7, 53.

261. WCS (Wildlife Conservation Society). People and Wildlife Now Threatened by Rapid Destruction of Central America's Forests. Available online: https://newsroom.wcs.org/News-Releases/articleType/ArticleView/articleId/10332/People-and-Wildlife-Now-Threatened-by-Rapid-Destruction-of-Central-Americas-Forests.aspx (accessed on 5 October 2021).

262. McSweeney, K.; Nielsen, E.A.; Taylor, M.J.; Wrathall, D.J.; Pearson, Z.; Wang, O.; Plumb, S.T. Drug Policy as Conservation Policy: Narco-Deforestation. Science 2014, 343, 489–490.

263. Fontecha, G.; Pinto, A.; Archaga, O.; Betancourt, S.; Escober, L.; Henríquez, J.; Valdivia, H.; Montoya, A.; Mejía, R. Assessment of Plasmodium falciparum Antimalarial Drug Resistance Markers in Pfcrt and Pfmdr1 Genes in Isolates from Honduras and Nicaragua, 2018–2021. Malar. J. 2021. under review.

264. Barbour, A.G.; Fish, D. The Biological and Social Phenomenon of Lyme Disease. Science 1993, 260, 1610–1616.

265. Ogrzewalska, M.; Uezu, A.; Jenkins, C.N.; Labruna, M.B. Effect of Forest Fragmentation on Tick Infestations of Birds and Tick Infection Rates by Rickettsia in the Atlantic Forest of Brazil. EcoHealth 2011, 8, 320–331.

266. Wood, C.L.; Lafferty, K.D. Biodiversity and Disease: A Synthesis of Ecological Perspectives on Lyme Disease Transmission. Trends Ecol. Evol. 2013, 28, 239–247.

267. MacDonald, A.J. Abiotic and Habitat Drivers of Tick Vector Abundance, Diversity, Phenology and Human Encounter Risk in Southern California. PLoS ONE 2018, 13, e0201665.

268. Diuk-Wasser, M.A.; VanAcker, M.C.; Fernandez, M.P. Impact of Land Use Changes and Habitat Fragmentation on the Eco-Epidemiology of Tick-Borne Diseases. J. Med. Entomol. 2021, 58, 1546–1564.

269. Mendoza, H.; Rubio, A.V.; García-Peña, G.E.; Suzán, G.; Simonetti, J.A. Does Land-Use Change Increase the Abundance of Zoonotic Reservoirs? Rodents Say Yes. Eur. J. Wildl. Res. 2020, 66, 1–5.

270. Keesing, F.; Ostfeld, R.S. Impacts of Biodiversity and Biodiversity Loss on Zoonotic Diseases. Proc. Natl. Acad. Sci. USA 2021, 118, e2023540118.

271. Montenegro, D.C.; Bitencourth, K.; de Oliveira, S.v.; Borsoi, A.P.; Cardoso, K.M.; Sousa, M.S.B.; Giordano-Dias, C.; Amorim, M.; Serra-Freire, N.M.; Gazêta, G.S.; et al. Spotted Fever: Epidemiology and Vector-Rickettsia-Host Relationship in Rio de Janeiro State. Front. Microbiol. 2017, 8, 505.

272. Toepp, A.J.; Willardson, K.; Larson, M.; Scott, B.D.; Johannes, A.; Senesac, R.; Petersen, C.A. Frequent Exposure to Many Hunting Dogs Significantly Increases Tick Exposure. Vector Borne Zoonotic Dis. 2018, 18, 519–523.

273. Ellwanger, J.H.; Chies, J.A.B. The Triad "Dogs, Conservation and Zoonotic Diseases"—An Old and Still Neglected Problem in Brazil. Perspect. Ecol. Conserv. 2019, 17, 157–161.

274. Lönker, N.S.; Fechner, K.; Wahed, A.A. el Horses as a Crucial Part of One Health. Vet. Sci. 2020, 7, 28.

275. Álvarez, V.; Bonilla, R. Adultos y Ninfas de La Garrapata Amblyomma cajennense Fabricius (Acari: Ixodidae) En Equinos y Bovinos. Agron. Costarric. 2007, 31, 61–69.

276. Szabó, M.P.J.; Pinter, A.; Labruna, M.B. Ecology, Biology and Distribution of Spotted-Fever Tick Vectors in Brazil. Front. Cell. Infect. Microbiol. 2013, 3, 27.

277. Bermúdez, S.E.; Castro, A.M.; Trejos, D.; García, G.G.; Gabster, A.; Miranda, R.J.; Zaldívar, Y.; Paternina, L.E. Distribution of Spotted Fever Group Rickettsiae in Hard Ticks (Ixodida: Ixodidae) from Panamanian Urban and Rural Environments (2007–2013). EcoHealth 2016, 13, 274–284.

278. Ferrell, A.M.; Brinkerhoff, R.J.; Bernal, J.; Bermúdez, S.E. Ticks and Tick-Borne Pathogens of Dogs along an Elevational and Land-Use Gradient in Chiriquí Province, Panamá. Exp. Appl. Acarol. 2017, 71, 371–385.

279. Clarke-Crespo, E.; Moreno-Arzate, C.N.; López-González, C.A. Ecological Niche Models of Four Hard Tick Genera (Ixodidae) in Mexico. Animals 2020, 10, 649.

280. Dunn, L.H.; Clark, H.C. Notes on Relapsing Fever in Panama with Special Reference to Animal Hosts. Am. J. Trop. Med. Hyg. 1933, S1–S13, 201–209.

281. Álvarez-Calderon, V.; Hernández-Fonseca, V.; Hernández-Gamboa, J. Catálogo de Garrapatas Suaves (Acari: Argasidae) y Duras (Acari: Ixodidae) de Costa Rica. Brenesia 2005, 63–64, 81–88.

282. Bermúdez, C.S.E.; Zaldívar, A.Y.; Spolidorio, M.G.; Moraes-Filho, J.; Miranda, R.J.; Caballero, C.M.; Mendoza, Y.; Labruna, M.B. Rickettsial Infection in Domestic Mammals and Their Ectoparasites in El Valle de Antón, Coclé, Panamá. Vet. Parasitol. 2011, 177, 134–138.

283. Ogrzewalska, M.; Literák, I.; Capek, M.; Sychra, O.; Calderón, V.Á.; Rodríguez, B.C.; Prudencio, C.; Martins, T.F.; Labruna, M.B. Bacteria of the Genus Rickettsia in Ticks (Acari: Ixodidae) Collected from Birds in Costa Rica. Ticks Tick-Borne Dis. 2015, 6, 478–482.

284. Novakova, M.; Literak, I.; Chevez, L.; Martins, T.F.; Ogrzewalska, M.; Labruna, M.B. Rickettsial Infections in Ticks from Reptiles, Birds and Humans in Honduras. Ticks Tick-Borne Dis. 2015, 6, 737–742.

285. Troyo, A.; Moreira-Soto, R.D.; Calderon-Arguedas, Ó.; Mata-Somarribas, C.; Ortiz-Tello, J.; Barbieri, A.R.M.; Avendaño, A.; Vargas-Castro, L.E.; Labruna, M.B.; Hun, L.; et al. Detection of Rickettsiae in Fleas and Ticks from Areas of Costa Rica with History of Spotted Fever Group Rickettsioses. Ticks Tick-Borne Dis. 2016, 7, 1128–1134.

286. Lopes, M.G.; Junior, J.M.; Foster, R.J.; Harmsen, B.J.; Sanchez, E.; Martins, T.F.; Quigley, H.; Marcili, A.; Labruna, M.B. Ticks and Rickettsiae from Wildlife in Belize, Central America. Parasites Vectors 2016, 9, 1–7.

287. Polsomboon, S.; Hoel, D.F.; Murphy, J.R.; Linton, Y.-M.; Motoki, M.; Robbins, R.G.; Bautista, K.; Briceño, I.; Achee, N.L.; Grieco, J.P.; et al. Molecular Detection and Identification of Rickettsia Species in Ticks (Acari: Ixodidae) Collected from Belize, Central America. J. Med. Entomol. 2017, 54, 1718–1726.

288. Domínguez, L.; Miranda, R.J.; Torres, S.; Moreno, R.; Ortega, J.; Bermúdez, S.E. Hard Tick (Acari: Ixodidae) Survey of Oleoducto Trail, Soberania National Park, Panama. Ticks Tick-Borne Dis. 2019, 10, 830–837.

289. Esser, H.J.; Herre, E.A.; Kays, R.; Liefting, Y.; Jansen, P.A. Local Host-Tick Coextinction in Neotropical Forest Fragments. Int. J. Parasitol. 2019, 49, 225–233.

290. Esser, H.J.; Herre, E.A.; Blüthgen, N.; Loaiza, J.R.; Bermúdez, S.E.; Jansen, P.A. Host Specificity in a Diverse Neotropical Tick Community: An Assessment Using Quantitative Network Analysis and Host Phylogeny. Parasites Vectors 2016, 9, 1–14.

291. Alvarez, V.; Bonilla, R.; Chacón, I. Frecuencia Relativa de Boophilus microplus (Acari: Ixodidae) En Bovinos (Bos taurus y B. indicus) En Ocho Zonas Ecológicas de Costa Rica. Rev. Biol. Trop. 2003, 51, 427–434.

292. Alvarez, C.; Bonilla, R.; Chacón, I. Distribución de La Garrapata Amblyomma cajennense (Acari: Ixodidae) Sobre Bos Taurus y Bos indicus En Costa Rica. Rev. Biol. Trop. 2000, 48, 129–135.

293. Düttmann, C.; Flores, B.; Kadoch, Z.N.; Bermúdez, C.S. Hard Ticks (Acari: Ixodidae) of Livestock in Nicaragua, with Notes about Distribution. Exp. Appl. Acarol. 2016, 70, 125–135.

294. Bermúdez, S.; Domínguez, L.; Troyo, A.; Montenegro, V.; Venzal, J. Ticks Infesting Humans in Central America: A Review of Their Relevance in Public Health. Curr. Res. Parasitol. Vector Borne Dis. 2021, in press.

295. Bermúdez, C.S.E.; Castro, A.; Esser, H.; Liefting, Y.; García, G.; Miranda, R.J. Ticks (Ixodida) on Humans from Central Panama, Panama (2010–2011). Exp. Appl. Acarol. 2012, 58, 81–88.

296. de Rodaniche, E.C. Natural Infection of the Tick, Amblyomma cajennense, with Rickettsia rickettsii in Panama. Am. J. Trop. Med. Hyg. 1953, 2, 696–699.

297. Nieri-Bastos, F.A.; Marcili, A.; de Sousa, R.; Paddock, C.D.; Labruna, M.B. Phylogenetic Evidence for the Existence of Multiple Strains of Rickettsia parkeri in the New World. Appl. Environ. Microbiol. 2018, 84.

298. Carreno, R.; Durden, L.A.; Brooks, D.R.; Abrams, A.; Hoberg, E.P. Parelaphostrongylus tenuis (Nematoda: Protostrongylidae) and Other Parasites of White-Tailed Deer (Odocoileus virginianus) in Costa Rica. Comp. Parasitol. 2001, 68, 177–184.

299. Álvarez-Robles, E.; Fuentes-Rousselin, H.; Meoño-Sánchez, E.; Recinos-Donis, R.; Figueroa, L.; Guerra-Centeno, D. Preliminary Study of External Parasites of White-Tailed Deer (Odocoileus virginianus) from the Natural Reserve of the Parachute Brigade, San José, Escuintla Guatemala. Rev. Elect. Vet. 2018, 19, 1–11.

300. Bermúdez, S.; Miranda, R. Distribución de Los Ectoparásitos de Canis lupus familiaris L. (Carnivora: Canidae) de Panamá. Rev. MVZ Córdoba 2011, 16, 2274–2282.

301. Troyo, A.; Calderón-Arguedas, Ó.; Alvarado, G.; Vargas-Castro, L.E.; Avendaño, A. Ectoparasites of Dogs in Home Environments on the Caribbean Slope of Costa Rica. Rev. Bras. Parasitol. Vet. 2012, 21, 179–183.

302. Nava, S.; Estrada-Peña, A.; Petney, T.; Beati, L.; Labruna, M.B.; Szabó, M.P.J.; Venzal, J.M.; Mastropaolo, M.; Mangold, A.J.; Guglielmone, A.A. The Taxonomic Status of Rhipicephalus sanguineus (Latreille, 1806). Vet. Parasitol. 2015, 208, 2–8.

303. Álvarez-Hernández, G.; Roldán, J.F.G.; Milan, N.S.H.; Lash, R.R.; Behravesh, C.B.; Paddock, C.D. Rocky Mountain Spotted Fever in Mexico: Past, Present, and Future. Lancet Infect. Dis. 2017, 17, e189–e196.

304. Campos, S.D.E.; da Cunha, N.C.; Machado, C.d.S.C.; Telleria, E.L.; Cordeiro, M.D.; da Fonseca, A.H.; Toma, H.K.; dos Santos, J.P.C.; Almosny, N.R.P. Rickettsial Pathogens Circulating in Urban

Districts of Rio de Janeiro, without Report of Human Brazilian Spotted Fever. Rev. Bras. Parasitol. Vet. 2020, 29, 1–10.

305. Hun, L.; Herrero, L.; Fuentes, L.; Vargas, M. Tres Nuevos Casos de Fiebre Manchada de Las Montañas Rocosas En Costa Rica. Rev. Costarric. Cienc. Med. 1991, 12, 51–56.

306. Murgas, I.L.; Castro, A.M.; Bermúdez, S.E. Current Status of Amblyomma ovale (Acari: Ixodidae) in Panama. Ticks Tick-Borne Dis. 2013, 4, 164–166.

307. Barbieri, A.R.M.; Filho, J.M.; Nieri-Bastos, F.A.; Souza, J.C.; Szabó, M.P.J.; Labruna, M.B. Epidemiology of Rickettsia sp. Strain Atlantic Rainforest in a Spotted Fever-Endemic Area of Southern Brazil. Ticks Tick-Borne Dis. 2014, 5, 848–853.

308. Londoño, A.F.; Díaz, F.J.; Valbuena, G.; Gazi, M.; Labruna, M.B.; Hidalgo, M.; Mattar, S.; Contreras, V.; Rodas, J.D. Infection of Amblyomma ovale by Rickettsia sp. Strain Atlantic Rainforest, Colombia. Ticks Tick-Borne Dis. 2014, 5, 672–675.

309. Lamattina, D.; Tarragona, E.L.; Nava, S. Molecular Detection of the Human Pathogen Rickettsia parkeri Strain Atlantic Rainforest in Amblyomma ovale Ticks in Argentina. Ticks Tick-Borne Dis. 2018, 9, 1261–1263.

310. Vargas, V.M. Occurrence of the Bat Tick Ornithodoros (Alectorobius) kelleyi Cooley & Kohls (Acari: Argasidae) in Costa Rica and Its Relation to Human Bites. Rev. Biol. Trop. 1984, 32, 103–107.

311. Rangel, G.; Bermúdez, C.S.E. Nota Sobre Un Caso de Parasitismo de Ornithodoros sp. (Ixodida: Argasidae) En Una Mujer Proveniente de La Laja, Los Santos, Panamá. Rev. Méd. Panamá 2013, 37–39.

312. Bermúdez, S.E.; Castillo, E.; Pohlenz, T.D.; Kneubehl, A.; Krishnavajhala, A.; Domínguez, L.; Suárez, A.; López, J.E. New Records of Ornithodoros puertoricensis Fox 1947 (Ixodida: Argasidae) Parasitizing Humans in Rural and Urban Dwellings, Panama. Ticks Tick-Borne Dis. 2017, 8, 466–469.

313. Uspensky, I.V. Blood-Sucking Ticks (Acarina, Ixodoidea) as an Essential Component of the Urban Environment. Entomol. Rev. 2017, 97, 941–969.

314. Montenegro, V.M.; Bonilla, M.C.; Kaminsky, D.; Romero-Zúñiga, J.J.; Siebert, S.; Krämer, F. Serological Detection of Antibodies to Anaplasma spp., Borrelia burgdorferi Sensu Lato and Ehrlichia canis and of Dirofilaria immitis Antigen in Dogs from Costa Rica. Vet. Parasitol. 2017, 236, 97–107.

315. Sergio E. Bermúdez, C.; Lady Mejía, B.; Ligia Hernández; Dmitry, A. First Records of Ixodes boliviensis Neumann, 1904 and Dermacentor dissimilis Cooley, 1947 (Ixodida: Ixodidae) as Parasites of Domestic Mammals in Nicaragua. Syst. Appl. Acarol. 2015, 20, 462–464.

316. Foley, J.E.; Nieto, N.C.; Foley, P. Emergence of Tick-Borne Granulocytic Anaplasmosis Associated with Habitat Type and Forest Change in Northern California. Am. J. Trop. Med. Hyg. 2009, 81, 1132–1140.

317. Millins, C.; Gilbert, L.; Medlock, J.; Hansford, K.; Thompson, D.B.; Biek, R. Effects of Conservation Management of Landscapes and Vertebrate Communities on Lyme Borreliosis Risk in the United Kingdom. Philos. Trans. R. Soc. B Biol. Sci. 2017, 372.