Journal of Medical Entomology, 2017, 1–24
doi: 10.1093/jme/tjw209
Forum

Forum

Emergent and Reemergent Arboviruses in South America and the Caribbean: Why So Many and Why Now?

Carlos Brisola Marcondes,[1,2] Marta Contigiani,[3] and Raquel Miranda Gleiser[4]

[1]Departamento de Microbiologia, Imunologia e Parasitologia, Centro de Ciências Biológicas, Universidade Federal de Santa Catarina, Florianópolis, Santa Catarina 88040-900, Brazil (cbrisolamarcondes@gmail.com), [2]Corresponding author, e-mail: cbrisolamarcondes@gmail.com, [3]Emeritus Professor, Instituto de Virologia Dr. J. M. Vanella, Enfermera Gordillo Gomez s/n, Ciudad Universitaria, National University of Córdoba, Córdoba, Argentina (martacontigiani@gmail.com), and [4]Centro de Relevamiento y Evaluación de Recursos Agrícolas y Naturales (CREAN) – Instituto Multidisciplinario de Biología Vegetal (IMBIV), Universidad Nacional de Córdoba (UNC) and Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), Córdoba, Argentina (raquelgleiser@hotmail.com)

Subject Editor: William Reisen

Received 7 June 2016; Editorial decision 20 October 2016

Abstract

Several arboviruses have emerged and/or reemerged in the New World in the past decades. Zika and chikungunya viruses, formerly restricted to Africa and perhaps Asia, invaded the continent, causing great concern. Dengue virus outbreaks have continued to occur in almost all countries, with millions of cases per year. West Nile virus rapidly invaded North America, and now cases have been found in Central and South America. Other arboviruses, such as Mayaro and Eastern equine encephalitis viruses, have increased their activity and have been found in new regions. Changes in pathogenicity have been documented for some viruses leading to unexpected disease. A diverse mosquito fauna, changing climate and vegetation, increased travel, and unplanned urbanization producing conditions for the proliferation of *Aedes aegypti* (L.), *Culex quinquefasciatus* Say, and other vectors have combined to strongly influence changes in the distribution and incidence of several arboviruses. The need for thorough studies of the mosquito fauna and modifications of environmental conditions, mostly in urban areas strongly influenced by social, political, and economic factors, is emphasized.

Key words: arbovirus, *Aedes aegypti*, ecology, dengue, Zika, urbanization

RESUMEN Varios arbovirus han emergido y/o reemergido en el Nuevo Mundo en las últimas décadas. Los virus Zika y chikungunya, anteriormente restringidos a África y quizás Asia, invadieron el continente, causando gran preocupación; además siguen ocurriendo brotes causados por el virus dengue en casi todos los países, con millones de casos por año. El virus West Nile invadió rápidamente América del Norte, y ya se han encontrado casos en América Central y del Sur. Otros arbovirus, como Mayaro y el virus de la encefalitis equina del este han aumentado su actividad y se han encontrado en nuevas regiones. Se han documentado cambios en la patogenicidad de algunos virus que conducen a enfermedades inesperadas. Una fauna diversa de mosquitos, cambios climáticos y en la vegetación, aumento de los viajes, y urbanizaciones no planificadas que generan condiciones adecuadas para la proliferación de *Aedes aegypti* (L.), *Culex quinquefasciatus* Say y otros mosquitos vectores, se han combinado para influir fuertemente en los cambios en la distribución y la incidencia de varios arbovirus. Se enfatiza la necesidad de realizar estudios exhaustivos de la fauna de mosquitos y modificaciones de las condiciones ambientales, sobre todo en las zonas urbanas fuertemente influenciadas por factores sociales, políticos y económicos.

Palabras clave: arbovírus, *Aedes aegypti*, ecología, dengue, Zika, urbanización

Several endemic and introduced mosquito-borne arboviruses are of historic and recent public and veterinary health concern in the New World (Table 1). Endemic Mayaro virus (MAYV), for example, is associated with monkeys in some localities in the Amazon basin, Trinidad, Venezuela, and Colombia, and recently, it has been reported in patients diagnosed in the southeastern Brazilian state of São Paulo (Coimbra et al. 2007), where *Aedes aegypti* (L.), a good experimental vector (Long et al. 2011), is abundant. In addition,

© The Authors 2017. Published by Oxford University Press on behalf of Entomological Society of America.
All rights reserved. For Permissions, please email: journals.permissions@oup.com

Table 1. Main arboviruses found in the New World

Genus	Antigenic complex	Viral species	Associated clinical syndrome	Main vector	Current distribution	References
Alphavirus	Eastern Equine Encephalitis (EEEV)	EEEV	FIE	*Culiseta melanura*	USA	Weaver et al. (2012)
		Madariaga	FIE	*Culex (Melanoconion)*	Central and South America	Arrigo et al. (2010), Go et al. (2014)
	Venezuelan Equine Encephalitis (VEEV)	VEEV (epizootic)	FIE	Mammalophilic mosquitoes (*Aedes, Psorophora*)	South, Central and North America (Texas)	Weaver et al. (2004) Weaver and Reisen (2010)
		VEEV (enzootic)	FIE	*Culex, Aedes (Ochlerotatus)*	South and Central America	Weaver et al. (2004) Brault et al. (2004)
		Everglades	FIE	*Culex cedecei*	North America (Florida)	Aguilar et al. (2011)
		Mucambo	FIA	*Culex (Melanoconion)*	South America	Aguilar et al. (2011)
		Tonate	FIE	Unknown	Brazil USA (Colorado)	Hommel et al. (2000) Aguilar et al. (2011)
		Pixuna	FIA	Mammalophilic mosquitoes? Mammalophilic mosquitoes?	Brazil Argentina	Shope et al. (1964) Pisano et al. (2010b)
		Rio Negro	FIA	Mammalophilic mosquitoes	Argentina	Pisano et al. (2014)
	Semliki Forest	Mayaro	FIA	*Haemagogus* [S] *Aedes (Stegomyia) aegypti* [U]	South America	Muñoz and Navarro (2012)
		Chikungunya	FIA	*Ae. (Diceromyia) furcifer, Ae. (Ste.) africanus Ae. aegypti, Ae. (Ste.) albopictus* [U]	Africa, Asia Europe, America	Coffey et al. (2014); Weaver (2014); Powers (2015)
Flavivirus	Dengue	Dengue	FIH	*Aedes aegypti/Ae. albopictus*	Worldwide	Fares et al. (2015)*
	Yellow Fever	Yellow Fever	FIE	*Haemagogus* [S], *Aedes aegypti* [U]	Africa, America	Huang et al. (2014)
	Japanese Encephalitis	Japanese Encephalitis	FIE	*Culex*	Asia, Oceania	Van der Hurk et al. (2009)
		West Nile Virus	FIE	*Culex*	Worldwide?	Kilpatrick et al. (2005) Turell et al. (2005a)
		Saint Louis Encephalitis	FIA	*Culex*	North, Central and South America	Reisen (2003) Diaz et al. (2013a)
	Spondweni	Zika	FIA/FIE	*Aedes aegypti*	Africa, Asia, Australia continent (Oceania), South and Central America	Hayes (2009) Duffy et al. (2009)
	Ntaya	Ilheus/Rocio	FI/FIE	*Aedes, Psorophora*	South America	Pauvolid-Corrêa (2013) Coimbra et al. (2008)
Orthobunyavirus		BUN/Cache Valley	FIA/FIE?	*Aedes (Ochlerotatus)? Anopheles?*, other *Aedes?*	Africa, North, Central and South America	Holden and Hess (1959) Vasconcelos and Calisher (2016)
		Oropouche		*Culicoides paraensis*, *Cx. quinquefasciatus* and possibly *Ae. serratus* and *Coquillettidia venezuelensis*.	South America	Vasconcelos and Calisher (2016)

FIE, febrile illness and encephalitis; FIA, febrile illness with arthralgia; FI, febrile illness; FIH, febrile illness with hemorrhage; S, sylvan; U, urban; potential for introduction in the Americas.

*Both species were cited, but emphasizing the lower susceptibility of *Ae. albopictus*, based on Lambrechts et al. (2010).

recently introduced West Nile virus (WNV), Zika virus (ZIKV), and chikungunya virus (CHIKV) and others such as yellow fever virus (YFV) and dengue virus (DENV), introduced centuries ago, afflict populations in almost all countries (WHO 2016). WNV, originally from Africa, has been found from the United States to Buenos Aires in Argentina (Díaz et al. 2008a). CHIKV and ZIKV, previously restricted to Africa and perhaps Asia, have dispersed to most countries in the Americas; the only countries still free of autochthonous cases

are Uruguay and Chile (except for a sexually transmitted ZIKV case – http://www.who.int/csr/don/15-april-2016-zika-chile/en/; ZIKV has been found in Easter Island). DENV, CHIKV, WNV, and, more recently, ZIKV together have resulted in the highest rate of illness and mortality among emerging and/or reemerging viruses in the New World (Liang et al. 2015).

These recent invasions coupled with an increased occurrence (or awareness?) of native arboviral diseases have prompted interest in the factors leading to arbovirus emergence/reemergence. The epidemiologies of the North American viruses and of recent introductions to that region have been amply covered in the literature. Herein, we review the most important emergent and/or reemergent arboviruses in South America and the Caribbean and the factors possibly causing this emergence and suggest possible solutions for the problem. Owing to the large number of mosquito-transmitted arboviruses in the continent, only those most relevant (or known) from a medical point of view are reviewed herein. Abbreviations for mosquito genera proposed by Reinert (1975) were utilized, with *Aedes* considered as a comprehensive genus (Wilkerson et al. 2015).

Mosquito-Borne Arboviruses – History and Epidemiology

Reeves (2001) proposed the word arbovirus (initially as arborvirus) for viruses transmitted by arthropods in the 1950s, according to his autobiographical review. More than 500 species and varieties of arboviruses have been found globally, with more than half transmitted by mosquitoes. The New World, with its large area and great diversity of environments, supports many of them (Arbovirus Catalog 2016). Globalization and intensification of maritime traffic since the 16th century, mostly promoted by Portuguese, Spanish, and British vessels, were responsible not only for the introduction into the Americas of vegetal cultures, such as mangoes, apples, and sugarcane, but also arboviruses, such as YFV and DENV. Others, such as MAYV, already were circulating in sylvatic environments and have spread to new areas, causing disease and difficulty for diagnosis.

The YFV was probably brought from Africa to the New World in trading and slave ships, along with *Ae. aegypti* (L.), causing epidemics in cities from Baltimore to Buenos Aires (Spielman and D'Antonio 2002, Fernández et al. 2013). Although a disease considered as YF was referenced in the battle of Vega Real or Santo Cerro, fought in May 24, 1495 (https://es.wikipedia.org/wiki/Batalla_de_la_Vega_Real), by Columbus against the Indians (Nogueira 2009), the African origin of YFV has been accepted, and the concurrent introduction of the efficient vector, *Ae. aegypti*, undoubtedly promoted its transmission in urban areas in the Americas. YFV was the first virus demonstrated to cause human disease (Brès 1986). Controlled in most North and South American cities after the discovery of the role of *Ae. aegypti* in the transmission cycle in the beginning of the 20th century, its continued sylvatic circulation was proven in the 1930s (Soper et al. 1933), when the zoonotic cycle was described among New World monkeys and canopy mosquitoes, mostly of *Haemagogus* and *Sabethes* genera (Vasconcelos 2003). The reduction of control of *Ae. aegypti* in Rio de Janeiro in 1915, a mistake to be repeated several times, was responsible for the resurgence of YFV in the city (Barbosa 1929). In South America, although 95% of the current cases have been reported from Peru, Bolivia, Brazil, and Colombia, 10 other countries have conditions suitable for the occurrence of YFV (PAHO 2015). Although recent cases in Brazil have usually been reported from the Amazon forest, the center-west region, and western parts of populated states in the east, a suspected lethal human case was reported in 2015 from Natal, in the extreme northeast of the country (PAHO 2016).

Although DENV was referenced in Chinese texts in the Chin dynasty and formally described in the Tang and Northern Sung dynasties (265–992 AD), the disease was restricted to Southeast Asia, with suspected outbreaks in French West Indies and Panama in the 17th century. Starting in Batavia, present-day Jakarta, in 1779, DENV reached pandemic proportions in 1788, when it was described from Philadelphia by Benjamin Rush (Gubler 1998, Hanley et al. 2013), taking approximately 10 yr to circle the globe. DENV is now present in almost all countries in the Americas (except Canada, continental Chile, and some Caribbean islands, as Uruguay reported the first DENV outbreak in 2016; Quian 2016), with an estimated 2.3 million cases in 2015 (PAHO 2016). Successive and cocirculation of all four serotypes has led to more serious hemorrhagic fever linked to successive infections by different serotypes. Although mostly confined to urban areas and transmitted by *Ae. aegypti*, indications of adaptation to sylvatic environments have been found (Marcondes and Tauil 2010, Hemme et al. 2016).

The temporary control of *Ae. aegypti*, considered sufficient to interrupt YFV transmission, was replaced by the planning and implementation of an eradication campaign throughout the Americas in the 1950s; 21 countries were considered by Panamerican Health Organization (PAHO) to be free of the mosquito by the 1960s, whereas the United States, Venezuela, French Guiana, Suriname, Guiana, and several islands in the Caribbean remained infested (Soper 1963, Teixeira and Barreto 1996). Owing to apathy and resistance to DDT, *Ae. aegypti* became again widely distributed and now seems to be very difficult to control and even more so to eradicate. The species was reported in Brazil in Bahia and Rio de Janeiro states in 1976 and 1980, respectively (Schatzmayr 2000). Most Brazilian *Ae. aegypti* populations analyzed by mitochondrial DNA were found to be related to Southeast Asian genotypes, perhaps explaining the occurrence of several DENV epidemics in the country (Mousson et al. 2005). An exception was a population from Roraima that was related genetically to African populations and whose infection rate with DENV and YFV was similar to populations from Maracay, Venezuela (Lourenço-de-Oliveira et al. 2002, 2004).

After several passages by nervous tissue-deprived chicken embryo cells, Hugh Smith, working with Max Theiler, developed an attenuated strain from the Asibi strain of YFV, called 17D, which has been successfully utilized for vaccination globally to interrupt/prevent/control YFV (Frierson 2010). Although immunity produced by the application of a single primary dose of yellow fever 17D vaccine is considered enough for lifelong protection (Wieten et al. 2016), some failures in children caused the maintenance of a 10-yr periodicity of vaccination in endemic or epidemic circumstances, mostly for travelers and laboratory workers (Monath and Vasconcelos 2015).

Although the basic reproduction number (R_o) for YFV is 43% lower than that for DENV, the high indices of infestation by *Ae. aegypti* in several Brazilian cities indicate a continued risk for urban transmission (Massad et al. 2003). Butler (2016) emphasized the high risk of urbanization of YFV in Africa and South America, owing to infestation by *Ae. aegypti* and low incidence of vaccination and natural immunity in these populations. However, the small number of infected persons traveling from forests to urban areas infested by *Ae. aegypti* and the short infectious period in humans make the current urbanization of YFV unprobable.

CHIKV and ZIKV were recently introduced into the New World and were rapidly transmitted among humans in urban areas by *Ae. aegypti*. Chikungunya, previously called *kindinga pepo* in Swahili

(meaning "sudden cramp-like seizure caused by a bad spirit"), probably caused epidemics in Caribbean islands and southern United States in 1827-1828, following an outbreak in 1823 in Zanzibar, and pandemics of this disease have crossed the Indian Ocean at roughly 40- to 50-yr intervals. The name "dengue" was utilized for such clinical disease, but afterwards was restricted to true DENV infections, without the associated articular sequelae. In 1952, CHIKV was first isolated from an exanthematous patient and received this name from the Makonde dialect, meaning "that which bends up" (see Halstead 2015 and refs). Kuno (2015) extensively reviewed the history of etiologic confusion between DENV and CHIKV. Chikungunya, after causing several epidemics in Africa and Asia, many of them involving hundreds of thousands of people, was first reported in the island of Saint Martin in the Caribbean in October of 2013, and by the end of 2015, transmission had been reported in more than 50 territories (Faria et al. 2016, Weaver and Lecuit 2015). Even though *Ae. aegypti* is assumed to be the main vector because of vectorial capacity and feeding preference on humans, an Asian strain of CHIKV previously was shown to be much more efficient at infecting *Ae. albopictus* (Skuse) than *Ae. aegypti* (Turell et al. 1992). Moreover, an E1-A226V mutation in CHIKV tracked to the 2005–2006 Indian Ocean epidemic increased infectivity and reduced dissemination time in *Ae. albopictus*, thus accelerating transmission from infectious *Ae. albopictus* to the naïve human population (Tsetsarkin et al. 2007), and the dispersal of this variant may explain outbreaks in Europe (Bordi et al. 2008).

The first South American case of ZIKV was reported by the national authorities of Chile in Easter Island in February 2014 (Tognarelli et al. 2016), then Brazil in 2015 (Zanluca et al. 2015), and Argentina (Ministerio de Salud de la Nación 2016). The recent finding of natural infection of monkeys by ZIKV (Favoretto et al. 2016) in Brazil and by DENV in Puerto Rico (Hemme et al. 2016) indicates spillover from the infected human population and possibly the establishment of enzootic cycles. If this is followed by persistent transmission among these mammals of CHIKV, as occurs in Africa (Chevillon et al. 2008) and Asia (Apandi et al. 2009), containment will be very difficult and could create an epidemiological situation similar to YFV.

There was concern that DENV, CHIKV, and ZIKV, simultaneously occurring in Rio de Janeiro, constituted a risk for athletes and tourists in Olympic Games 2016 (Wilson and Schlagenhauf 2016), and that introduction into new areas was probable by visitors returning to their country of origin. However, the Olympics 2016 were held during the Southern Hemisphere winter, when low rainfall and relatively cool temperatures were less suitable for mosquito activity, and in the southeastern region, where the risk of infection by ZIKV was much lower than in the northeast (Lancet 2016). The risk of introduction of ZIKV through returning tourists and athletes after Olympic Games was considered negligible for most countries, and although 19 of them fulfilled most requisites for introduction of ZIKV after the games, only four countries (Chad, Djibouti, Eritrea, and Yemen) registered a significant rise in travel to Rio de Janeiro in August 2016 (Grills et al. 2016). Still, the simultaneous circulation of DENV, CHIKV, and ZIKV threatens the tourism industry of countries such as Brazil and other tropical areas that have detected the elevated activity of these viruses.

WNV was initially discovered in Africa (Smithburn et al. 1940), but in recent years, it has spread throughout Europe, the Mediterranean, and Asia (Kramer et al. 2008). After its introduction into the New World in 1999, possibly from Israel (Lanciotti et al. 1999), WNV became widely distributed in North and Central America (Komar and Clark 2006), with infections also reported from South America (Diaz et al. 2008a, Melandri et al. 2012, Osorio et al. 2012, Vieira et al. 2015), becoming perhaps the widest distributed arbovirus globally (Reisen 2013). Wild birds, including migrants, are involved in the transmission cycle of WNV, because they may act as amplifying hosts. Although the role of migratory birds in spreading zoonotic arboviruses remains speculative, laboratory and field studies indicate that migrating passerine birds could potentially disperse WNV (Reed et al. 2003, Komar and Clark 2006, Owen and Moore 2006). Osorio et al. (2012) discuss that the genetic similarity between the Colombia and Louisiana WNV isolates may be explained by migratory birds connecting these two regions, either directly or through the Caribbean. Infected birds also have been hypothesized to disperse Eastern equine encephalitis virus (EEEV) in North America, based on molecular epidemiologic patterns (Arrigo et al. 2010). Although no outbreaks of arboviruses in North America could be attributed to transportation between northern and southern latitudes by infected birds, their potential (sporadic?) involvement is indicated based on the finding of South American strains of EEEV in the Mississippi valley (Calisher et al. 1971). As reviewed by Rappole et al. (2000), several routes for the migration of birds could transport arboviruses across and between both continents, similar to that observed in the Old World (Hubálek and Halouzka 2000). A careful study of these avian species and their associated mosquitoes at several areas along known migration routes is needed for arboviruses whose amplifying hosts are birds.

Rocio virus, first described in the 1970s in the south Brazilian state of Sao Paulo, caused about 100 deaths and neurologic sequelae in 200 patients (Iversson and Coimbra 1984), and has been found in equines in four of five surveyed Brazilian states (Silva et al. 2014). It is evident that the distribution of endemic arboviruses in the Americas is much greater than is known.

Characterization and Importance of Arboviruses From the New World

Several tropical diseases, among which are included those produced by arboviruses, are considered by the World Health Organization (WHO) to be "neglected" and remain a continuing public health challenge (Le Beaud 2008). Arboviruses of medical and veterinary importance mainly belong to three families: Bunyaviridae, Flaviviridae, and Togaviridae (Table 1). Many of these zoonotic arboviruses (such as WNV) are maintained within cycles frequently involving avian hosts and spill over to occasionally infect humans and equines that do not produce viremias sufficient for mosquito infection and are not necessary for virus maintenance, as they represent an accident during biological transmission among vectors and hosts (Weaver and Barrett 2004). The complex maintenance dynamics of zoonotic viruses such as Saint Louis encephalitis virus (SLEV), WNV, EEEV, and Venezuelan equine encephalitis virus (VEEV) is better described as a transmission network than a specific transmission cycle (Diaz et al. 2013b).

In contrast, anthroponoses caused by ZIKV, CHIKV, DENV, and YFV infection, which evolved from primate–mosquito sylvan cycles, can generate viremia titers in the human host high enough to infect vector mosquitoes, frequently *Ae. aegypti* (Weaver and Barrett 2004). Following centuries of coadaptation, arbovirus such as DENV and CHIKV are efficiently transmitted by *Ae. aegypti* and *Ae. albopictus* mosquitoes and are amplified by humans in urban environments (Weaver and Reisen 2010). *Aedes albopictus* is relatively unsusceptible to YFV (Miller et al. 1989), possibly because their mutual contact in the Americas and Africa is relatively recent (Hanley et al. 2013).

Anthroponoses

Anthroponoses are mainly vectored by *Ae. aegypti* (and in some areas by other *Stegomyia* species), owing to their preference for humans and the likelihood that the female will feed multiple times during each gonotrophic cycle. *Aedes aegypti* is a mosquito adapted to urban areas, typically laying eggs in vessels containing clean water and even exploiting cesspits, as observed in Puerto Rico (Burke et al. 2010) and Brazil (Gil et al. 2015). The intermittent water supply in many cities induces the residents to store water in badly preserved domestic storage vessels and creates opportunities for the development of this mosquito and the transmission of several arboviruses. It is highly susceptible to YFV (Black et al. 2002, Lourenço-de-Oliveira et al. 2004) and, at least in the New World and Asia, is the principal vector of DENV. Its adaptation to urban areas and resistance to insecticides have prevented the reduction of populations sufficient to interrupt the transmission cycle of arboviruses and have facilitated viral dispersal to new areas. Even though *Ae. aegypti* proliferation is usually associated to precarious sanitary conditions of most cities, this is not necessarily always the case, as affluent communities may offer larval habitats in their gardens, as occurs in Key West, Florida (Radke et al. 2009). In temperate Buenos Aires, Argentina, the spatial heterogeneity in *Ae. aegypti* populations is mainly driven by temperature patterns (De Majo et al. 2013), and abundant populations are common in residential (even wealthy; Schweigmann, personal communication) areas (Carbajo et al. 2006; De Majo et al. 2013).

Aedes albopictus is well-adapted to suburban and rural areas, and besides being a good vector of several arboviruses, it may be a bridge vector for YFV between sylvatic and urban areas (Gomes et al. 1999, Gratz 2004, Paupy et al. 2013). Even in central Africa, with a diversified mosquito fauna, the invasion by *Ae. albopictus* has been considered as potentially increasing the transmission rate of ZIKV, CHIKV, and DENV (Ngoagouni et al. 2015). It was considered as the vector responsible for the 2001-2002 outbreak of DENV in Hawaii (Effler et al. 2005) in the absence of *Ae. aegypti*. Specific populations of *Ae. albopictus* may be highly susceptible to CHIKV, having been incriminated as the vector in an extensive outbreak (265,000 cases in 770,000 inhabitants, and 237 deaths) in La Réunion in 1977 (Charrell et al. 2007); a later mutation of CHIKV increased its adaptation to *Ae. albopictus* mosquitoes (Tsetsarkin et al. 2007). Experimentally, it is a good vector of WNV (Brustolin et al. 2016) and several other arboviruses (*Flavivirus*, *Alphavirus*, *Bunyavirus*, and others; Paupy et al. 2009), but because it is mostly mammalophagic, its importance for the transmission of viruses with birds as reservoirs would be mostly as a bridge vector.

Zika Virus

ZIKV is a member of genus *Flavivirus*, which includes viruses such as DENV, YFV, Japanese encephalitis virus (JEV), WNV, SLEV, and Rocio that are associated with important human diseases, and the most recently introduced arbovirus into the Americas. ZIKV was first isolated in 1947 in the Zika Forest (Kampala, Uganda; Dick et al. 1952), remained relatively silent for the next 60 yr, but recently has rapidly spread throughout Micronesia and the Americas (similar to CHIKV), reaching epidemic proportions especially in Brazil. Prior to 2007, it was known to only be responsible for causing sporadic human infections in Africa and Asia. That year, an outbreak occurred in Yap (Federated States of Micronesia; Duffy et al. 2009), where ZIKV was first detected out of Africa and Asia, leading to awareness that the virus could spread to other Pacific islands. In 2013-2014, an important ZIKV outbreak was reported in French Polynesia, co-circulating with CHIKV and DENV. During that outbreak, neurological and autoimmune complications (Guillain–Barré syndrome) were documented. The virus further spread to other Pacific Islands such as New Caledonia, Cook, Vanuatu, Solomon, and Easter Island, where Chile national authorities confirmed in February 2014, the first ZIKV autochthonous transmission case. Later in 2015, Brazil confirmed autochthonous transmission in the northeast of the country (Zanluca et al. 2015), from where the virus rapidly dispersed throughout the country and then to most of South America (Vorou 2016). Currently, many countries and territories are affected by autochthonous ZIKV transmission, including Colombia, Venezuela, Paraguay, and Bolivia. In the fall of 2016, Argentina had an outbreak of 18 confirmed indigenous cases in Tucuman province (Ministerio de Salud de la Nación 2016), preceded by confirmed imported cases from the same and different provinces (OPS-OMS 2016). These recent outbreaks of ZIKV infection in different regions of South America and the world indicate that this virus as well as CHIKV and DENV (associated with urbanization and globalization) can continue to spread wherever the *Ae. aegypti* vector is present or can be established in the future. Infection by ZIKV has been strongly suspected as causing microcephaly in newborns when infection occurred during pregnancy; this was suspected in French Polynesia (Besnard et al. 2014), but documented by Mlakar et al. (2016), who described ZIKV infection in a fetus aborted in Slovenia from a woman infected in Brazil, and by Perez et al. (2016), who described vertical transmission with fetal malformations in an imported case in a 17-wk pregnant woman in Spain. Rasmussen et al. (2016), analyzing the available data, concluded that there was a causal relationship between ZIKV and microcephaly and other serious brain anomalies. In February 2016, the WHO announced that these ZIKV outbreaks were a public health emergency of international concern (PHEIC; Petersen et al. 2016).

ZIKV has been isolated from sylvatic *Aedes (Stegomyia)* mosquitoes in Africa (Berthet et al. 2014), but the main urban vector in Africa and Asia is *Ae. aegypti*, even though other vectors may include *Ae. albopictus* (human outbreak in Gabón, Central Africa, in 2007; Paupy et al. 2009). Although assumed to be the main vectors in the Americas, their role is not clear. Vector competence assessments of *Ae. aegypti* and *Ae. albopictus* from different regions of North, Central, and South America suggest that, although they were susceptible to infection, they were unexpectedly low competent vectors for this virus (Chouin-Carneiro et al. 2016). Moreover, ZIKV detection/isolation from field-collected *Ae. aegypti* in the Americas has proved to be difficult according to several researchers attending the ZIKA workshops held in March 2016 in Recife, Brazil (http://www.theglobeandmail.com/news/world/who-may-be-leading-brazil-down-wrong-path-on-zika-virus/article29390468/). At least 25 species of mosquitoes have been found to be infected by this virus (Pujhari and Rasgon 2016), highlighting the need to evaluate whether other vectors may be involved in the New World.

There is laboratory and field evidence that *Culex quinquefasciatus* Say, an abundant urban mosquito and opportunistic species regarding host preferences (Molaei et al. 2007), could be involved as a vector (Ayres 2016, Guedes et al. 2016). However, *Culex pipiens* L. from California and New Jersey and *Cx. quinquefasciatus* from two areas in Florida could not be experimentally infected by ZIKV from Puerto Rico (Yan-Jang et al. 2016); neither could *Cx. quinquefasciatus* from Rio de Janeiro be infected by ZIKV from the same locality (Fernandes et al. 2016). In Chiapas, Mexico, 15 of 55 pools of *Ae. aegypti* were positive for ZIKV, whereas no *Cx. quinquefasciatus* positive pool was found (Guerbois et al. 2016). As the capacity of transmission of an arbovirus by mosquitoes may be influenced by experimental conditions (Kenney and Brault 2014, Long et al. 2011), including viremia, origin of mosquitoes, and strain of virus, their role

needs to be more thoroughly investigated. Regional variations on susceptibility to ZIKV, similar to those observed for WNV in *Cx. pipiens* complex (Vaidyanathan & Scott 2007, Reisen et al. 2008, Micieli et al. 2013), could influence the results of tests. Regional variations in bloodfeeding preferences may also influence the epidemiological involvement of these mosquitoes in virus transmission.

Experience accumulated over time shows that when a virus is introduced into new regions or ecosystems, new species of mosquitoes may be involved in the transmission cycle, resulting in a different behavior of the virus, making it either more or less severe compared with the region from which it came (Higgs 2016). The recent finding of ZIKV RNA in saliva of patients, more frequently than in blood (Musso et al. 2015b), produces an additional tool for diagnosis, and infective particles in saliva and urine (Bonaldo et al. 2016) also could provide an alternative mode of direct transmission. As WNV has been found in cloacal and oral samples from several birds (Komar et al. 2002), these findings in humans were not surprising. Moreover, there is concern that sexual transmission may be important for the spread of this virus, as has been found in the Americas (McCarthy 2016) and other continents (D'Ortenzio et al. 2016).

Chikungunya and Mayaro Viruses

CHIKV and MAYV produce outbreaks of acute febrile illness with arthropathy. Despite their different continental origins, CHIKV (Africa) and MAYV (America) are closely related genetically within the Semliki Forest Complex of the genus *Alphavirus* (*Togaviridae*; Sabattini et al. 1998b). Similar to DENV, CHIKV is transmitted to humans in urban cycles by *Ae. aegypti* and *Ae. albopictus* (Petersen and Powers 2016).

Between 1960 and 1980, CHIKV was isolated repeatedly from different regions of Africa and Asia, and major epidemics occurred in India and the Philippines. In 2004, the virus emerged in the Indian Ocean Islands (La Reunion, Lamu, Comoros, Seychelles, and others), where the most prevalent vector was *Ae. aegypti*. Later, after a mutation occurred in the gene encoding the surface protein of the African strain that increased its capacity to infect, to replicate, and to be efficiently transmitted by the Asian mosquito *Ae. albopictus* (Tsetsarkin et al. 2007), it spread to other areas where the latter mosquito was the dominant vector (Reunion Island, Mauritius). Subsequently, CHIKV caused explosive and extensive epidemics, with millions of cases in India, after a silent period of more than 30 yr (Simon et al. 2008, Sudeep and Parashar 2008). In 2006, imported cases by CHIKV were reported in different countries in Europe, with France and Italy having higher risk, owing to the number of visitors coming from endemic regions, mainly La Reunion and India, respectively. In 2007, a localized outbreak was reported for the first time in Europe in northeastern Italy, where *Ae. albopictus* was the vector (Rezza 2009). Between 2006 and 2011, imported cases were reported in the United States, Canada, Caribbean Islands, Brazil, Oceania, and Australia associated with travelers from India and Indian Ocean islands (Coffey et al. 2014, Morrison 2014). In late 2013, CHIKV was locally transmitted on the French Caribbean island of Saint Martin, from where it spread throughout the Americas. By 2014, CHIKV local transmission had been identified in 41 countries and territories in the Caribbean, Central America, South America, and North America (Weaver 2014, Powers 2015). Again, the mosquitoes that transmitted the virus were *Ae. aegypti* and *Ae. albopictus*. As CHIKV was new (or a recent reintroduction; Halstead 2015) to the Americas, most humans in the region were not immune, indicating that they were readily infected and spread the virus to other mosquitoes.

Genetic studies have identified three different lineages for CHIKV: Eastern, Central and Southern Africa (ECSA); West Africa; and Asian genotypes. Volk et al. (2010) identified a fourth lineage, the Indian Ocean lineage, which spread from the Comoros islands in 2004 and caused a large outbreak in India and Southeast Asia in 2005-2008 (Volk et al. 2010, Faria et al. 2016). The Asian lineage, more adapted to *Ae. aegypti*, was isolated from Saint Martin island and dispersed in the Americas (Weaver 2014). In 2014, a separate introduction of the ECSA lineage was documented in Feira de Santana, Brazil: a CHIKV genotype previously undetected in the Americas (Nunes et al. 2015).

Little is known about vertebrates that may be involved in CHIKV maintenance cycles. In urban populations, during epidemic periods, the virus is maintained by mosquito–human–mosquito transmission. In nonepidemic periods, reservoirs may be nonhuman primates or other unidentified vertebrates. An outbreak in monkeys could occur when herd immunity is low; these animals develop viremias but no serious clinical manifestations (Wolfe et al. 2001, Chen et al. 2010). The viremia obtained in the experimental infection of several reptiles and amphibians is temperature-dependent (Hartwig et al. 2016), suggesting a more complex maintenance network of CHIKV, possibly facilitating the overwintering of the virus. CHIKV was less frequently detected in saliva and urine from humans than in their blood (Musso et al. 2016).

MAYV probably is not strictly an anthroponosis, and its sylvan transmission cycle could be similar to that of YFV, with monkeys as hosts and *Haemagogus* mosquitoes as vectors. This virus was first isolated from a febrile human in Trinidad in 1954 (Anderson et al. 1957). It was subsequently isolated from humans with undifferentiated febrile illness in northern Brazil and from humans, primates, and wild mosquitoes in Bolivia, Brazil, Colombia, Venezuela, and Peru (Torres et al. 2004, Auguste et al. 2015). Activity has also been detected in Central America (Muñoz and Navarro 2012) and Mexico (PAHO 2015). MAYV outbreaks of low magnitude have been reported to occur in the Pan-Amazonia forest (Muñoz and Navarro 2012, Vasconcelos and Calisher 2016). Transmission here may be by *Haemagogus* mosquitoes, which inhabit forested canopy (Hoch et al. 1981, Muñoz and Navarro 2012, Powers et al. 2006). An increase in travel and ecotourism-based forest excursions could enhance the risk of infection with MAYV and other forest viruses, and the possibility of spreading these viruses internationally (Terzian et al. 2014). In fact, in recent years, MAYV has been increasingly imported from South America to Europe by travel-related infections (Receveur et al. 2010, Slegers et al. 2014).

At the molecular level, two MAYV genotypes can be identified, genotypes D (of wide distribution) and L (of restricted distribution), which differ by more than 15% in terms of their nucleic acids (Powers et al. 2006). Human outbreaks in Brazil in 1978 and 1991 contributed to the epidemiological knowledge of this virus. Antibodies were detected in a high percentage in *Callithrix* monkeys, and viral isolations were made from *Haemagogus janthinomys* Dyar mosquitoes (Hoch et al. 1981). It was further found that MAYV in infected humans can infect and be transmitted by *Ae. aegypti*, raising the possibility of urban outbreaks (Long et al. 2011).

Demographic changes and human activities (urbanization, deforestation, intensive farming) that produce dramatic changes in the environment may facilitate the frequency of clinical cases of this virus (Muñoz and Navarro 2012, Assunção-Miranda et al. 2013). Although MAYV was reported in urban areas of Manaus, possibly associated to bites of sylvatic mosquitoes (Mourão et al. 2012), with factors related to infections analyzed (Abad-Franch et al. 2012), the

nonoccurrence of urban outbreaks may be owing to its difficulty of transmission from viremic humans to *Ae. aegypti* (Long et al. 2011) and other urban mosquitoes. High susceptibility and capacity of *Ae. albopictus* to transmit this virus in the laboratory (Smith and Francy, 1991) have not yet been confirmed in nature. Although *Cx. quinquefasciatus* was found infected by MAYV (Serra et al. 2016) and experimentally infected, no transmission was obtained (Aitken and Anderson 1959).

Zoonoses

Alphaviruses

Alphaviruses such as EEEV and VEEV are pathogenic for humans and equines and cluster within neurotropic viruses of New World origin, based on their original geographic distributions. MAYV, also of New World origin, is an exception, as it is associated with febrile illness and arthralgia (similar to dengue-like disease).

Eastern Equine Encephalitis Virus Complex

EEEV has been detected along the eastern coast, from Canada and the United States, through the Gulf of Mexico, Central America, and Caribbean islands, to South America (Go et al. 2014). EEEV is classified as the only species in the complex; originally, isolates were grouped into North and South American strains based on antigenic properties (Casals 1964). Later, following further antigenic and phylogenetic analysis, they were classified into four subtypes that correspond to four genetic lineages (I to IV). North American EEEV (NA EEEV) strains and most strains from the Caribbean comprise subtype/lineage I, whereas subtypes/lineages II to IV include South and Central American EEEV (SA EEEV) strains (Brault et al. 1999). Comparisons of EEEV strains from North and South America show molecular, epidemiological, and ecological differences. North American strains are associated with high virulence in humans and equines, whereas South American strains are pathogenic for equines but attenuated for humans (Brault et al. 1999, Young et al. 2008, Arrigo et al. 2010, Weaver et al. 2012). The results of Arrigo et al. (2010) support evolutionary and ecological diversification between NA and SA EEEV and suggest that NA and SA lineages should be considered independent species within the EEE complex.

In the United States, epizootics in horses and humans have occurred intermittently; however, during the past decade, increased activity and an expansion into new areas have been observed, with an increase of reported cases in the northeastern U.S. states (Molaei et al. 2015, Saxton-Shaw et al. 2015) and Canada (Rocheleau 2013). Likewise, simultaneous EEEV (lineage III) and VEEV outbreaks were reported in 2010 in Panama (Carrera et al. 2013); the EEEV strain here was later named as Madariaga virus (Vittor et al. 2016).

In South America, numerous epizootics by EEEV, some of them with thousands of cases, have been reported in Argentina, Brazil, Colombia, and Venezuela. Following almost 24 yr with no equine isolations, recent epizootics in horses were reported from Brazil in 2008-2009 (Silva et al. 2011, de Novaes Oliveira et al. 2014) and Panama (Carrera et al. 2013). An interesting discovery was the isolation of Madariaga virus from a child during an outbreak of alphavirus in Panama in 2010 (Luciani et al. 2015). This virus originally was isolated over 60 yr ago in Argentina (Sabattini et al. 1985) and until 2010 was not associated with human disease (Vittor et al. 2016). During equine epizootics in Argentina and Brazil, high seroprevalence was detected in the human population without clinical cases. To date, human cases were reported in Brazil (1956) and Trinidad (1970) (Scott and Weaver 1989, Sabattini et al. 1998b, de Novaes Oliveira et al. 2014).

In North America, EEEV is maintained in an enzootic cycle involving birds and ornithophilic mosquitoes, *Culiseta melanura* (Coquillett), in freshwater swamps (Molaei et al. 2016). Several mosquito species have been incriminated as potential bridge vectors across its geographic range (Armstrong and Andreadis 2010, Bingham et al. 2016, Molaei et al. 2015). In the Caribbean, Central America, and South America, viral transmission occurs enzootically and several mosquito species are involved as vectors (Turell et al. 2005b, Arrigo et al. 2010); however, the ecology of South American EEEV is poorly understood. Multiple isolations have been made from *Culex (Melanoconion)* spp. mosquitoes collected in Central and South America. Serological studies indicate that small mammals and birds serve as maintenance hosts. Moreover, bats, opossum, and reptiles previously infected with EEEV were detected serologically, although the role of these hosts (if any) in the transmission cycle is unknown (Arrigo et al. 2010). In the Amazon region, EEEV strains are maintained by birds and rodents (hosts) and the mosquitoes *Culex pedroi* Sirivanadarn & Belkin (enzootic vector; Turell et al. 2008) and *Aedes taeniorhynchus* (Wiedemann) (epizootic vector; Vasconcelos et al. 1991).

Venezuelan Equine Encephalitis Virus

Members of the VEEV complex cause periodic outbreaks of highly debilitating disease in the Americas. VEEV forms an integrated complex of six serological subtypes (I to VI actually are considered viral species, each with one to six variants), each differing in their epidemiological characteristics and pathogenicity for humans and equines (mainly horses; Steele and Twenhafel 2010, Weaver and Reisen 2010, Hubálek et al. 2014). VEEV subtypes are divided into two epidemiological groups: epidemic/epizootic viruses and enzootic viruses. Subtypes IAB and IC belong to the epidemic/epizootic group of viruses, which emerge periodically to cause outbreaks of severe illness in both horses and humans. Transmission cycles involve equines, which act as efficient amplification hosts, and mosquito vectors (Weaver et al. 2004). The case-fatality rate of VEEV is low in human infections (usually less than 1%), but infection is typically highly debilitating and sometimes results in permanent neurological sequelae (Johnson and Martin 1974, Quiroz et al. 2009). Outbreaks of VEE in humans and equids have been reported in several countries, including Venezuela, Colombia, Peru, Ecuador, Costa Rica, Nicaragua, Honduras, El Salvador, Guatemala, Panama, Mexico, and the United States. Ecologic and genetic factors are involved in the emergence of the epizootic subtypes. Molecular-level studies indicate that the epizootic strains (IAB and IC) arise from closely related enzootic VEE strains, particularly subtype-variety ID strains. Changes at the surface glycoprotein E2 level increase the virus' ability to infect the epidemic mosquito vector *Ae. taeniorhynchus* and to replicate in equids (Brault et al. 2004, Weaver and Reisen 2010, Aguilar et al. 2011). The remaining subtypes (ID, IE, IF, and II to VI; Table 1) are not virulent for equids, and are considered enzootic strains (Weaver et al. 2004). Enzootic vectors are species of the *Culex (Melanoconion)* subgenus, whereas the epizootic vectors involved are *Aedes* and *Psorophora* species; *Aedes sollicitans* (Walker) and *Ae. taeniorhynchus* exhibit high infection rates, depending on the region of the Americas, and the latter is probably the most important epizootic vector in South America (Weaver et al. 2004, Weaver and Reisen 2010, Aguilar et al. 2011).

Although infection by epidemic/epizootic strains has been observed in humans, sheep, dogs, bats, rodents, and some birds, no

major epidemics have been recorded in the absence of a concurrent equine outbreak. Humans develop substantial viremia titers following infection with both epizootic and enzootic strains (Weaver et al. 2004, Quiroz et al. 2009). However, they probably do not act as amplifying hosts owing to a low exposure to mosquito bites. Still, because of this viremia, transmission by humans to mosquitoes should not be discarded. Besides, considering that peridomestic mosquito vectors such as *Ae. aegypti* and *Ae. albopictus* are capable of transmission after infection with oral doses comparable with human viremia titers (Fernández et al. 2003, Ortiz et al. 2008), there is a potential risk of urban epidemics, as highlighted by Weaver and Reisen (2010). Enzootic subtypes complete their cycle in wild habitats, and involve mosquito vectors (*Culex* (*Melanoconion*) spp.) feeding on a variety of rodents (the most probable hosts), birds, and other vertebrates. However, isolations also have been made from *Aedes*, *Mansonia*, *Psorophora*, *Haemagogus*, *Sabethes*, *Deinocerites*, and *Anopheles* mosquitoes (Brault et al. 2004, Weaver and Reisen 2010, Aguilar et al. 2011). These enzootic viruses are attenuated and unable to amplify in equines, but most of them can cause illness in humans. Only subtype IE has been related to an encephalitis outbreak in horses in Mexico, but equines were unable to amplify the virus (Deardorff and Weaver 2010, Adams et al. 2012).

The enzootic subtypes active in South America include subtype ID, Mosso das Pedras (IF), Mucambo and Tonate (III), Cabassou (IV), Pixuna (V), and Rio Negro (VI); the latter very closely related phylogenetically with subtype IF (Aguilar et al. 2011). Subtype ID has caused outbreaks of DENV-like illness, with some cases of neurological disease in Bolivia and Peru (Aguilar et al. 2009, 2011). Even though febrile illness is known from subtype III, there is scarce documentation of this subtype (Aguilar et al. 2004, 2011).

In Brazil, the VEEV subtype IF circulates in the southeast and causes febrile illness and diarrhea in humans. It has also been isolated in the Amazon region (Calisher et al. 1982). In Argentina, activity of Rio Negro (subtype VI) virus, which was isolated from *Cx.* (*Mel.*) *delpontei* Duret and rodents of the genus *Akodon* in the subtropical area of Chaco and Formosa provinces, has been known for more than three decades. It is associated with the production of acute febrile disease but has been only recognized as a health problem in this country (Contigiani et al. 1993; Cámara et al. 2003; Pisano et al. 2012, 2013). Pixuna (subtype IV) virus, first isolated from *Anopheles nimbus* (Theobald) in 1961 in northern Brazil, also has been detected in Argentina (Pisano et al. 2010a,b). Enzootic circulation of multiple subtypes within a region (i.e., Argentina, Peru, and Venezuela) could result in the emergence of epizootic/epidemic strains. The interaction among strains and genetic mechanisms of adaptation to new hosts are postulated as the mechanisms for the emergence of epizootic strains (Aguilar et al. 2011, Pisano et al. 2014).

Zoonotic Flavivirus

Among the zoonotic flaviviruses, SLEV and Rocio viruses only circulate in the Americas, WNV was introduced almost two decades ago, and JEV remains restricted to Asia (Van der Hurk et al. 2009).

Saint Louis Encephalitis and West Nile Viruses

The neurotropic flaviviruses, SLEV and WNV, are of special concern owing to their wide distribution, potential disease severity, and general impact on human and animal health (Diaz et al. 2008a). SLEV historically was widely distributed in the Americas. In the United States, SLEV was one of the main causes of arbovirus encephalitis epidemics until the introduction of WNV in 1999 (Monath 1980, Reisen 2003), after which SLEV largely disappeared until a recent outbreak of human disease in Phoenix, AZ, in 2015 (Venkat et al. 2015). Historically, urban epidemics of encephalitis due to SLEV in other American countries have been rare, focal, or of small magnitude, or remain undetected (Spence 1980). This low incidence in Central and South America may be owing to attenuated viral strains and/or enzootic cycles involving mosquitoes that do not feed on humans frequently. In some regions, an inadequate case notification system and laboratory diagnostic capability may have precluded passive surveillance. Actually, it is a coincidence that the SLE cases in Argentina (South America) were diagnosed shortly after WNV introduction into North America. In fact, an increase in human encephalitis cases alerted authorities and led to the detection of SLEV. Research on arboviruses has been ongoing in Argentina since the mid-1960s.

Currently, SLEV is a reemerging arbovirus in South America and has been responsible for febrile disease and human encephalitis cases in Argentina in 2002, 2005, and 2010, and in Brazil in 2004 and 2006 (Spinsanti et al. 2003, 2008; Mondini et al. 2007; Rosa et al. 2013), with cases in horses reported in several Brazilian states (Pauvolid-Correa et al. 2010, Silva et al. 2014). The first human epidemic of SLEV outside of North America was reported in 2005, when 47 laboratory-confirmed clinical cases of SLE, including nine fatalities, were reported in Córdoba Province (Argentina) (Spinsanti et al. 2008).

SLEV ecology is well-characterized for the United States (Reisen 2003), whereas for the rest of the Americas, it remains practically unknown, except for Argentina, where research has provided advances in SLEV ecological characterization. In this region, *Cx. quinquefasciatus* is assumed to be the principal vector (Diaz et al. 2013a), and the main avian hosts are Picui Ground Doves (*Columbina picui*) (Sabattini et al. 1998a) and Eared Doves (*Zenaida auriculata*) (Diaz et al. 2008b), although antibody also has been found in other species (Quaglia et al. 2014, Beltran et al. 2015). Urban conditions for the proliferation of *Cx. quinquefasciatus* (Pires and Gleiser 2010, Gleiser and Zalazar 2010) and availability of amplifying hosts may explain recent increases in the occurrence of cases; however, this should be further assessed.

SLEV strains generally cluster according to their geographic origin and can be divided into eight genotypes (I to VIII; Kramer and Chandler 2001). Genotypes I and II circulate mainly in North America, genotypes III to VIII circulate mainly in Central and South America, and genotype V is also present in Florida. Humans and domestic mammals are excluded from the basic transmission cycle, because the viremia titers are insufficient to infect vector mosquitoes. SLEV epidemiology is driven by climatic, entomological, virus, and host factors, which form a complex network of interactions that still are not fully understood in South America (Diaz et al. 2013b).

WNV was first recognized as a human pathogen in 1937, when it was isolated from a febrile woman in Uganda (West Nile district) (Hayes 2001). Later, its activity was reported in Africa, Asia, Australia, Europe (Chancey et al. 2015), and North America since 1999, where it causes human and equine encephalitis outbreaks every year and became a causal factor for the decline of some wild bird populations (Roehrig 2013). By 2002, WNV reached Mexico and the Caribbean islands. The first report of WNV activity in the southern cone of South America dates from April 2006, but serological evidence from birds indicates that the virus was introduced and established in Argentina as early as January 2005 (Diaz et al. 2008a). Today, it is active in most countries of the Americas. Contrary to the United States, human encephalitis outbreaks by

WNV have not been reported in Latin American countries, although epizootic events in equines have been documented in Mexico (Estrada-Franco et al. 2003) and Argentina (Morales et al. 2006).

WNV is maintained through biological transmissions in which *Culex* spp. mosquitoes are involved as vectors and Passeriformes birds as hosts. The most comprehensive knowledge regarding the ecology of WNV is based on studies carried out in the United States, where most aspects of its transmission dynamics have been analyzed (Reisen 2013). The main vector species include *Cx. pipiens*, *Culex tarsalis* Coquillett, *Culex restuans* Theobald, *Cx. quinquefasciatus*, and *Culex nigripalpus* Theobald (Sardelis et al. 2001), whereas only a few non-*Culex* species have been considered potential vectors, such as *Ae. albopictus* and *Aedes vexans* (Turell et al. 2005a). Although preferably biting birds, mosquitoes of the *Cx. pipiens* complex may also bite mammals (Reisen 2013). *Culex nigripalpus* has eclectic host selection patterns in Brazil, biting rodents and birds (Laporta et al. 2008), and this mosquito and *Cx. quinquefasciatus* are potential vectors of WNV in Brazil. In North America, several bird species are reservoir and amplifying hosts, and crows and jays frequently die from the infection (Kilpatrick et al. 2007, Hubálek et al. 2014). In contrast, although serosurveys indicate widespread transmission in free-ranging birds, bird disease has not been evidenced in South America and the Caribbean (Diaz et al. 2008a, Komar and Clark 2006). Amplifying hosts are still unknown but likely candidates include passerine birds such as turdids (thrushes), common species of Furnariidae (a family absent from temperate North America; Diaz et al. 2008a), and Picui Ground Doves, which had the highest peak viremia titers of the longest duration [$10^{4.8}$ log PFU/mL serum (range $10^{2.9–6.2}$; 4–5 d duration)], with no mortality (Diaz et al. 2011). The successful experimental infection of alligators by WNV through several routes indicated a possible role of these animals as hosts of the virus (Klenk et al. 2004), with potential economic importance for commercial farms.

Rocio Virus

This flavivirus is classified as a genotype of Ilheus virus (ILHV; ICTV 2014), very near JEV (Coimbra et al. 2008), and was isolated in the east of the Brazilian state of São Paulo in the middle of the 1970s, when it caused hundreds of deaths and permanent neurologic sequelae (Iversson and Coimbra 1984). It has been found in several Brazilian states, was isolated from *Psorophora ferox* (von Humboldt) (Souza Lopes et al. 1981), and experimentally infected this mosquito and *Aedes scapularis* Rondani (Mitchell et al. 1986). North American *Cx. tarsalis* and *Cx. pipiens* were good experimental vectors but not *Ps. ferox* and *Cx. nigripalpus* (Mitchell et al. 1981). *Psorophora ferox* is widely distributed in the Americas and, as indicated by variations on egg morphology (Linley and Chadee 1990), may be a species complex, with possible regional differences in susceptibility to viruses. Several birds and mammals in the south and southeast of Brazil presented positive titers for this virus (Iversson and Coimbra 1984), but its epidemiology has been poorly studied, even though it is of considerable public health concern.

ILHV has been reported in Panama, Trinidad, Colombia, French Guiana, Brazil, Bolivia, Argentina, and Ecuador (Pauvolid-Corrêa et al. 2013). The infection may be asymptomatic or cause a DENV-like febrile syndrome, and although encephalitis has been infrequently reported, no encephalitis outbreaks have been attributed to ILHV (Sabattini et al. 1998a, Pauvolid-Corrêa et al. 2013). It was isolated from a pool of *Ae. scapularis* trying to bite humans in the Brazilian Pantanal region (Pauvolid-Corrêa et al. 2013).

Japanese Encephalitis Virus

This virus is widely distributed in Asia, where *Culex tritaeniorhynchus* Giles is the principal vector, and pigs and several birds serve as reservoirs. Autochthonous transmission has never been reported in the Americas, although several species of mosquitoes already associated with the transmission of flaviviruses and widely distributed in the Americas, like *Cx. quinquefasciatus*, *Ae. albopictus*, and *Ae. japonicus*, might be effective or secondary vectors (Reeves and Hammon 1946, Van der Hurk et al. 2009). JEV may have been dispersed throughout Southeast Asia during World War II, probably through transportation of infected *Cx. tritaeniorhynchus* by planes. The same mechanism has been proposed for the potential introduction into the United States, mostly in the Midwestern states, where intensive swine production would provide amplifying reservoirs (Van der Hurk et al. 2009).

Ardeidae birds are the most important reservoirs, among 90 species of wild and domestic birds already found infected. The rich fauna of Ardeidae (Bitterns, Herons, and Allies) and other birds in the Americas would offer multiple hosts for this virus. If JEV were introduced, it is likely to follow the path of WNV dispersal, creating additional difficulty for surveillance and differential diagnosis with SLEV and WNV (Nett et al. 2009). As all above ways of introduction would be viable for South America, *Cx. quinquefasciatus* and other *Culex (Culex)* occurring in this continent should be tested. Extensive breeding of pigs, mostly in the South of Brazil, would offer potential hosts for the virus.

Bunyaviridae

Orthobunyavirus

Orthobunyavirus is one of five genera of the family Bunyaviridae, consisting of 48 viral species with over 150 strains/isolates grouped in 19 serogoups (Plyusnin et al. 2012). The type species is Bunyamwera virus (BUNV), originally isolated from *Aedes* sp. in Uganda (Africa) in 1943 during a YFV outbreak (Smithburn et al. 1946). The identification of each virus within each genus is quite difficult because of limited molecular characterization (Schmaljohn and Nichol 2006). In tropical areas of South America, there are numerous isolates of this genus that cause human illness. Among them are Bunyanwera, Oropouche, Gamboa, Guaroa, and members of the group C viruses.

In the Americas, the first *Orthobunyavirus* isolated was Cache Valley virus (CVV), from *Culiseta inornata* Williston in the United States in 1956 (Holden and Hess 1959). Currently, according to the classification by the International Committee of Taxonomy of Viruses, CVV is considered a strain of BUNV (Plyusnin et al. 2012). Several strains have been recovered from different species of mosquitoes of the genera *Anopheles*, *Culex*, *Culiseta*, *Ochlerotatus*, and *Psorophora* in Argentina, Brazil, Colombia, Ecuador, Mexico, Panama, and the United States (Andreadis et al. 2014, Tauro et al. 2015).

BUNV is considered the causative agent of neural pathologies (encephalitis, meningitis, and febrile syndrome) and CNS defects in humans (Mangiafico et al. 1988, Sexton et al. 1997, Campbell et al. 2006). CVV was recovered from *Cs. inornata* in Argentina in 2009, and a fever case was confirmed as a BUNV infection by serology. Interestingly, this case was presumptively diagnosed clinically as DENV (Tauro et al. 2012). Finally, various seroepidemiological studies suggest that BUNV could be the etiologic agent of congenital malformations in humans, because a correlation was established between the occurrence of malformations in newborns and detection of NTAb against this virus in their mothers (Edwards et al. 1993).

In the United States, BUNV was found associated with the occurrence of febrile and neurologic disease in domestic and wild animals, including congenital malformations in domestic animals that were infected when pregnant (Edwards et al. 1993).

During the 1950s and 1960s, two strains were recovered from equine encephalitis cases in Guyana and Colombia; in Argentina, two new BUNV strains were isolated from equine encephalitis and abortion in 2013 (Spence and Downs 1968, Santamartin et al. 1973, Tauro et al. 2015). There are no vaccines or treatments to protect animals from BUNV infection. One of the possible solutions is to make reproductive crosses outside the period of greatest activity of vectors, thus reducing the risk of infection during pregnancy. However, in places where winter is moderate or there are unexpected weather changes, vector activity can be extended, increasing the risk of infection.

Oropouche Virus

Oropouche virus (OROV), another bunyavirus of public health impact in South America, was first isolated in Trinidad and Tobago in 1955, from the blood of a febrile patient and from a pool of *Coquillettidia venezuelensis* (Theobald) mosquitoes (Anderson et al. 1961). This virus was associated with several major epidemics of febrile illness in the Amazon region of Brazil and Peru and in Panama. The high prevalence of antibodies detected in inhabitants of forest and rural regions of Amazon region suggests an endemic circulation of this virus.

OROV has been isolated from various vertebrate sources (humans, monkeys, and edentates) and arthropods (mosquitoes *Cx. quinquefasciatus*, *Cq. venezuelensis, Aedes serratus* (Theobald), and biting midges, *Culicoides paraensis* (Goeldi)). Studies have shown that OROV is maintained in nature by two cycles: a sylvatic and an urban cycle. The sylvatic cycle is characterized by monkeys, sloths, and birds as potential hosts and *Ae. serratus*, *Cq. venezuelensis*, and *Culicoides* spp. as vectors (Figueiredo 1999). In urban settings, an alternative epidemic cycle can take place where humans generate a viremia high enough to infect *C. paraensis*. This arthropod is the principal urban vector involved in OROV epidemics (Pinheiro et al. 1981). Studies of the molecular biology of the *N* gene (SRNA) of 28 different OROV strains indicated the existence of three genotypes, designated I, II, and III (Saeed et al. 2000).

Why Are Arboviruses Now Spreading Through the New World?

Arboviral diseases may emerge owing to evolutionary changes in the virus resulting in novel virulent forms, which first appear in epidemic form. However, most emergence is more likely explained by increases in the geographic distribution of vectors and viruses and not by the evolution of new viruses (Monath 1993).

Among the general factors that may explain recent emergences or re-emergences of arboviral diseases in the New World are: 1) urban sprawl resulting from human population increase and movement from rural areas to expanding towns and cities that lack proper housing policy, water service, and sewage disposal systems (Moura et al. 2014); 2) increases in the volume and speed of national and international travel and commerce, where not only the vectors but also infectious (human) hosts may spread the pathogens (Aranda et al. 2006); and 3) landscape change that affects conditions for vector proliferation and may alter contact between vectors and reservoir hosts (Vasconcelos et al. 2001, Chastel 2012, Moura et al. 2014, Monath and Vasconcelos 2015). Intentional introduction of pathogens associated with arthropods ("arboterrorism") (Tabachnick et al. 2011) is another possible means for introductions that should not be disregarded.

The presence of an endemic virus may enhance or facilitate infection with a newly introduced virus. For example, the antibody-dependent enhancement (ADE) hypothesis (proposed to explain life-threatening complications that lead to DENV hemorrhagic fever) suggests that antibodies generated during an infection with DENV may drive greater ZIKV replication and increase the risk of complications following infection with ZIKV. Thus, the rapid spread of ZIKV throughout South America and the Caribbean and the emergence of more severe disease may be linked to the widespread previous (or concurrent) exposure to DENV (Dejnirattisai et al. 2016).

Besides vector transmission, modes of transmission by a number of arboviruses, including DENV, ZIKV, and WNV, include blood transfusion (Tambyah et al. 2008, Musso et al. 2014), organ transplantation (Iwamoto et al. 2003), and perinatal transmission (Besnard et al. 2014). Sexual transmission of ZIKV has been reported (Foy et al. 2011, D'Ortenzio et al. 2016). Laboratory accidents involving VEEV have been frequent, with >150 cases reported, and this virus has been developed as a biological weapon (Zacks and Paessler 2010). If a virus is introduced into a new region, it may cause a high enough viremia in local hosts, e.g., a horse in VEEV, to start an outbreak.

Urban Sprawl

The world population grew from 3,322 billion in 1965 to 7,350 billion in 2015, a 121% increase, and growth for South, Central, and North America was 105, 135, and 67%, respectively (https://ourworldindata.org/world-population-growth/). Concurrently, the proportion of humans living in urban centers increased to where as of 2012, more than half of the earth's population now lives in cities. This largely urban population growth occurred without a significant lowering of the Gini coefficient (a statistical measure of wealth dispersion intended to represent the income distribution of a nation's residents was proposed by the Italian statistician Corrado Gini in 1912 – https://en.wikipedia.org/wiki/Gini_coefficient). For example, in Brazil, this coefficient fluctuated from 0.59 in 1966 to 0.53 in 2010 (https://www.wider.unu.edu/project/wiid-%E2%80%93-world-income-inequality-database), even with gross domestic product (GDP) growth, illustrating that a great proportion of the human population survives in precarious conditions of inadequate housing and water supply, favoring the proliferation of mosquitoes and contact with human populations. Urban sprawl resulting from disorganized urbanization processes, especially in underdeveloped or developing countries, results in overall poor health conditions. Limited access to water service, sanitation, and sewage disposal systems generates conditions for mosquitoes to proliferate and leads to overall poor health conditions, including the increased transmission of DENV (Moura et al. 2014). This is especially true for areas with intermittent or no piped water supply that results in the storage of water for domestic use and leads to conditions suitable for the production of *Ae. aegypti*.

Increased Travel and Commerce

Increases in the volume and speed of national and international travel and the increased volume of international commerce mediate vector, host, and virus dispersal (Aranda et al. 2006). The spread of an arbovirus requires, first, vector invasion or availability of suitable native vectors, followed by transportation of the arbovirus per se, either by dispersal of the host or the vector (or intentional

introductions in the case of bioterrorism). For transmission to be established, availability of vertebrate host populations capable of producing viremias sufficient to infect local arthropod vectors and of adequate vectors at the destination is required, as well as suitable climatic conditions at the time of introduction. As an example, if birds or mosquitoes infected by WNV had entered in New York during the winter, introduction of the virus would be almost impossible. Frequently, the invasion by the vector has preceded the arrival of the virus, facilitating establishment.

Massive movements of humans for various reasons, including the slave trade from 1530 to 1850, may have introduced YFV and DENV into the New World. A canoeing competition was purportedly the source of ZIKV introduction into Brazil (Musso 2015), but a more plausible hypothesis would be the World Soccer Cup in 2014, with a great increase of travel from Asia, associated with the introduction of the Asian strain of the virus (Salvador and Fujita 2016). Zika probably followed the path of DENV and CHIKV in dispersal from Africa to Asia and then the New World (Musso et al. 2015a), and was associated with movements of humans and the availability of *Ae. aegypti*, previously established throughout the Americas after the collapse of the eradication program.

Dispersal of *Ae. aegypti* along roads is cited by Diaz-Nieto et al. (2013) as a likely explanation of their movement among densely populated cities separated by wetlands or farmlands in the southeast of Argentina, extending the known southern distribution limit for the species. Moreover, analysis of *Ae. aegypti* mitochondrial DNA haplotypes showed three haplotype groups (Rondan Dueñas et al. 2009). One group appears to be a relict population from the previous eradication campaigns of the 1970s, whereas the other two strongly matched populations lying along the main commercial routes connecting Brazil and Argentina on the east and Bolivia and Argentina on the northwest, stressing the importance of passive transportation as a major factor favoring *Ae. aegypti* dispersal in Argentina.

Propagule pressure for invasion depends on several factors: level/frequency of vehicle traffic, age of mosquitoes that are transported, and the length of travel on the vehicle that influences the chances of a passenger being bitten by an infected mosquito. The used tire trade is often referenced as a means of mosquito spread (see, for example, review by Eritja et al. 2005); however, transportation by vehicles is also possible. Adult and immature *Ae. aegypti* infestation rates of different vehicle types were assessed in the Peruvian Amazon, as an aid to predict *Ae. aegypti* range expansion and for DENV mitigation purposes. Vehicles surveyed included six different aquatic types, ranging from large barges with cargo holds in the bottom of the boat, that carry passengers and cargo throughout the Peruvian Amazon, to small water taxis that carry cargo and passengers locally in the Iquitos region, without cargo holds. The two terrestrial vehicle types surveyed were van-sized buses and taxis that travel along the major road out of Iquitos. Large barges were the vehicles most heavily and frequently infested with *Ae. aegypti*, whereas mosquitoes were not found in taxis and smaller water vehicles. Over 17 mosquito species were detected in all; among these, *Cx. quinquefasciatus* was even more common than *Ae. aegypti*. These two species were found as immature stages in containers and floor puddles in large barges; interestingly, a higher proportion of positive vehicles occurred during dates with less precipitation (Guagliardo et al. 2015a). These results indicate that riverboats are a significant source of *Ae. aegypti* spread, and that a few boats were responsible for most mosquito movement over long distances. Within large barges, a few oviposition sites were responsible for much of the larval production (Guagliardo et al. 2015b). Buses, on the other hand, had very low infestation rates in this region. In contrast, in Spain, cars and other land vehicles were suspected as a means of the recent dispersal of *Ae. albopictus* on the peninsula (Alarcón-Elbal et al. 2014).

The potential for the dispersal of container-associated mosquitoes, evident for *Ae. aegypti* and *Ae. albopictus*, has been confirmed for other vectors of the same genus. *Aedes notoscriptus* (Skuse), a vector of arboviruses and *Dirofilaria immitis*, highly prevalent during the dry season in roof gutters in Australia (Montgomery and Ritchie 2002) and in several containers (Derraik et al. 2008), including bromeliads, in New Zealand (Derraik 2009), was recently found in Los Angeles, with a high potential for expansion (Peterson and Campbell 2015). Other container-associated invasive species of *Aedes* of ecological and health concern are *Ae. japonicus* (Theobald), already showing widespread distribution, and *Aedes atropalpus* (Coquillett) (native to this continent but invasive in Europe; Medlock et al. 2015). The first has been proved to be an experimental vector of several arboviruses (Williges et al. 2008), including JEV, DENV, and CHIKV (Takashima and Rosen 1989, Schaffner et al. 2011), and the second as efficient as *Ae. triseriatus* for La Crosse virus (Freier and Beier 1984).

Availability of Hosts and Vectors

The Americas extend from 55° S to more than 69° N, an extreme situation that provides a huge diversity of climates, fauna, and vegetation, from tundra and glaciers to tropical forest, and from deserts to rainforests. The linkage of South America to Central America (plus North America) in the Pliocene promoted an exchange of fauna, more evident in mammals, with marsupials going northwards and most others (except probably monkeys and caviomorph rodents, which were transported by rafts from Africa; https://en.wikipedia.org/wiki/List_of_South_American_mammalssouthwards). This exchange certainly influenced the movement of arboviruses and mosquitoes.

The mosquito fauna is much diversified, with 862 species found in South America, 462 in continental Central America, and 192 in North America (mosquitocatalog.org) of 3,550 species in the world (http://mosquito-taxonomic-inventory.info/valid-species-list), emphasizing the greater diversity species in the first two subcontinents. This offers great opportunity for viruses to adapt to suitable vectors and reservoirs. In addition to the diversified mosquito fauna, the same pattern of diversity was seen for mammals, with 1,291, 651, and 467 species (IUCN 2016), and for birds, with 3,495, 1,591, and 2,194 species (http://avibase.bsc-eoc.org/checklist.jsp?region=index&lang=PT&list=clements) for South America, Central America, and North America, respectively, offering many potential reservoirs for arboviruses.

Besides *Ae. aegypti* and *Ae. albopictus* and the *Culex* mosquitoes, cited above, several additional mosquito species with vector potential occur in American countries from the United States to Argentina, and are available for transmission, if adequate epidemiological conditions occur. For example, floodwater *Aedes (Ochlerotatus)* species (*Ae. albifasciatus* (Macquart), *Ae. scapularis*, and *Ae. taeniorhynchus*) and *Ps. ferox* have been incriminated as vectors of Rocio and other arboviruses. *Aedes albifasciatus* can reach very high densities (Gleiser et al. 2000) and is a vector of the WEEV in South America (Mitchell et al. 1987, Avilés et al. 1992) and possibly Maguari virus (Sabattini et al. 1985). *Aedes scapularis* was experimentally infected by MAYV (Aitken and Anderson 1959); has been suspected as vector of YFV in Colombia, besides VEEV, Kairi, Melao, Cache Valley, etc. (Arnell 1976); and is

adapted to peridomiciliary environments (Forattini et al. 1995). *Aedes taeniorhynchus* is a vector of several arboviruses (Causey et al. 1962, Rivas et al. 1997, Coffey and Weaver 2005). From *Ps. ferox*, VEEV (Causey et al. 1961) and ROCV (Souza Lopes et al. 1981) were isolated in Brazil, ILHV and UNAV in Peru (Turell et al. 2005b), and EEEV (Cupp et al. 2004) and WNV (Granwehr et al. 2004) in the United States, which was not experimentally transmitted (Turell et al. 2005a). As most vectors vary geographically in regard to their susceptibility to infection and transmission, as shown for WNV within the *Culex pipiens* complex (Vaidyanathan and Scott 2007, Reisen et al. 2008, Micieli et al. 2013), local assessments should be carried out to confirm their involvement in arbovirus transmission.

Influence of Climate on the Distribution of Arboviruses in the New World

Mosquitoes are ectotherms, meaning that the regulation of their body temperature depends on external heat sources. Even though they may exhibit some degree of behavioral thermoregulation, their body temperature is essentially dependent on climate and ambient weather conditions. Therefore, the time interval between the acquisition of an infectious agent by a vector and the vector's ability to transmit to other susceptible vertebrate hosts, i.e., the pathogen's extrinsic incubation period (EIP), is temperature-dependent (Dohm et al. 2002, Patz et al. 2003, Tjaden et al. 2013). Therefore, climatic conditions and extreme weather events can exert direct effects on vectorborne disease transmission patterns (Patz et al. 2003, Anyamba et al. 2014). In fact, the potential effects of climate change on vectorborne disease distribution are a recurrent topic in the scientific literature (see, for example, reviews by Tabachnick 2010, Morin et al. 2013, Naish et al. 2014, Parham et al. 2015, Reisen 2015, Ebi and Nealon 2016). On the other hand, uncertainty is added to models considering mostly warming temperature effects, because exposure to cooler temperatures may increase susceptibility of mosquitoes to some virus infections. For example, an inverse relationship between temperature of extrinsic incubation and vector competence was first described for *Cx. tarsalis* infection with western equine encephalitis virus (Kramer et al. 1983), and later was experimentally shown for *Ae. aegypti* infection with CHIKV or YFV and *Ae. albopictus* infection with YFV, probably through destabilized RNA interference (Adelman et al. 2013). Mosquito responses to temperature may also vary depending on the range of diurnal temperature fluctuation: large temperature ranges increased immature development times and decreased adult survival and the number of gonotrophic cycles completed in *Ae. aegypti* compared with constant or small-range temperature fluctuations (Carrington et al. 2013).

Vezzani and Carbajo (2008 and references there) reviewed information related to *Aedes* vectors and DENV in Argentina since *Ae. aegypti* was reintroduced in 1986. Before the Panamerican eradication campaign in the 1950s, *Ae. aegypti* only was found in the north and center of the country (Bejarano 1979). Besides the expected seasonal variations in abundance in relation to temperature and precipitation, interannual variation was related to El Niño and La Niña events. Moreover, theoretical assessments about the effects of temperature and humidity saturation deficit on vector survival and the duration of gonotrophic cycles suggested an increasing favorability of meteorological conditions for *Ae. aegypti* during the second half of the past century, which may have favored the range expansion of *Ae. aegypti* in the Americas (Vezzani and Carbajo 2008, Carbajo et al. 2012). The southern range expansion of the mosquito may be partially explained by local populations adapting to winter conditions, as suggested by higher survival rates and lower development times at low temperatures (<14°C) of cohorts from Buenos Aires compared with other studies (De Majo et al. 2016). A recent analysis of oviposition activity in Buenos Aires city (near the southern continental distribution limit) between 1998 and 2014 showed steady increase in abundance in the past years but no significant changes in environmental and climatic conditions, further supporting the hypothesis of an ongoing adaptation to colder temperature conditions (Fischer et al. 2016).

Analysis of the association of monthly DENV incidence for the period 1985–2007 with climate variables in Mexico showed that weather significantly influenced DENV incidence in a nonlinear way (Colón-González et al. 2013). The models predicted an increase in DENV transmission in the majority of Mexican provinces under future climate change scenarios, with greater impact in endemic provinces and decreases in the north of the Yucatán Peninsula owing to reduced precipitation decreasing breeding sites. In contrast to studies suggesting that access to piped water should reduce DENV incidence by reducing potential larval habitats, lowest risk was found for people using rainwater harvesting as opposed to people with a piped water supply. The authors attributed this apparent contradiction to intermittent water delivery, making water storage necessary also for people reliant on piped water.

Brady et al. (2014) used an empirically parameterized mechanistic model that explicitly considered a temperature-dependent EIP and adult survival to elucidate the thermal limits of *Ae. aegypti* and *Ae. albopictus* persistence and DENV transmission. Introduction suitability was characterized as the number of days in a year where arrival of a DENV-infected human would lead to transmission in a totally uninfected population, and persistence suitability was the number of days in the year where onward DENV transmission could occur if a constant source of infectious humans were available (changes in host susceptibility owing to exposure to the virus were not considered in the model). Aside from socioeconomic community susceptibility and other non-climate-related issues, temperature was found to constrain the absolute extent of transmission and limit levels of DENV endemicity (Brady et al. 2014). Considerable differences are evidenced in the introduction and persistence suitability maps for both species, indicating that temperature limits both the absolute geographic limits and levels of DENV endemicity. Few environments were receptive to DENV introduction on every day of the year and many temperate environments, such as areas of North America and central Argentina, have a limited "window" for transmission, indicating that even if mosquitoes in these areas were frequently exposed to infectious humans, temperature would prevent year-round transmission and the area from becoming endemic. In agreement, limited seasonal outbreaks have been reported for these regions (Waterman et al. 2015). Historical accounts of DENV outbreaks at temperate latitudes coincide with unusual weather conditions that may have improved conditions for vectors and pathogen transmission. For example, in 1780, there was a bilious remitting fever outbreak historically attributed to DENV that occurred in the city of Philadelphia following an unusually hot summer with uncommonly numerous mosquitoes present during the autumn (Packard 2016) [also see Packard (2016) for a discussion about the limitations of retrospective epidemiology].

Nevertheless, as Brady et al. (2014) acknowledge in their temperature-centered analysis, DENV transmission is still rare by only *Ae. albopictus*, which may be explained by differences in bloodfeeding behavior and the degree of mosquito–human population overlap. Still, this species easily adapts to new environments, and thus, with increasing urbanization, these behavioral patterns could change and their vectorial role could become more relevant.

In fact, they have been identified as the vector for some outbreaks where *Ae. aegypti* was absent or with limited distribution, for example, the Seychelles islands in 1977–1978 (Metselaar et al. 1980) and the Hawaii epidemic of 2001–2002 (Effler et al. 2005). As a consequence of rapid changes in its overall distribution, there is growing evidence that *Ae. albopictus* is becoming more important in causing DENV outbreaks (Rezza 2012).

Campbell et al. (2015) used ecological niche modeling approaches (using MAXENT and WorldClim climate data archive) to assess the distributional potential of *Ae. aegypti* and *Ae. albopictus* at present and future climate conditions. Bias owing to uneven sampling effort for the genus was considered in the models. Their maps of potential distributions mostly replicate the actual known distributions and reflect that *Ae. aegypti* has a more extensive distribution than *Ae. albopictus*. Most likely owing to its diapausing eggs, *Ae. albopictus* is able to exploit more temperate areas in the northern hemisphere, whereas *Ae. aegypti* has a wider distribution at lower latitudes, perhaps because of its tolerance of low humidity. Interestingly, predicted distributions under climate change scenarios do not reflect major shifts in these vector species. Potential shifting predictions include broader establishment for both vector species in eastern North America and farther south in southern South America, particularly for *Ae. aegypti*. In the southern cone of South America, *Ae. albopictus* is expected to be restricted to Uruguay and the northeastern Argentina provinces, which is consistent with reports of the species only in Misiones province since 1998 (Rossi et al. 1999, Vezzani and Carbajo 2008).

Distribution models (mostly climate-based) show potentially suitable areas according to their environmental characteristics. However, anthropogenic infrastructure and transport may bias or enhance the potential spread of vectors in certain areas. Thomas et al. (2014) combined current and predicted climatic suitability maps for *Ae. albopictus* with transportation data to identify European areas at risk for the establishment of this vector. They used information about the amount of inbound cargo at top European harbors from countries where *Ae. albopictus* occurs and freight data for European railways and inland-waterways (tracking of the amount of general cargo being moved between European Regions; road transport data, although assumed of relevance, were excluded in this study). The analysis evidenced that transport by ship moved along corridors of high climatic suitability for disease vector establishment. Land transportation, on the other hand, bridges climatic suitable areas that otherwise may be segregated for vectors (and arbovirus) by efficient natural barriers, such as mountain ranges, lakes, or rivers.

When mapping high- and low-risk areas for targeting surveillance and control, it should be considered that besides climate, risk factors should also include socioeconomic factors (Reiter et al. 2003, Carbajo et al. 2012) that vary by season and lagged climate variables that vary by locality (Stewart Ibarra et al. 2013). Owing to (micro) habitat conditions offered by humans, demographic variables combined with climate may be more adequate to predict the occurrence of DENV (Carbajo et al. 2012) and other viruses borne by synanthropic mosquitoes. Lifestyle conditions such as restricting activities to fully air-conditioned buildings may restrict contact with vectors in otherwise adequate environments (Reiter et al. 2003).

What Can Be Done to Prevent Dispersal and Consequences of Arbovirus Invasion?

Since Walter Reed and collaborators successfully proved Finlay's "mosquito hypothesis" of YFV transmission, the most frequently used approach to control mosquito-borne disease has focused on vector control in areas with high transmission risk or disease incidence levels. However, broad experience has shown that any control method should be part of a broader integrated vector control program, as reviewed by Maciel-de-Freitas et al. (2012). For most viruses that are not restricted to humans, control rather than eradication is more likely, because elimination of the enzootic cycle is extremely difficult. In the United States, most intervention programs for arboviruses such as WNV attempt to suppress vector populations below levels where spillover transmission to humans remains infrequent. In Puerto Rico, nonhuman primates have serologically tested positive for DENV; however, it is not clear whether this shows enzootic transmission or virus spillover from humans to monkeys (Hemme et al. 2016). If a stable enzootic cycle exists between mosquitoes and other mammals such as nonhuman primates, the elimination of the virus may be improbable and recurrent emergence expected.

A stochastic spatial population dynamics model of *Ae. aegypti* based on the life cycle of the mosquito and its dispersal was developed for Buenos Aires (Otero et al. 2008). The main conclusions regarding vector eradication were that in large cities in temperate climates, focusing exclusively on potential refugia (such as cemeteries) would not be efficient, because persistence of mosquito populations relies on a recurrent dispersal–extinction cycle. In addition, control/eradication campaigns should be performed during the winter, when only immature stages are present, which cannot disperse. This has rarely been done, owing to the focus on lowering of the incidence of disease and the attention of the population and media. The frequent practice of temporarily contracting programs during winter and training workers during outbreaks is inefficient, wastes resources, and frequently results in the onset of control after most humans are infected. Also, small tree holes and other natural water containers used as larval habitats may be overlooked by control personnel and become reinfestation foci (Mangudo et al. 2015).

Because infection with DENV, CHIKV, MAYV, and ZIKV produces very similar clinical symptoms and the severity of disease may vary, differential laboratory diagnosis is recommended for all cases. However, mitigation actions focusing on vector control would be similar because of the shared involvement of *Ae. aegypti* as the principal vector.

For YFV, vaccination was very important for the control of the disease in Africa, whereas mosquito control was more important in the Americas (Barrett and Higgs 2007). Vaccination continues to be fundamental for the protection of travelers entering high-risk areas and must take place before traveling. Vaccines against YFV should be utilized not only in populations inhabiting endemic areas, like the Amazon forest, but also in those populations exposed to sylvatic vectors and/or infested by *Ae. aegypti* in the east of South America (Butler 2016). Shortage of YFV vaccine, produced by only four major manufacturers, recently has caused difficulties in the control of the disease in Angola, Congo, and Uganda in 2016 (http://www.eturbonews.com/72199/world-health-organization-global-stockpile-yellow-fever-vaccine-). Six million doses were administered in 2016, leaving manufacturers to recover the international stock of 6.2 million doses. The Brazilian Government currently is analyzing the possibility to reduce the individual dose from 0.5 to 0.1 ml (as was done recently in Africa, to provide 5X doses while providing similar protection; Woodall and Yuill 2016). Although the duration of the protection must be investigated, the immediate result of protection of populations and prevention of deaths is most important. The possibility of expansion of YFV from Africa to Asia, following the path of ZIKV and CHIKV (Wassermann et al. 2016), may aggravate the

scarcity of vaccines. Still, the incidence of severe neurotropic collateral effects in 1/200,000–400,000 applications and in 1/50,000 in persons above 60 yr of age (Monath 2016) makes use of the 17D vaccination not without risk in large campaigns.

Production of efficient vaccines for DENV has been very difficult owing to the circulation of four serotypes; a fifth one recently identified in southeastern Asia (Mustafa et al. 2014) will probably be soon circulated in other areas, emphasizing the need of integrated control. The absence of an animal model, differential reaction to serotypes, and the incomplete understanding of which "immune profile" will induce protection or disease are important limitations to consider for vaccine development (Thomas 2011). Tests will need long follow-up or Phase-4 studies to evaluate their efficacy versus the risk of producing serious disease. Test of a vaccine produced by Sanofi-Pasteur, in Asia (Capeding et al. 2014) and Latin America (Villar et al. 2014), presented some positive results in Phase-3 tests for children, but as applications at 3, 6, and 12 months were necessary, utility is cumbersome and not all children received all doses. An unexplained higher incidence of hospitalization for DENV in Year 3 for children younger than 9 yr (Hadinegoro et al. 2015) and the risk of ADE, frequent in DENV, were cited as limitations (Russell and Halstead 2016), and the possibility of enhancement of ZIKV transmission was suspected for CYD-TDV (Dengvaxia, Sanofi Pasteur) in the first DENV vaccine approved by the WHO (http://www.cidrap.umn.edu/news-perspective/2016/07/contrary-DENV-vaccine-response-hints-possible-problems-zika).

Although 10% of cases of YFV are serious, with a high risk of death (Vasconcelos 2003), prevention of disease and transmission by vaccination is the best measure to protect the population in outbreaks and for exposure to infection in endemic areas, like the jungles of South America. Vaccines against other viruses are necessary and must be produced, but even if vaccines were available for the major vectorborne diseases, vector surveillance and control should not be disregarded because of the risk of other novel or introduced viruses.

Although previous infection of hamsters by JEV and DENV protects the animals against infection by YFV (Xiao et al. 2003), and this may have some influence in the nonoccurrence of YFV in Asia, the risk of introduction of YFV into Asia cannot be disregarded (Wassermann et al. 2016). Immunologically mediated interference between DENV and YFV deserves more studies, as immigrants from India (likely DENV-immune) were protected from YFV disease (Monath 1989). For the Americas, where the JEV was never found and the incidence of DENV is not as high as in Asia, the current populations would probably not be protected against YFV.

For the above *Ae. aegypti*-associated arboviruses, the most important approach for control is the extreme reduction of vector populations and the protection against bites. This can be obtained by the improvement of water supply, preventing the utilization of storage vessels, checking and elimination of potential larval habitats, public health education, utilization of repellents, and adequate protective clothes and screening houses. Utilization of bednets, at least for babies and infected people, would be useful, but the predominantly diurnal biting habit of *Ae. aegypti* should be considered. The adaptation of *Ae. aegypti* to cesspits (Burke et al. 2010, Gil et al. 2015) may result from strong selection pressure resulting from container removal, and therefore, surveillance is paramount. In fact, failure of interventions targeting containers above the ground and adulticing led to the discovery of unsealed septic tanks and other cryptic underground larval habitats that were more productive for *Ae. aegypti* throughout the year, independent of rainfall, and produced larger adults (Mackay et al 2009). Based on the bioclimatic conditions related to urbanization, besides artificial or man-made larval habitats, landscaping may create natural containers such as phytotelmata (Mangudo et al. 2012) that are not easily identified and accessed for mosquito control and whose removal may be limited. Therefore, a more integrated mosquito management approach combining other control means besides container removal may be necessary.

Novel Methods of Mosquito Control

Because the major vectors of DENV and malaria, except *Ae. albopictus*, are not naturally infected by *Wolbachia*, whereas many mosquito species that are infected are poor vectors (Maciel-de-Freitas et al. 2012), this suggests that the presence of certain *Wolbachia* symbionts may play a role in pathogen interference (as has been shown in laboratory assays). Thus, there is a global initiative for introducing *Wolbachia* infections into *Ae. aegypti* as a biological control approach for the interruption of DENV transmission (Maciel-de-Freitas et al. 2012). This bacterium has also blocked transmission of ZIKV by *Ae. aegypti* (Dutra et al. 2016). The Release of Insects carrying Dominant Lethal genes (RIDL), involving the liberation of males with a gene preventing development, has shown some potential, but the method is limited by nonperpetuation in wild populations (Yakob and Walker 2016) and several potential risks, including the survival of 3.5% of descendants of sterilized males and the failed selection of only males, mixed with 0.5% of females, for release (Pollack 2011). Even though changes in susceptibility to infection and/or increases of vectorial capacity for other pathogens are unknown, this should be examined in this novel and artificially induced bacteria–mosquito association. It would be advisable to develop more complete and long-term tests on these modified insects to check potential risks associated with their release and establishment. It also should be mentioned that population replacement will not reduce mosquito biting nuisance and affected human populations may demand control, thereby creating cross purposes of releases.

Females of some mosquito species such as *Ae. aegypti* show a "skip-oviposition" behavior, whereby females lay a few eggs in each of several oviposition sites. A novel tactic that is showing promising population reduction takes advantage of this behavior and relies on the females to disseminate tiny particles of larvicides (such as pyriproxyfen) from "dissemination stations" treated with larvicide dust-particles to larval habitats that may not be detected by control personnel, thus improving coverage (Abad-Franch et al. 2015).

For sewage-associated mosquitoes such as the *Cx. pipiens* complex, improvement of sewage and wastewater transport systems and personal protection, mostly by bed nets, are very important. For other vectors, additional measures must be chosen and adopted, and it is difficult to recommend a general scheme of control, owing to the diversity of vectors and conditions.

Concluding Remarks

The rapid spread of anthroponotic arboviruses DENV, CHIKV, and ZIKV has been facilitated by the thriving populations of *Ae. aegypti* in most neotropical urbanizations that allowed local transmission to be established. For these introduced viruses that circulate between humans and mosquitoes, it is expected that the frequency and scope of epidemics will eventually decrease owing to herd immunity in humans (Petersen and Powers 2016). Still, if no adequate measures are taken to control the mosquito population, the stage is set for cases to continue occurring and to facilitate the introductions of other pathogens.

The recent emergence of multiple arboviruses from sylvatic cycles and introductions of exotic viruses new to the Americas emphasize the importance of establishing or expanding regional surveillance, research, and control programs. Studies of the mosquito fauna and its natural infection with arboviruses in regions utilized by humans must be developed. No mosquito species may be considered as not important, even if not having (already) been incriminated as a vector of some arbovirus. As an example, VEEV IC emerged from subtype ID after a small mutation adapted this virus to equines, humans, and vectors besides the usual *Culex (Melanoconion)*, causing the widespread epidemics of 1969–1972 extending from Colombia to the United States and causing thousands of deaths of humans and equines (Brault et al. 2004). At least 1,300 human cases occurred in Colombia and Venezuela in 1995, with clinical cases in 11–20% of exposed populations (Meslin 1997).

The isolation of 187 species of arboviruses by Evandro Chagas Institute from 1954 to 1988 (Vasconcelos et al. 2001) indicates the great diversity of possible health problems and the volume of work needed. Vigilance of natural infection of birds (e.g., out of the United States; Diaz et al. 2008a, b) and mammals must be part of routine surveillance activities, to evaluate the risk of transmission by local mosquitoes. The recent survey in São José do Rio Preto in the Brazilian state of São Paulo found 34 and 46 species of mosquitoes in urban areas and forest patches, respectively (approximately 70 species in total; Dibo et al. 2012), is an example of such studies. If possible, these collections should be associated with testing for infection by arboviruses.

As shown for Rocio virus, initially found in the south of the Brazilian state of São Paulo, and afterwards in several other states, the distribution of an arbovirus is not only related to range expansion but also knowledge of the existing range. Often for arboviruses, perceived increases in distribution actually chronically improved surveillance and diagnosis. For the distribution of arboviruses, if no research was done in a locality, the observation of Berkeley (1710), "If a tree falls in a forest and no one is around to hear it, does it make a sound?," must be remembered. The knowledge of taxonomy of mosquitoes, mostly of the large and difficult *Culex (Melanoconion)* subgenus, must be improved, if necessary utilizing molecular techniques, for a better understanding of epidemiology and to obtain an efficient control.

Vigilance for arbovirus surveillance and control of vectors must be carefully maintained. Some instances of neglect in the United States, such as the abandonment of control of *Ae. aegypti* in the 1950s (Soper 1963), and the "dwindling public health funding" limiting state and local capacity to react to the introduction of WNV in New York in 1999 (Roehrig 2013) indicate that these problems are not exclusive to less developed countries. Arboterrorism (Tabachnick et al. 2011) is an additional reason for the organization of an efficient network of laboratories and control districts.

Acknowledgments

We thank Dr. William Reisen (UC, Davis) for useful comments, Drs. Pedro Tauil (UnB, Brasília) and Andrew van den Hurk (Dept of Health, Queensland) for information on arboviruses, and Dr. Mauricio Graipel (LAMAQ/UFSC, Florianópolis) for the information on numbers of species of mammals and birds in the Americas. We also acknowledge two unknown referees for several useful comments on manuscript and Helio Schwartsman (Folha de São Paulo, São Paulo) for the indication of G. Berkeley's comment.

References Cited

Abad-Franch, F., G. H. Grimmer, V. S. Paula, L. T. Figueiredo, W. S. Braga, and S. L. Luz. 2012. Mayaro virus infection in Amazonia: A multimodel inference approach to risk factor assessment. PLoS Negl. Trop. Dis. 6: e1846. doi: 10.1371/journal.pntd.0001846

Abad-Franch, F., E. Zamora-Perea, G. Ferraz, S. D. Padilla-Torres, and S.L.B. Luz. 2015. Mosquito- disseminated Pyriproxyfen yields high breeding-site coverage and boosts juvenile mosquito mortality at the neighborhood scale. PLoS Negl Trop Dis. 9: e0003702. doi:10.1371/journal.pntd.0003702.

Adams, A. P., R. Navarro-Lopez, F. J. Ramirez-Aguilar, I. Lopez-Gonzalez, G. Leal, J. M. Flores-Mayorga, A. P. Travassos da Rosa, K. D. Saxton-Shaw, A. J. Singh, E. M. Borland, et al. 2012. Venezuelan equine encephalitis virus activity in the Gulf Coast region of Mexico, 2003-2010. PLoS Negl. Trop. Dis. 6: e1875. doi: 10.1371/journal.pntd.0001875.

Adelman, Z. N., M.A.E. Anderson, M. R. Wiley, M. G. Murreddu, G. H. Samuel, E. M. Norazzi, and K. M. Myles. 2013. Cooler temperatures destabilize RNA interference and increase susceptibility of disease vector mosquitoes to viral infection. PLoS Negl. Trop. Dis. 7: e2239. doi:10.1371/journal.pntd.0002239.

Aguilar, P. V., I. P. Greene, L. L. Coffey, G. Medina, A. C. Moncayo, M. Anishchenko, G. V. Ludwig, M. J. Turell, M. L. O'Guinn, J. Lee, et al. 2004. Endemic Venezuelan equine encephalitis in northern Peru. Emerg. Infect. Dis. 10: 880–888.

Aguilar, P. V., A. P. Adams, V. Suárez, L. Beingolea, J. Vargas, S. Manock, J. Freire, W. R. Espinoza, V. Felices, A. Diaz, et al. 2009. Genetic characterization of Venezuelan equine encephalitis virus from Bolivia, Ecuador and Peru: Identification of a new subtype ID lineage. PLoS Negl. Trop. Dis. 3: e514.

Aguilar, P. V., J. G. Estrada Franco, R. Navarro-Lopez, C. Ferro, A. D. Haddow, and S. C. Weaver. 2011. Endemic Venezuelan equine encephalitis in the Americas: Hidden under the dengue umbrella. Future Virol. 6: 721–740.

Aitken, T. H., and C. R. Anderson. 1959. Virus transmission studies with Trinidadian mosquitoes II. Further observations. Am. J. Trop. Med. Hyg. 8: 41–45.

Alarcón-Elbal, P. M., S. D. Estrella, I. R. Arrondo, F. Collantes, J. A. D. Iniesta, J. Morales-Bueno, P. F. Sánchez-López, C. Amela, M. J. Sierra-Moros, R. Molina, et al. 2014. Updated distribution of *Aedes albopictus* (Diptera: Culicidae) in Spain: New findings in the mainland Spanish Levante. Mem. Inst. Oswaldo Cruz 109: 782–786.

Anderson, C. R., W. G. Downs, G. H. Wattley, N. W. Ahin, and A. A. Reese 1957. Mayaro virus: a new human disease agent. II. Isolation from the blood of patients in Trinidad, B. W. I. Am. J. Trop. Med. 6: 1012–1016.

Anderson, C. R., L. P. Spence, W. G. Downs, and T.H.G. Aitken. 1961. Oropouche virus: A new human disease agent from Trinidad, West Indies. Am. J. Trop. Med. Hyg. 10: 574–578.

Andreadis, T. G., P. M. Armstrong, J. F. Anderson, and A. J. Main. 2014. Spatial-temporal analysis of Cache Valley virus (Bunyaviridae: Orthobunyavirus) infection in anopheline and culicine mosquitoes (Diptera: Culicidae) in the northeastern United States, 1997-2012. Vector Borne Zoonotic Dis. 14: 763–773. doi: 10.1089/vbz.2014.1669.

Anyamba, A., J. L. Small, S. C. Britch, C. J. Tucker, E. W. Pak, C. A. Reynolds1, J. Crutchfield, and K. J. Linthicum. 2014. Recent weather extremes and impacts on agricultural production and vector-borne disease outbreak patterns. PLoS ONE 9: e92538. doi:10.1371/journal.pone.0092538.

Apandi, Y., W. A. Nazni, Z. A. Noor Azleen, I. Vythilingam, M. Y. Noorazian, A. H. Azahari, S. Zainah, and H. L. Lee. 2009. The first isolation of chikungunya virus from non-human primates in Malaysia. J. Gen. Mol. Virol. 1: 35–39.

Aranda, C., R. Eritja, and D. Roiz. 2006. First record and establishment of the mosquito *Aedes albopictus* in Spain. Med. Vet. Entomol. 20: 150–152.

Arbovirus Catalog International Catalog of Arboviruses. 2016. (https://wwwn.cdc.gov/arbocat/VirusBrowser.aspx) (accessed 25 May 2016).

Armstrong, P. M., and T. G. Andreadis. 2010. Eastern equine encephalitis virus in mosquitoes and their role as bridge vectors. Emerg. Infect. Dis. 16: 1869–1874.

Arnell, J. H. 1976. Mosquito studies (Diptera, Culicidae). XXXIII. A revision of the Scapularis group of *Aedes (Ochlerotatus)*. Control Am. Entomol. Inst. 13: 1–144.

Arrigo, C., A. P. Adams, and S. C. Weaver. 2010. Evolutionary patterns of eastern equine encephalitis virus in North versus South America suggest ecological differences and taxonomic revision. J. Virol. 84: 1014–1025.

Assunção-Miranda, I., C. Cruz-Oliveira, and A. T. Da Poian. 2013. Molecular mechanisms involved in the pathogenesis of Alphavirus-induced arthritis. BioMed Res. Intern. 11 p. http://dx.doi.org/10.1155/2013/973516.

Auguste, A. J., J. Liria, N. L. Forrester, D. Giambalvo, M. Moncada, K. C. Long, D. Morón, N. de Manzione, R. B. Tesh, E. S. Halsey, et al. 2015. Evolutionary and ecological characterization of Mayaro Virus strains isolated during an outbreak, Venezuela, 2010. Emerg. Infect. Dis. 21: 1742–1750. doi: 10.3201/eid2110.141660.

Avilés, G., M. S. Sabattini, and C. J. Mitchell. 1992. Transmission of Western equine encephalomyelitis virus by Argentine *Aedes albifasciatus* (Diptera: Culicidae). J. Med. Entomol. 29: 850–853.

Ayres, C.F.J. 2016. Identification of Zika virus vectors and implications for control. Lancet Inf. Dis. 16: 278–279.

Barbosa, J. P. 1929. Pequena história da febre amarela no Brasil. [A short history of yellow fever in Brazil]. Arq. Hyg. 3: 329–338. [in Portuguese].

Barrett, A.T.D., and S. Higgs. 2007. Yellow fever: A disease that has yet to be conquered. Annu. Rev. Entomol. 52: 209–229.

Bejarano, J.F.R. 1979. Estudio sobre fiebre amarilla selvática en la República Argentina. Ministerio de Bienestar Social de la Nacion, Secretaria de Estado de Salud Pública, República Argentina. *apud* Carbajo, A. E., N. Schweigmann, S. I. Curto, A. de Garín, and R. Bejarano. 2001. Dengue transmission risk maps of Argentina. Trop. Med. Intern. Health 6: 170–183.

Beltran, F. J., L. A. Diaz, B. Konigheim, J. Molina, J. B. Beaudoin, M. Contigiani, and L. I. Spinsanti. 2015. Evidencia serológica de circulación del virus de la encefalitis de San Luis en aves de la Ciudad Autónoma de Buenos Aires, Argentina. [Serological evidence of St. Louis encephalitis virus circulation in birds from Buenos Aires City, Argentina]. Rev. Argent. Microbiol. 47: 312–316. [in Spanish].

Berkeley, G. 1710. A treatise concerning the principles of human knowledge. Akron Rhames, Dublin.

Berthet, N., E. Nakouné, B. Kamgang, B. Selekon, S. Descorps-Declère, A. Gessain, J. C. Manuguerra, and M. Kazanji. 2014. Molecular characterization of three Zika flaviviruses obtained from sylvatic mosquitoes in the Central African Republic. Vector Borne Zoonotic Dis. 14: 862–865.

Besnard, M., S. Lastère, A. Teissier, V. M. Cao-Lormeau, and D. Musso. 2014. Evidence of perinatal transmission of Zika virus, French Polynesia, December 2013 and February 2014. Euro Surveill. 19: pii=20751. (http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20751)

Bingham, A. M., N. D. Burkett-Cadena, H. K. Hassan, and T. R. Unnasch. 2016. Vector competence and capacity of *Culex erraticus* (Diptera: Culicidae) for Eastern Equine Encephalitis virus in the southeastern United States. J. Med. Entomol. 53: 473–476.

Black, W. C. IV, K. E. Bennett, N. Gorrochótegui-Escalante, C. V. Barillas-Mury, I. Fernández-Salas, M. L. Muñoz, J. A. Farfán-Alé, K. E. Olson, and B. J. Beaty. 2002. Flavivirus susceptibility in *Aedes aegypti*. Arch. Med. Res. 33: 379–388.

Bonaldo, M. C., I. P. Ribeiro, N. S. Lima, A.A.C. dos Santos, L.S.R. Menezes, S.O.D. da Cruz, I. S. de Mello, N. D. Furtado, E. E. de Moura, L. Damasceno, et al. 2016. Isolation of infective Zika virus from urine and saliva of patients in Brazil. PLoS Negl. Trop. Dis. 10: e0004816. doi:10.1371/journal.pntd.0004816.

Bordi, L., F. Carletti, C. Castilletti, R. Chiappini, V. Sambri, F. Cavrini, G. Ippolito, A. Di Caro, and M. R. Capobianchi. 2008. Presence of the A226V mutation in autochthonous and imported Italian Chikungunya virus strains. Clin. Infect. Dis. 47: 428–429.

Brady, O. J., N. Golding, D. M. Pigott, M. U. C. Kraemer, J. P. Messina, R. C. Reiner, Jr., T. W. Scott, D. L. Smith, P. W. Gething, and S. I. Hay. 2014. Global temperature constraints on *Aedes aegypti* and *Ae. albopictus* persistence and competence for DENV virus transmission. Parasit. Vector 7: 338.

Brault, A. C., A. M. Powers, C. L. Chavez, R. N. Lopez, M. F. Cachón, L. F. Gutierrez, W. Kang, R. B. Tesh, R. E. Shope, and S. C. Weaver. 1999. Genetic and antigenic diversity among eastern equine encephalitis viruses from North, Central, and South America. Am. J. Trop. Med. Hyg. 61: 579–586.

Brault, A. C., A. M. Powers, D. Ortiz, J. G. Estrada-Franco, R. Navarro-Lopez, and S. C. Weaver. 2004. Venezuelan equine encephalitis emergence: Enhanced vector infection from a single amino acid substitution in the envelope glycoprotein. Proc. Natl. Acad. Sci. USA 101: 11344–11349.

Brès, P.J.L. 1986. A century of progress in combating yellow fever. Bull. WHO 64: 775–786.

Brustolin, M., S. Talavera, C. Santamaría, R. Rivas, N. Pujo, L. C. Aranda, E. Marquès, M. Valle, M. Verdún, N. Pagès, et al. 2016. *Culex pipiens* and *Stegomyia albopicta* (=*Aedes albopictus*) populations as vectors for lineage 1 and 2 West Nile virus in Europe. Med. Vet. Entomol. 30: 166–173.

Burke, R., R. Barrera, M. Lewis, T. Kluchinsky, and D. Clarborn. 2010. Septic tanks as larval habitats for the mosquitoes *Aedes aegypti* and *Culex quinquefasciatus* in Playa-Payita, Puerto Rico. Med. Vet. Entomol. 24: 117–123. doi: 10.1111/j.1365-2915.2010.00864.x

Butler, D. 2016. Fears rise over yellow fever's next move. Nature. 532: 155–156.

Calisher, C. H., K.S.C. Maness, R. D. Lord, and P. H. Coleman. 1971. Identification of two South American strains of eastern equine encephalomyelitis virus from migrant birds captured on the Mississippi delta. Am. J. Epidemol. 94: 172–178.

Calisher, C. H., R. M. Kinney, O. de Souza Lopes, D. W. Trent, T. P. Monath, and D. B. Francy. 1982. Identification of a new Venezuelan equine encephalitis virus from Brazil. Am. J. Trop. Med. Hyg. 31: 1260–1272.

Cámara, A., G. Díaz, V. Vega, M. Basualdo, and M. Contigiani. 2003. Seroprevalence of antibodies to Venezuelan equine encephalitis complex (subtypes IAB and VI) in humans from General Belgrano Island, Formosa, Argentina. Rev. Inst. Med. Trop. S. Paulo 45: 201–204.

Campbell, G. L., J. D. Mataczynski, E. S. Reisdorf, J. W. Powell, D. A. Martin, A. J. Lambert, T. E. Haupt, J. P. Davis, and R. S. Lanciotti. 2006. Second human case of Cache Valley Virus disease. Emerg. Inf. Dis. 12: 854–856.

Campbell, L. P., C. Luther, D. Moo-Llanes, J. M. Ramsey, R. Danis-Lozano, and A. T. Peterson. 2015. Climate change influences on global distributions of dengue and chikungunya virus vectors. Phil. Trans. R. Soc. B. 370: 20140135. http://dx.doi.org/10.1098/rstb.2014.0135.

Capeding, M. R., N. H. Tran, S.R.S. Hadinegoro, H.I.H.J.M. Ismail, T. Chotpitayasunondh, M. N. Chua, C. Q. Luong, K. Rusmil, D. N. Wirawan, R. Nallusamy, et al. 2014. Clinical efficacy and safety of a novel tetravalent dengue vaccine in healthy children in Asia: A phase 3, randomised, observer-masked, placebo-controlled trial. Lancet 384: 1358–1365.

Carbajo, A. E., S. I. Curto, and N. J. Schweigmann. 2006. Spatial distribution pattern of oviposition in the mosquito *Aedes aegypti* in relation to urbanization in Buenos Aires: Southern fringe bionomics of an introduced vector. Med. Vet. Entomol. 20: 209–218.

Carbajo, A. E., M. V. Cardo, and D. Vezzani. 2012. Is temperature the main cause of dengue rise in non-endemic countries? The case of Argentina. Int. J. Health Geogr. 11: 26.

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

Carrington, L.B., S. N. Seifert, N. H. Willits, L. Lambrechts, and T. W. Scott. 2013. Large diurnal temperature fluctuations negatively influence Aedes aegypti (Diptera: Culicidae) life-history traits. J. Med. Entomol. 50: 43-51.

Casals, J. 1964. Antigenic variants of eastern equine encephalitis virus. J. Exp. Med. 119: 547–565.

Causey, O. R., C. E. Causey, O. M. Maroja, and D. G. Macedo. 1961. The isolation of arthropod-borne viruses, including members of two hitherto undescribed serological groups, in the Amazon region of Brazil. J. Trop. Med. Hyg. 10: 227–249.

Causey, O. R., R. F. Shope, P. Sutmoller, and H. Laemmert. 1962. Epizootic Eastern Equine Encephalitis in the Bragança region of Para Brazil. Bol. Fund. SESP 12: 39–45.

Chancey, C., A. Grinev, E. Volkova, and M. Rios. 2015. The global ecology and epidemiology of West Nile Virus. Biomed Res. Int. 2015: http://dx.doi.org/10.1155/2015/376230, in press.

Charrell, R. M., X. de Lamballerie, and D. Raoult. 2007. Chikungunya outbreaks – the globalization of vector borne diseases. N. Engl. J. Med. 56: 769–771.

Chastel, C. 2012. When some flaviviruses are throwing our certainties. Bull. Soc. Pathol. Exot. 105: 251–255.

Chen, C. I., D. C. Clark, P. Pesavento, N. W. Lerche, P. A. Luciw, W. K. Reisen, and A. C. Brault. 2010. Comparative pathogenesis of epidemic and enzootic Chikungunya viruses in a pregnant Rhesus macaque model. Am. J. Trop. Med. Hyg. 83:1249–1258.

Chevillon, C., L. Briant, F. Reanux, and C. Devaux. 2008. The Chikungunya threat: an ecological and evolutionary perspective. Trends Microbiol. 16: 80–88.

Chouin-Carneiro, T., A. Vega-Rua, M. Vazeille, A. Yebakima, R. Girod, D. Goindin, M. Dupont-Rouzeyrol, R. Lourenço-de-Oliveira, and A. B. Failloux. 2016. Differential Susceptibilities of *Aedes aegypti* and *Aedes albopictus* from the Americas to Zika Virus. PLoS Negl. Trop. Dis. 10: e0004543. doi:10.1371/journal.pntd.0004543.

Coffey, L. L., and S. C. Weaver. 2005. Susceptibility of *Ochlerotatus taeniorhynchus* and *Culex nigripalpus* for Everglades virus. Am. J. Trop. Med. Hyg. 73: 11–16.

Coffey, L. L., A. B. Failloux, and S. C. Weaver. 2014. Chikungunya virus-vector interactions. Viruses 11: 4628–4663.

Coimbra, T. L. M., C. L. S. Santos, A. Suzuki, S. M. C. Petrella, I. Bisordi, A. H. Nagamori, A. T. Marti, R. N. Santos, D. M. Fialho, S. Lavigne, et al. 2007. Mayaro virus: imported cases of human infection in São Paulo state, Brazil. Rev. Inst. Med. Trop. S. Paulo 49: 221–224.

Coimbra, T. L., R. N. Santos, S. Petrella, T. K. Nagasse-Sugahara, S. B. Castrignano, and C. L. Santos. 2008. Molecular characterization of two Rocio flavivirus strains isolated during the encephalitis epidemic in São Paulo State, Brazil and the development of a one-step RT-PCR assay for diagnosis. Rev. Inst. Med. Trop. São Paulo 50: 89–94.

Colón-González, F. J., C. Fezzi, I. R. Lake, and P. R. Hunter. 2013. The effects of weather and climate change on dengue. PLOS Negl. Trop. Dis. 7: e253.

Contigiani, M. S., M. de Basualdo, A. Cámara, A. Ramírez, G. Díaz, D. González, S. Medeot, and D. Osuna. 1993. Presencia de anticuerpos contra virus de la encefalitis equina Venezolana subtipo VI en pacientes con enfermedad febril aguda. Rev. Argent. Microbiol. 14: 212–220. [Presence of antibodies gainst Venezuelan Equine Encephalitis subtype VI in patients with acute febrile disease], and in the end: [in Spanish].

Cupp, E. W., K. J. Tennessen, W. K. Oldland, H. K. Hassan, G. E. Hill, C. R. Katholi, and T. R. Unnasch. 2004. Mosquito and arbovirus activity during 1997–2002 in a wetland in Northeastern Mississippi. J. Med. Entomol. 41: 495–501.

Deardorff, E. R., and S.C. Weaver. 2010. Vector competence of *Culex (Melanoconion) taeniopus for* equine-virulent subtype IE strains of Venezuelan equine encephalitis virus. Am. J. Trop. Med. Hyg. 82: 1047–1052. doi: 10.4269/ajtmh.2010.09-0556.

Dejnirattisai. W., P. Supasa, W. Wongwiwat, A. Rouvinski, G. Barba-Spaeth, T. Duangchinda, A. Sakuntabhai, V.-M. Cao-Lormeau, P. Malasit, F. A. Rey, et al. 2016. Dengue virus sero-cross-reactivity drives antibody-dependent enhancement of infection with ika virus. Nature Immun. 17:1102–1108. doi:10.1038/ni.3515.

De Majo, M. S., S. Fischer, M. Otero, and N. Schweigmann. 2013. Effects of thermal heterogeneity and egg mortality on differences in the population dynamics of *Aedes aegypti* (Diptera: Culicidae) over short distances in temperate Argentina. J. Med. Entomol. 50: 543–551.

De Majo, M. S., P. Montini, and S. Ficher. 2016. Egg hatching and survival of immature stages of *Aedes aegypti* (Diptera: Culicidae) under natural temperature conditions during the cold season in Buenos Aires, Argentina. J. Med. Entomol. (in press). doi: 10.1093/jme/tjw131.

De Novaes Oliveira, R., K. Iamamoto, M. L. Silva, S. M. Achkar, J. G. Castilho, E. D. Ono, R. S. Lobo, P. E. Brandão, P. Carnieli Jr, M. L. Carrieri, et al. 2014. Eastern equine encephalitis cases among horses in Brazil between 2005 and 2009. Arch. Virol. 159: 2615–2620.

Derraik, J.G.B. 2009. The mosquito fauna of phytotelmata in native forests in the Auckland region of New Zealand. J. Vector Ecol. 34: 157–159.

Derraik, J.G.B., D. Slaney, and P. Weinstein. 2008. Effect of height on the use of artificial larval container habitats by *Aedes notoscriptus* in native forest and zoo in Wellington, New Zealand. New Zealand Entomol. 31: 31–34.

Diaz, L. A., N. Komar, A. M. Visintín, M. J. Dantur Juri, M. Stein, R. Lobo Allende, L. I. Spinsanti, B. S. Konigheim, J. J. Aguilar, M. Laurito, et al. 2008a. West Nile virus in birds, Argentina. Emerg. Infect. Dis. 14: 689–691.

Diaz, L. A., M. Occelli, F. L. Almeida, W. R. Almirón, and M. S. Contigiani. 2008b. Eared dove (Zenaida auriculata, Columbidae) as host for St. Louis encephalitis virus (Flaviviridae, Flavivirus). Vector Borne Zoon. Dis. 8: 277–282. doi: 10.1089/vbz.2007.0168.

Diaz, L. A., F. S. Flores, M. S. Contigiani. 2011. Viremia profiles and host competence index for West Nile virus (Flavivirus, Flaviviridae) in three autochthonous birds species from Argentina. J. Ornithol. 152: 21–25. doi: 10.1007/s10336-010-0538-4.

Diaz, L. A., F. S. Flores, M. Beranek, M. E. Rivarola, W. R. Almirón and M. S. Contigiani. 2013a. Transmission of endemic St Louis encephalitis virus strains by local Culex quinquefasciatus populations in Cordoba, Argentina. Trans. R. Soc. Trop. Med. Hyg. 107: 332–334.

Diaz, L. A., F. S. Flores, A.Quaglia, and M. Contigiani. 2013b. Intertwined arbovirus transmission activity: reassessing the transmission cycle paradigm. Front. Physiol. 3: 493.

Diaz-Nieto, L. M., A. Macia, M. A. Perotti, and C. M. Beron. 2013. Geographical limits of the southeastern distribution of *Aedes aegypti* (Diptera, Culicidae) in Argentina. PLoS Negl. Trop. Dis. 7: e1963. doi:10.1371/journal.pntd.0001963.

Dibo, M. R., R. M. T. Menezes, C. P. Ghirardelli, A. L. Mendonça, and F. Chiaravalloti Neto. 2012. Presença de culicídeos em município de porte médio do Estado de São Paulo e risco de ocorrência de febre do Nilo Ocidental e outras arboviroses. Rev. Soc. Bras. Med. Trop. 44: 496–503. [Presence of Culicids in a midsize municipality in the state of São Paulo and the risk of occurrence of West Nile fever and other arboviruses] and [in Portuguese]

Dick, G. W., S. F. Kitchen, and A. J. Haddow. 1952. Zika virus. I. Isolations and serological specificity. Trans. R. Soc. Trop. Med Hyg. 46: 509–520.

Dohm, D. J., M. L. O'Guinn, and M. J. Turell. 2002. Effect of environmental temperature on the ability of *Culex pipiens* (Diptera: Culicidae) to transmit West Nile Virus. J. Med. Entomol. 39:221–225.

D'Ortenzio, E., S. Matheron, Y. Yazdanpanah, B. Hubert, G. Piorkowski, M. Maquart, D. Descamps, F. Damond, and I. Leparc-Goffart. 2016. Evidence of sexual transmission of Zika virus. N. Engl. J. Med. 374: 2195–2198.

Duffy, M. R., T. H. Chen, W. T. Hancock, M. Powers, J. L. Kool, R. S. Lanciotti, M. Pretrick, M. Marfel, S. Holzbauer, C. Dubray, et al. 2009. Zika virus outbreak on Yap Island, Federated States of Micronesia. N. Engl. J. Med. 360: 2536–2543.

Dutra, H. L. C., M. N. Rocha, F. B. S. Dias, S. B. Mansur, E. P. Caragata, and L. A. Moreira. 2016. *Wolbachia* blocks currently circulating Zika virus isolates in Brazilian *Aedes aegypti* mosquitoes. Cell Host & Microbe: http://dx.doi.org/10.1016/j.chom.2016.04.021.

Ebi, K. L., and J. Nealon. 2016. Dengue in a changing climate. Environ. Res. 151: 115–123.

Edwards, J. F. 1993 Cache Valley Virus. Vet. Clin. North America: Food An. Pract. 10: 515–524.

Effler, P. V., L. Pang, P. Kitsutani, V. Vorndam, M. Nakata, T. Ayers, J. Elm, T. Tom, P. Reiter, J. G. Rigau-Perez, et al. 2005. Dengue fever, Hawaii, 2001–2002. Emerg. Inf. Dis. 11: 742–749.

Eritja, R., R. Escosa, J. Lucientes, E. Marquès, R. Molina, D. Roiz, and S. Ruiz. 2005. Worldwide invasion of vector mosquitoes: present European distribution and challenges for Spain. Issues Bioinv. Sci. 7: 87–97.

Estrada-Franco, J. G., R. Navarro-Lopez, D. W. C. Beasley, L. Coffey, A.-S. Carrara, A. Travassos da Rosa, T. Clements, E. Wang, G. V. Ludwig, A. C. Cortes, et al. 2003. West Nile Virus in Mexico: evidence of widespread circulation since July 2002. Emerg. Inf. Dis. 9: 1604–1607.

Fares, R. C., K. P. Souza, G. Añez, and M. Rios. 2015. Epidemiological scenario of dengue in Brazil. Biomed. Res. Int.: 321873. doi: 10.1155/2015/321873.

Faria N. R., J. Lourenço, E. M. de Cerqueira, M. M. de Lima, O. Pybus, and L. C. J. Alcantara. 2016. Epidemiology of Chikungunya virus in Bahia, Brazil, 2014-2015. PLOS Currents Outbreaks. 2016 Feb 1. Edition 1. doi: 10.1371/currents.outbreaks.c97507e3e48efb946401755d468c28b2.

Favoretto, S., D. Araújo, D. Oliveira, N. Duarte, F. Mesquita, P. Zanotto, and E. Durigon. 2016. First detection of Zika virus in neotropical primates in

Brazil: a possible new reservoir. Biorv. http://dx.doi.org/10.1101/049395. Accessed June 2nd, 2016.

Fernandes, R. S., S. S. Campos, A. Ferreira-de-Brito, R. M. Miranda, K. A. B. Silva, M. G. Castro, L. M. S. Raphael, P. Brasil, A.-B. Failloux, M. C. Bonaldo, et al. 2016. Culex quinquefasciatus from Rio de Janeiro is not competente to transmit the local Zika vírus. PLOS Negl. Trop. Dis. 10: e0004993. doi:10.1371/journal.pntd.0004993.

Fernández, M. L., M. Otero, N. Schweigmann, and H. G. Solari. 2013. A mathematically assisted reconstruction of the initial focus of the yellow fever outbreak in Buenos Aires (1871). Papers in Physics 5: art. 050002. DOI: http://dx.doi.org/10.4279/PIP.050002.

Fernández, Z., A. C. Moncayo, A. S. Carrara, O. P. Forattini, and S. C. Weaver. 2003. Vector competence of rural and urban strains of Aedes (Stegomyia) albopictus (Diptera: Culicidae) from São Paulo State, Brazil for IC, ID, and IF subtypes of Venezuelan Equine Encephalitis virus. J. Med. Entomol. 40: 522–527.

Figueiredo, L. T. M. 1999. Brazilian viruses in the family Bunyaviridae. Rev. Med. Ribeirão Preto 32: 154–158.

Fischer S., M. S. De Majo, L. Quiroga, M. Paez, and N. Schweigmann. 2016. Long-term spatio-temporal dynamics of the mosquito *Aedes aegypti* in temperate Argentina. Bull. Entomol. Res. (in press).

Forattini, O. P., I. Kakitani, E. Massad, and D. Marucci. 1995. Studies on mosquitoes (Diptera: Culicidae) and anthropic environment. 9- Synanthropy and epidemiological vector role of *Aedes scapularis* in South-Eastern Brazil. Rev. Saúde Públ. 29: 199–207.

Foy, B. D., K. C. Kobylinski, J. L. C. Foy, B. J. Blitvich, A. Travassos da Rosa, A. D. Haddow, R. S. Lanciotti, and R. B. Tesh 2011. Probable non–vector-borne transmission of Zika Virus. Emerg. Inf. Dis. 7: 880–882.

Freier, J. E., and J. C. Beier. 1984. Oral and transovarial transmission of La Crosse virus by *Aedes atropalpus*. Am. J. Trop. Med. Hyg. 33: 708–714.

Frierson, J. G. 2010. The yellow fever vaccine: A history. Yale J. Biol. Med. 83: 77–85.

Gil, L.H.S., T.H.A. Katsuragawa, A. A. de Lima , M. S. Tada, L. S. Ozaki, and J.G. Rebouças 2015. Rudimentary cesspits as breeding sites for *Aedes aegypti* in urban areas of Northern Brazil. Rev. Pan-Amaz. Saúde 6: 73–80.

Gleiser, R. M., and L. P. Zalazar. 2010. Distribution of mosquitoes in relation to urban landscape characteristics. Bull. Entomol. Res. 100: 153–158.

Gleiser, R. M., D. E. Gorla, and G. Schelotto. 2000. Population dynamics of *Aedes albifasciatus* (Diptera: Culicidae) south of Mar Chiquita Lake, central Argentina. J. Med. Entomol. 37: 21–26.

Go, Y. Y., U. B. Balasuriya, and C. K. Lee. 2014. Zoonotic encephalitides caused by arboviruses: Transmission and epidemiology of alphaviruses and flaviviruses. Clin. Exp. Vaccine Res. 58–77.

Gomes, A. C., M. D. Bitencourt, D. Natal, P.L.S. Pinto, L. F. Mucci, M. B. de Paula, P. R. Urbinatti, and J.M.S. Barata. 1999. *Aedes albopictus* em área rural do Brasil e implicações na transmissão de febre amarela silvestre. [*Aedes albopictus* in rural zone of Brazil and its implication in the sylvatic yellow fever transmission]. Rev. Saúde Públ. 33: 95–97. [in Portuguese].

Granwehr, B. P., K. M. Lillibridge, S. Higgs, P. W. Mason, J. F. Aronson, G. A. Campbell, and A. D. T. Barrett. 2004. West Nile virus: Where are we now? Lancet Inf. Dis. 4: 547–556.

Gratz, N. G. 2004. Critical review of the vector status of *Aedes albopictus*. Med. Vet. Entomol. 18: 215–227.

Grills, A., S. Morrison, B. Nelson, J. Miniota, A. Watts, and M. S. Cetron. 2016. Projected Zika virus importation and subsequent ongoing transmission after travel to the 2016 Olympic and Paralympic games - country-specific assessment, July 2016. MMWR Morb. Mortal. Wkly. Rep. 65: 711–715.

Guagliardo, S. A., A. C. Morrison, J. L. Barboza, E. Requena, H. Astete, G. Vazquez-Prokopec, and U. Kitron. 2015a. River boats contribute to the regional spread of the dengue vector *Aedes aegypti* in the Peruvian Amazon. PLoS Negl. Trop. Dis. 9: e0003648. doi:10.1371/journal.pntd.0003648.

Guagliardo, S. A., A. C. Morrison, J. L. Barboza, D. M. Wesson, L. Ponnusamy, H. Astete, G. Vazquez-Prokopec, and U. Kitron. 2015b. Evidence for *Aedes aegypti* (Diptera: Culicidae) oviposition on boats in the Peruvian Amazon. J. Med. Entomol. 52: 726–729. doi: 10.1093/jme/tjv048

Gubler, D. 1998. Dengue and dengue hemorrhagic fever. Clin. Microbiol. Rev. 11: 480–496.

Guedes, D.R.D., M.H.S. Paiva, M.M.A. Donato, P. P. Barbosa, L. Krokovsky, S.W.S. Rocha, K. L. A. Saraiva, M. M. Crespo, R.M.R. Barbosa, C.M.F. Oliveira, et al. 2016. Zika virus replication in the mosquito *Culex quinquefasciatus* in Brazil. BioRxiv doi: http://dx.doi.org/10.1101/073197, accessed 14 November 2016.

Guerbois, M., I. Fernandez-Salas, S. R. Azar, R. Danis- Lozano, C. M. Alpuche-Aranda, G. Leal, I. R. Garcia-Malo, E. E. Diaz- Gonzalez, M. Casas-Martinez, S. L. Rossi, et al. 2016. Outbreak of Zika virus infection, Chiapas State, Mexico, 2015, and first confirmed transmission by *Aedes aegypti* mosquitoes in the Americas. J. Infect. Dis. 214: 1349–1356.

Hadinegoro, S. L., J. L. Arredondo-García, M. R. Capeding, C. Deseda, T. Chotpitayasunondh, R. Dietze, H. I. Hj, M. Ismail, H. Reynales, K. Limkittikul, D. M. Rivera-Medina, et al. 2015. Efficacy and long-term safety of a dengue vaccine in regions of endemic disease. N. Engl. J. Med. 373: 1195–1206. (http://www.nejm.org/doi/pdf/10.1056/NEJMoa1506223)

Halstead, S. B. 2015. Reappearance of Chikungunya, formerly called dengue, in the Americas. Emerg. Inf. Dis. 21: 557–561.

Hanley, K. A., T. P. Monath, S. C. Weaver, S. L. Rossi, R. L. Richman, and N. Vasilakis. 2013. 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. 19: 292–311. dx.doi.org/10.1016/j.meegid.2013.03.008.

Hartwig, A., A. Bosco-Laut, and R. Bowen. 2016. Chikungunya virus in non-mammalian species? A possible new reservoir. New Horiz. Transl. Med. 2: 128.

Hayes, C. G. 2001. West Nile virus: Uganda, 1937, to New York City, 1999. Ann. N. Y. Acad. Sci. 951: 25–37.

Hayes, E. B. 2009. Zika virus outside Africa. Emerg. Infect. Dis. 15: 1347–1350.

Hemme, R. R., R. Lopez-Ortiz, B. Rivera Garcia, T. M. Sharp, and R. L. Galloway. 2016. Serological evidence of infection with endemic human pathogens among free-ranging old world monkeys in Puerto Rico. Am. J. Trop. Med. Hyg. 94: 1095–1099.

Higgs, S. 2016. Zika Virus: Emergence and emergency. Vector Borne Zoonotic. Dis. 16: 75–76.

Hoch, A. L., N. E. Peterson, J. W. LeDuc, and F. P. Pinheiro. 1981. An outbreak of Mayaro virus disease in Belterra, Brazil: III. Entomological and ecological studies. Am. J. Trop. Med. Hyg. 30: 689–698.

Holden, P., and A. D. Hess. 1959. Cache Valley virus, a previously undescribed mosquito-borne agent. Science 130: 1187–1188.

Hommel, D., J. M. Heraud, A. Hulin, and A. Talarmin. 2000. Association of tonate virus (Subtype IIIB of the Venezuelan Equine Encephalitis Complex) with encephalitis in a human. Clin. Infect. Dis. 30: 188–190.

Huang, Y. J., S. Higgs, K. M. Horne, and D. L. Vanlandingham. 2014. Flavivirus-mosquito interactions. Viruses 6: 4703–4730.

Hubálek, Z., and J., Halouzka. 2000. West Nile fever – a reemerging mosquito-borne viral disease in Europe. Emerg. Inf. Dis. 5: 643–650.

Hubálek, Z., I. Rudolf, and N. Nowotny. 2014. Arbovirus pathogenic for domestic and wild animals. Adv. Virus Res. 89: 201–275.

ICTV. 2014. Virus Taxonomy: 2014 Release. http://www.ictvonline.org/virusTaxonomy.asp, accessed 21 June, 2016.

IUCN. 2016. (http://www.iucnredlist.org/) (accessed 15 May 2016).

Iversson, L. B., and T. L. M. Coimbra. 1984. Encefalite na região do Vale Do Ribeira, São Paulo, Brasil, no período pós-epidêmico de 1978 a 1983. Situação do diagnóstico etiológico e características epidemiológicas. [Encephalitis in the Valley of Ribeira region, São Paulo, Brazil, in the post-endemic period from 1978 to 1983: status of the etiological diagnosis and epidemiological characteristics]. Rev. Saúde Públ. 18: 323–332.

Iwamoto, M., D. B. Jernigan, A. Guasch, M. J. Trepka, C. G. Blackmore, W. C. Hellinger, S. Zaki, R. S. Lanciotti, S. E. Lance-Parker, C. A. Diaz-Granados, et al. 2003. Transmission of West Nile virus from an organ donor to four transplant recipients. N. Engl. J. Med. 348: 2196–2203.

Johnson, K. M., and D. H. Martin. 1974. Venezuelan equine encephalitis. Adv. Vet. Sci. Comp. Med. 18: 79–116.

Kenney, J. L., and A. C. Brault. 2014. The role of environmental, virological and vector interactions in dictating biological transmission of arthropod-borne viruses by mosquitoes. Adv. Virus Res. 89: 39–83.

Kilpatrick, A. M., L. D. Kramer, S. R. Campbell, E. O. Alleyne, A. P. Dobson, and P. Daszak. 2005. West Nile virus risk assessment and the bridge vector paradigm. Emerg. Infect. Dis. 11: 425–429.

Kilpatrick, A. M., S. L. Ladeau, and P. P. Marra. 2007. The ecology and impact of West Nile virus in the Western Hemisphere. Auk 124: 1121–1136.

Klenk, K., J. Snow, K. Morgan, R. Bowen, M. Stephens, F. Foster, P. Gordy, S. Beckett, N. Komar, D. Gubler, et al. 2004. Alligators as West Nile virus amplifiers. Emerg. Inf. Dis. 10: 2150–2155.

Komar, N., and G. G. Clark. 2006. West Nile virus activity in Latin America and the Caribbean. Rev. Panam. Salud Publ. 19: 112–117.

Komar, N., R. Lanciotti, R. Bowen, S. Langevin, and M. Bunning. 2002. Detection of West Nile virus in oral and cloacal swabs collected from bird carcasses. Emerg. Infect. Dis. 8: 741–742.

Kramer, L. D., and L. J. Chandler. 2001. Phylogenetic analysis of the envelope gene of St. Louis encephalitis virus. Arch. Virol. 146: 2341–2355.

Kramer, L. D., J. L. Hardy, and S. B. Presser. 1983. Effect of temperature of extrinsic incubation on the vector competence of *Culex tarsalis* for western equine encephalomyelitis virus. Am. J. Trop. Med. Hyg. 32: 1130–1139.

Kramer, L. D., L. M. Styer, and G. D. Ebel. 2008. A global perspective on the epidemiology of West Nile Virus. Annu. Rev. Entomol. 53: 61–81.

Kuno, G. 2015. A re-examination of the history of etiologic confusion between dengue and Chikungunya. PLoS Negl. Trop. Dis. 9: e0004101. doi:10.1371/journal.pntd.0004101.

Lambrechts, L., T. W. Scott, and D. Gubler. 2010. Consequences of the expanding global distribution of *Aedes albopictus* for dengue transmission. PLoS Negl. Trop. Dis. 4: e646.

Lancet, 2016. Zika virus at the games: Is it safe? [Editorial]. Lancet 16: 619.

Lanciotti, R. S., J. T. Roehrig, V. Deubel, J. Smith, M. Parker, K. Steele, B. Crise, K. E. Volpe, M. B. Crabtree, et al. 1999. Origin of the West Nile virus responsible for an outbreak of encephalitis in the northeastern United States. Science 286: 2333–2337.

Laporta, G. Z., T. B. Crivelaro, E. C. Vicentin, P. Amaro, M. S. Branquinho, and M. A. M. Sallum. 2008. *Culex nigripalpus* Theobald (Diptera, Culicidae) feeding habit at the Parque Ecológico. Rev. Bras. Entomol. 52: 663–668.

Le Beaud, A. D. 2008. Why arboviruses can be neglected tropical diseases. PLoS Negl. Trop. Dis. 2: 247.

Liang, G., X. Gao, and E. A. Gould. 2015. Factors responsible for the emergence of arboviruses; strategies, challenges and limitations for their control. Emerg. Microb. Infect. 4: e18. doi:10.1038/emi.2015.18

Linley, J. R., and D. D. Chadee. 1990. Fine structure of the eggs of *Psorophora columbiae, Ps. cingulata* and *Ps. ferox* (Diptera: Culicidae). Proc. Entomol. Soc. Wash. 92: 497–511.

Long, K. C., S. A. Ziegler, S. Thangamani, N. L. Hausser, T. J. Kochel, S. Higgs, and R. B. Tesh. 2011. Experimental transmission of Mayaro virus by *Aedes aegypti*. Am. J. Trop. Med. Hyg. 85: 750–757.

Lourenço-de-Oliveira, R., M. Vazeille, A.M.B. Filippis, and A. B. Failloux. 2002. Oral susceptibility to yellow fever virus of *Aedes aegypti* from Brazil. Mem. Inst. Oswaldo Cruz 97: 437–439.

Lourenço-de-Oliveira, R., M. Vazeille, A.M.B. de Filippis, and A. B. Failloux. 2004. *Aedes aegypti* in Brazil: Genetically differentiated populations with high susceptibility to dengue and yellow fever viruses. Trans. R. Soc. Trop. Med. Hyg. 98: 43–54.

Luciani, K., I. Abadía, A. O. Martínez-Torres, J. Cisneros, I. Guerra, M. García, D. Estripeaut, and J. P. Carrera. 2015. Madariaga virus infection associated with a case of acute disseminated encephalomyelitis. Am. J. Trop. Med. Hyg. 92: 1130–1132.

Maciel-de-Freitas, R., R. Aguiar, R. V. Bruno, M. C. Guimarães, R. Lourenço-de-Oliveira, M. H. F. Sorgine, C. J. Struchiner, D.S.L. O'Neill, L. Scott, and L. A. Moreira. 2012. Why do we need alternative tools to control mosquito-borne diseases in Latin America? Mem. Inst. Oswaldo Cruz 107: 828–829.

Mackay, A. J., M. Amador, A. Diaz, J. Smith, and R. Barrera. 2009. Dynamics of *Aedes aegypti* and *Culex quinquefasciatus* in septic tanks. J. Am. Mosq. Control Assoc. 25: 409–416.

Mangiafico, J. A., J. L. Sanchez, L. T. Figueiredo, J. W. LeDuc, and C. J. Peters. 1988. Isolation of a newly recognized Bunayamwera serogroup virus from a febrile human in Panama. Am. J. Trop. Med. Hyg. 39: 593–596.

Mangudo, C., J. P. Aparicio, and R. M. Gleiser. 2015. Use of treeholes as larval habitat by *Aedes aegypti* in urban, suburban and forested environments in San Ramón de la Nueva Orán, Argentina. Bull. Entomol. Res. 105: 679–684.

Marcondes, C. B., and P. L. Tauil. 2010. Dengue Silvestre: Devemos nos preocupar? [Sylvatic dengue: should we be worried?]. Rev. Soc. Bras. Med. Trop. 44: 263–264. [in Portuguese].

Massad, E., M. N. Burattini, F. A. B. Coutinho, and L. F. Lopez. 2003. Dengue and the risk of urban yellow fever reintroduction in São Paulo State, Brazil. Rev. Saúde Públ. 37: 477–484.

McCarthy, M. 2016. Zika virus was transmitted by sexual contact in Texas, health officials report. Br. Med. J. 352: i720. doi: 10.1136/bmj.i720.

Medlock, J. M., K. M. Hansford, V. Versteirt, B. Cull, H. Kampen, D. Fontenille, G. Hendrickx, H. Zeller, W. Van Bortel, and F. Schaffner. 2015. An entomological review of invasive mosquitoes in Europe. Bull. Entomol. Res. 105: 637–663. doi:10.1017/S0007485315000103

Melandri, V., A. Guimarães, N. Komar, M. L. Nogueira, A. Mondini, A. Fernandez-Sesma, J. Alencar, and I. Bosch. 2012. Serological detection of West Nile virus in horses and chicken from Pantanal, Brazil. Mem. Inst. Oswaldo Cruz 107: 1073–1075.

Meslin, F. X. 1997. Global aspects of emerging and potential zoonoses: A global perspective. Emerg. Infect. Dis. 3: 223–228.

Metselaar, D., C. R. Grainger, K. G. Oei, D. G. Reynolds, M. Pudney, C. J. Leake, P. M. Tukei, R. M. D'Offay, and D.I.H. Simpson. 1980. An outbreak of type 2 dengue fever in the Seychelles, probably transmitted by *Aedes albopictus* (Skuse). Bull. World Health Org. 58: 937–943.

Micieli, M. V., A. C. Matacchiero, E. Muttis, D. M. Fonseca, M. T. Aliota, and L. D. Kramer. 2013. Vector competence of Argentine mosquitoes (Diptera: Culicidae) for West Nile virus (Flaviviridae: Flavivirus). J. Med. Entomol. 50: 853–862.

Miller, B. R., C. J. Mitchell, and M. E. Ballinger. 1989. Replication, tissue tropisms and transmission of yellow fever virus in *Aedes albopictus*. Trans. R. Soc. Trop. Med. Hyg. 83: 252–255.

Ministerio de Salud de la Nación. República Argentina. 2016. Infección por virus Zika. Bol. Integr. Vigil. 311: 21–22.

Mitchell, C. J., T. P. Monath, and C. B. Cropp. 1981. Experimental transmission of Rocio virus by mosquitoes. Am. J. Trop. Med. Hyg. 30: 465–472.

Mitchell, C. J., O. P. Forattini, and B. R. Miller. 1986. Vector competence experiments with Rocio virus and three mosquito species from the epidemic zone in Brazil. Rev. Saúde Públ. 20: 171–177.

Mitchell, C. J., T. P. Monath, M. S. Sabattini, J. F. Daffner, C. B. Cropp, C. H. Calisher, R. F. Darsie, Jr., and W. L. Jakob. 1987. Arbovirus isolations from mosquitoes collected during and after the 1982-1983 epizootic of western equine encephalitis in Argentina. Am. J. Trop. Med. Hyg. 36: 107–113.

Mlakar, J., M. Korva, N. Tul, M. Popović, M. Poljšak-Prijatelj, J. Mraz, M. Kolenc, K. R. Rus, T. V. Vipotnik, V. F. Vodušek, et al. 2016. Zika virus associated with microcephaly. N. Engl. J. Med. DOI: 10.1056/NEJMoa1600651, accessed 2 March 2016.

Molaei, G., T. G. Andreadis, P. M. Armstrong, R. Bueno, Jr., J. A. Dennett, S. V. Real, C. Sargent, A. Bala, Y. Randle, H. Guzman, A. Travassos da Rosa, et al. 2007. Host feeding pattern of *Culex quinquefasciatus* (Diptera: Culicidae) and its role in transmission of West Nile Virus in Harris County, Texas. Am. J. Trop. Med. Hyg. 77: 73–81.

Molaei, G., P. M. Armstrong, A. C. Graham, L. D. Kramer, and T. G. Andreadis. 2015. Insights into the recent emergence and expansion of eastern equine encephalitis virus in a new focus in the Northern New England USA. Parasit. Vectors 8: 516. doi: 10.1186/s13071-015-1145-2.

Molaei, G., M. C. Thomas, T. Muller, J. Medlock, J. J. Shepard, P. M. Armstrong, and T. G. Andreadis. 2016. Dynamics of vector-host interactions in avian communities in four eastern equine encephalitis virus foci in the northeastern U.S. PLoS Negl. Trop. Dis. 10: e0004347. doi:10.1371/journal.pntd.0004347

Monath, T. P. 1980. St. Louis encephalitis. Am. Publ. Hlth. Assoc., Washington, DC.

Monath, T. P. 1989. Yellow fever, pp. 139–231. *In* T. P. Monath (ed.), The arboviruses: Ecology and epidemiology. CRC Press, Boca Raton, FL.

Monath, T. P. 1993. Arthropod-borne viruses, pp. 138–148. *In* S. S. Morse (ed.), Emerging viruses. Oxford University Press, New York, NY.

Monath, T. P. 2016. Dengue and yellow fever — Challenges for the development and use of vaccines. N. Engl. J. Med. 357: 2222–2225.

Monath, T. P., and P.F.C. Vasconcelos. 2015. Yellow fever. J. Clin. Virol. 64: 160–173.

Monath, T. P., M. S. Sabattini, R. Pauli, J. F. Daffner, C. J. Mitchell, G. S. Bowen, and C. B. Cropp. 1985. Arbovirus investigations in Argentina, 1977-1980. IV. Serologic surveys and sentinel equine program. Am. J. Trop. Med. Hyg. 34: 966–975.

Mondini, A., I. L. S. Cardeal, E. Lázaro, S. H. Nunes, C. C. Moreira, and P. Rahal. 2007. Saint Louis encephalitis virus, Brazil. Emerg. Infect. Dis. 13: 176–178.

Montgomery, B. L., and S. A. Ritchie. 2002. Roof gutters: A key container for *Aedes aegypti* and *Ochlerotatus notoscriptus* (Diptera: Culicidae) in Australia. Am. J. Trop. Med. Hyg. 57: 244–246.

Morales, A. 2016. Arbovirus en Argentina: Situación actual y nuevos desafíos para la Vigilancia Laboratorial. X Jorn. Region. Mosquitos. Mar del Plata, Buenos Aires, Argentina.

Morales, M. A., M. Barrandeguy, C. Fabbri, J. B. Garcia, A. Vissani, K. Trono, G. Gutierrez, S. Pigretti, H. Menchaca, N. Garrido, et al. 2006. West Nile Virus isolation from equines in Argentina, 2006. Emerg. Inf. Dis. 12: 1559–1561.

Morin, C. W., A. C. Comrie, and K. C. Ernst. 2013. Climate and dengue transmission: Evidence and implications. Environ. Health Perspect. 121: 1264–1272.

Morrison, T. E. 2014. Reemergence of chikungunya virus. J. Virol. 20: 11644–11647.

Moura, P. M., T. N. Docile, A. Arnóbio, and R. Figueiró. 2014. Deforestation and disordered urban growth in the state of Rio de Janeiro: Impacts on the dynamics of Dengue. Cad. UniFOA 24: 81–89.

Mourão, M.P.G., M. S. Bastos, R. P. Figueiredo, J.B.L. Gimaque, E. S. Galusso, V. M. Kramer, C.M.C. Oliveira, F. G. Naveca, and L.T.M. Figueiredo. 2012. Mayaro Fever in the City of Manaus, Brazil, 2007–2008. Vector Borne Zoonotic. Dis. 12: 42–46. doi: 10.1089/vbz.2011.0669

Mousson, L., C. Dauga, T. Garrigues, F. Schaffner, M. Vazeille, and A. B. Failloux. 2005. Phylogeography of *Aedes (Stegomyia) aegypti* (L.) and *Aedes (Stegomyia) albopictus* (Skuse) (Diptera: Culicidae) based on mitochondrial DNA variations. Genet. Res. 86: 1–11.

Muñoz, M., and J. C. Navarro. 2012. Virus Mayaro: Un arbovirus reemergente en Venezuela y Latinoamérica. [Mayaro: A re-emerging arbovirus in Venezuela and Latin America]. Biomedica 2: 286–302. [in Spanish].

Musso, D., T. Nhan, E. Robin, C. Roche, D. Bierlaire, K. Zisou, A. Shan Yan, V. M. Cao-Lormeau, and J. Broult. 2014. Potential for Zika virus transmission through blood transfusion demonstrated during an outbreak in French Polynesia, November 2013 to February 2014. Euro Surveill. 19: 20761. (http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20761)

Musso, D., V. M. Cao-Lormeau, and D. Gubler. 2015b. Zika virus: Following the path of dengue and chikungunya? Lancet 386: 244–246.

Musso, D., C. Roche, T. X. Nhan, E. Robin, A. Teissier, and V. M. Cao-Lormeau. 2015b. Detection of Zika virus in saliva. J. Clin. Virol. 68: 53–55.

Musso, D., A., Teissier, E. Rouault, S. Teururai, and J. J. de Pina, Tu-Xuan Nhan. 2016. Detection of chikungunya virus in saliva and urine. Virol. J. 13: 102. DOI 10.1186/s12985-016-0556-9.

Mustafa, M. S., V. Rasotji, S. Jain, and V. Gupta. 2014. Discovery of fifth serotype of dengue virus (DENV-5): A new public health dilemma in dengue control. Med. J. Armed Forces India 71: 67–70.

Naish, S., P. Dale, J. S. Mackenzie, J. McBride, K. Mengersen, and S. Tong. 2014. Climate change and dengue: A critical and systematic review of quantitative modelling approaches. Infect. Dis. BMC 14: 167. doi: 10.1186/1471-2334-14-167.

Nett, R. J., G. L. Campbell, and W. K. Reisen. 2009. Potential for the emergence of Japanese Encephalitis virus in California. Vector Borne Zoonotic Dis. 9: 511–517.

Ngoagouni, C., B. Kamgang, E. Nakouné, C. Paupy, and M. Kazanji. 2015. Invasion of *Aedes albopictus* (Diptera: Culicidae) into central Africa: What consequences for emerging diseases? Parasit. Vectors 8: 191. doi: 10.1186/s13071-015-0808-3.

Nogueira, P. 2009. The early history of yellow fever. Yellow fever, a symposium in commemoration of Carlos Juan Finlay, 1955. Paper 10. (http://jdc.jefferson.edu/yellow_fever_symposium/10)

Nunes, M. R., N. R. Faria, J. M. Vasconcelos, N. Golding, M. U. Kraemer, L. F. Oliveira, R. S. Azevedo, D. E. Silva, E. V. Silva, S. P. Silva, et al. 2015. Emergence and potential for spread of Chikungunya virus in Brazil. BMC Med. 13: 102. doi: 10.1186/s12916-015-0348-x.

OPS-OMS. 2016. Zika-actualización epidemiológica. (accessed 31 March 2016).

Ortiz, D. I., W. Kang, and S. C. Weaver. 2008. 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. 45: 1117–1125.

Osorio, J. E., K. A. Ciuoderis, J. G. Lopera, L. D. Piedrahita, D. Murphy, J. LeVasseur, L. Carrillo, M. C. Ocampo, and E. Hofmeister. 2012. Characterization of West Nile viruses isolated from captive American flamingoes (*Phoenicopterus ruber*) in Medellin, Colombia. Am. J. Trop. Med. Hyg. 87: 565–572.

Otero, M., N. Schweigmann, and H. G. Solari. 2008. A stochastic spatial dynamical model for *Aedes aegypti*. Bull. Math. Biol. 70: 1297–1325.

Owen, J. C., and F. R. Moore. 2006. Seasonal differences in immunological condition of three species of thrushes. Condor 108: 390–399.

Packard, R. M. 2016. "Break-Bone" fever in Philadelphia, 1780: Reflections on the history of disease. Bull. Hist. Med. 90:193–221. (https://www.press.jhu.edu/journals/bulletin_of_the_history_of_medicine/future_publications/pre_print_content/Packard.pdf)

PAHO. 2015. (http://www.paho.org/hq/index.php?option=com_content&view=article&id=9476%3A2014-yellow-fever&catid=6648%3Amedia-center-fact-sheets&Itemid=40721&lang=pt) (accessed 2 June 2016).

PAHO. 2016. Epidemiological alert. Yellow fever, 22 April 2016. (http://www.paho.org/hq/index.php?option=com_docman&task=doc_view&Itemid=270&gid=34247&lang=en) (accessed 2 June 2016).

Parham, P. E., J. Waldock, G. K. Christophides, D. Hemming, F. Agusto, K. J. Evans, N. Fefferman, H. Gaff, A. Gumel, S. LaDeau, et al. 2015. Climate, environmental and socio-economic change: Weighing up the balance in vector borne disease transmission. Phil. Trans. R. Soc. Biol. 370: 20130551. http://dx.doi.org/10.1098/rstb.2013.0551

Patz, J. A., A. K. Githeko, J. P. McCarty, S. Hussein, U. Confalonieri, and N. de Wet. 2003. Chapter 6. Climate change and infectious diseases. *In* A. J. McMichael, D. H. Campbell-Lendrum, C. F. Corvalán, K. L. Ebi, A. Githeko, J. D. Scheraga and A. Woodward. Climate change and human health. WHO, Geneva.

Paupy, C., B. Ollomo, B. Kamgang, S. Moutailler, D. Rousset, M. Demanou, J. P. Hervé, E. Leroy, and F. Simard. 2009. Comparative role of *Aedes albopictus* and *Aedes aegypti* in the emergence of dengue and Chikungunya in Central Africa. Vector Borne Zoonotic Dis. 10: 259–266.

Paupy, C., H. Delatte, L. Bagny, V. Corbel, and D. Fontenille. 2013. *Aedes albopictus*, an arbovirus vector: From the darkness to the light. Microb. Infect. 11: 1187–1195.

Pauvolid-Corrêa, A., F. N. Tavares, E. V. da Costa, F. M. Burlandy, M. Murta, A. O. Pellegrin, M.F. Nogueira, and E. E. da Silva. 2010. Serologic evidence of the recent circulation of Saint Louis encephalitis virus and high prevalence of equine encephalitis viruses in horses in the Nhecolândia sub-region in South Pantanal, Central-West Brazil. Mem. Inst. Oswaldo Cruz 105: 829–833.

Pauvolid-Corrêa, A., J. L. Kenney, D. Couto-Lima, Z.M.S. Campos, H. G. Schatzmayr, R.M.R. Nogueira, A. C. Brault, and N. Komar. 2013. Ilheus virus isolation in the Pantanal, west-central Brazil. PLoS Negl. Trop. Dis. 7: e2318. doi: 10.1371/journal.pntd.0002318.

Perez, S., R. Tato, J. J. Cabrera, A. Lopez, O. Robles, E. Paz, A. Coira, M. P. Sanchez-Seco, A. Vazquez, R. Carballo, et al. 2016.Confirmed case of Zika virus congenital infection, Spain, March 2016. Euro Surveill. 21: 30261. DOI: http://dx.doi.org/10.2807/1560-7917.ES.2016.21.24.30261

Petersen, E., M. E. S. Wilson, B. Touch, P. McCloskey, M. Mwaba, O. Bates, F. Dar, M. Mattes, G. Kidd, G Ippolito, et al. 2016. Unexpected and rapid spread of Zika virus in the Americas – implications for public health preparedness for mass gatherings at the 2016 Brazil Olympic Games. Int. J. Infect. Dis. 44: 1115.

Petersen, L. R., and A. M. Powers. 2016. Chikungunya: Epidemiology. 19:5. F1000 Faculty Rev-82. doi: 10.12688/f1000research.7171.1

Peterson, A. T., and L. P. Campbell. 2015. Global potential distribution of the mosquito *Aedes notoscriptus*, a new alien species in the United States. J. Vector Ecol. 40: 191–194.

Pinheiro, F. P., A. L. Hoch, M. L. Gomes, and D. R. Roberts. 1981. Oropouche virus. IV. Laboratory transmission by Culicoides paraensis. Am. J. Trop. Med. Hyg. 30: 172–176.

Pires, D. A., and R. M. Gleiser. 2010. Mosquito fauna inhabiting water bodies in the urban environment of Córdoba city, Argentina, following a Saint Louis encephalitis outbreak. J. Vector Ecol. 35: 401–409.

Pisano, M. B., M. J. Dantur, V. E. Ré, L. A. Díaz, A. Farías, M. P. Sánchez Seco, A. Tenorio, W. R. Almirón, and M. S. Contigiani. 2010a. Cocirculation of Rio Negro Virus (RNV) and Pixuna Virus (PIXV) in Tucumán province, Argentina. Trop. Med. Int. Health. 7: 865–868.

Pisano, M. B., V. E. Ré, L. A. Díaz, A. Farías, M. Stein, M. P. Sanchez-Seco, W. R. Almirón, and M. S. Contigiani. 2010b. Enzootic activity of Pixuna and Rio Negro viruses (Venezuelan Equine Encephalitis complex) in a neotropical region of Argentina. Vector Borne Zoonotic Dis. 2: 199–201.

Pisano, M. B., L. I. Spinsanti, L. A. Díaz, A. A. Farías, W. R. Almirón, V. E. Ré, and M. S. Contigiani. 2012. First detection of Rio Negro virus (Venezuelan equine encephalitis complex subtype VI) in Córdoba, Argentina. Mem. Inst. Oswaldo Cruz 107: 125–158.

Pisano, M. B., G. Oria, G. Beskow, J. Aguilar, B. Konigheim, M. L. Cacace, L. Aguirre, M. Stein, and M. S. Contigiani. 2013. Venezuelan equine encephalitis viruses (VEEV) in Argentina: Serological evidence of human infection. PLoS Negl. Trop. Dis. 7: e2551. doi:10.1371/journal.pntd.0002551.

Pisano, M. B., C. Torres, V. E. Ré, A. A. Farías, M. P. Sánchez Seco, A. Tenorio, R. Campos, and M. S. Contigiani. 2014. Genetic and evolutionary characterization of Venezuelan equine encephalitis virus isolates from Argentina. Infect. Genet. Evol. 26: 72–79.

Plyusnin, A., B. Beaty, R. M. Elliott, R. Goldbach, R. Kormelink, A. Lundkvist, C. S. Schmaljohn, and R. B. Tesh. 2012. Family Bunyaviridae. *In* A.M.Q. King, M. J. Adams, E. B. Carstens and E. J. Lefkowitz (eds.), Virus taxonomy: Ninth report of the international committee on taxonomy of viruses. Elsevier Academic Press, San Diego, CA.

Pollack, A. 2011. Concerns are raised about genetically engineered mosquitoes. New York Times (30 October).

Powers, A. M. 2015. Risks to the Americas associated with the continued expansion of chikungunya virus. J. Gen. Virol. 96: 1–5.

Powers, A. M., P. V. Aguilar, L. J. Chandler, A. C. Brault, T. A. Meakins, D. Watts, K. L. RussellJ. Olson, P. F. Vasconceloss, et al. 2006. Genetic relationships among Mayaro and Una viruses suggest distinct patterns of transmission. Am. J. Trop. Med. Hyg. 75: 461–469.

Pujhari, S., and J. L. Rasgon. 2016. Zika virus: A newly emergent vector-borne public health threat in the Americas. PeerJ Preprints (https://peerj.com/preprints/1781v1/)

Quaglia, A. I., L. A. Diaz, H. Argibay, M. S. Contigiani, and M. D. Saggese. 2014. West Nile and st. Louis encephalitis viruses antibodies surveillance in captive and free-ranging birds of prey from Argentina. Ecohealth 11: 603–609.

Quian, J. 2016. Dengue: el rol del pediatra en esta nueva enfermedad. Situación epidemiológica, impacto y medidas preventivas [Dengue: The role of the pediatrician in this new disease. Epidemiological situation, impact and preventive measures]. Arch. De Pediatría Uruguay 87: 3–4. [in Spanish]

Quiroz, E., P. V. Aguilar, J. Cisneros, R. B. Tesh, and S. C. Weaver. 2009. Venezuelan equine encephalitis in Panama: Fatal endemic disease and genetic diversity of etiologic viral strains. PLoS Negl. Trop. Dis. 3: e472. doi:10.1371/journal.pntd.0000472

Radke, E. G., C. J. Gregory, K. W. Kintziger, E. K. Sauber-Schatz, E. A. Hunsperger, G. R. Gallagher, J. M. Barber, B. J. Biggerstaff, D. R. Stanek, K. M. Tomashek, et al. 2009. Dengue outbreak in Key West, Florida, USA. Emerg. Inf. Dis. 18: 135–137.

Rappole, J. H., S. R. Derrickson, and Z. Hubálek. 2000. Migratory birds and spread of West Nile Virus in the Western Hemisphere. Emerg. Inf. Dis. 6: 319–328.

Rasmussen, S. A., D. J. Jamieson, M. A. Honein, and L. R. Petersen. 2016. Zika virus and birth defects — reviewing the evidence for causality. N. Engl. J. Med. 374: 1981–1987.

Receveur, M. C., M. Grandadam, T. Pistone, and D. Malvy. 2010. Infection with Mayaro vírus in a French traveller returning from the Amazon region, Brazil, January, 2010. Euro Surveill. 15: 19563. (http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19563)

Reed, K. D., J. K. Meece, and J. S. Henkel. 2003. Birds, migration and emerging zoonoses: West Nile virus, Lyme disease, Influenza A and Enteropathogens. Clin. Med. Res. 1: 5–12.

Reeves, W. C. 2001. Partners: Serendipity in arbovirus research. J. Vector Ecol. 26: 1–6.

Reeves, W. C., and W. M. Hammon. 1946. Laboratory transmission of Japanese B encephalitis virus by seven species (three genera) of North American mosquitoes. J. Exp. Med. 83: 185–194.

Reinert, J. F. 1975. Mosquito generic and subgeneric abbreviations. Mosq. Syst. 7: 105–110.

Reisen, W. K. 2003. Epidemiology of St. Louis encephalitis virus. Adv. Virus Res. 61: 139–183.

Reisen, W. K. 2013. Ecology of West Nile virus in North America. Viruses 5: 2079–2105. doi:10.3390/v5092079.

Reisen, W. K. 2015. Vectorborne Diseases and climate change. *In* J. A. Patz and B. S. Levy (eds.), Climate change and public health. Oxford University Press, New York, NY.

Reisen, W. K., C. M. Barker, Y. Fang, and V. M. Martinez. 2008. Does variation in *Culex* (Diptera: Culicidae) vector competence enable outbreaks of West Nile virus in California? J. Med. Entomol. 45: 1126–1138.

Reiter, P., S. Lathrop, M. Bunning, B. Biggerstaff, D. Singer, T. Tiwari, L. Baber, M. Amador, J. Thirion, J. Hayes, et al. 2003. Texas lifestyle limits transmission of dengue virus. Emerg. Infect. Dis. 9: 86–89.

Rezza, G. 2009. Chikungunya and West Nile virus outbreaks: What is happening in north-eastern Italy? Eur. J. Public Health 19: 236–237.

Rezza, G. 2012. *Aedes albopictus* and the reemergence of dengue. BMC Public Health 12: 72. http://www.biomedcentral.com/1471-2458/12/72

Rivas, F., L. A., Diaz, L. M. Cardenas, E. Daza, L. Bruzon, A. Alcala, O. De la Hoz, O.F.M. Caceres, G. Aristizabal, J. W. Martinez, et al. 1997. Epidemic Venezuelan Equine Encephalitis in La Guajira, Colombia, 1995. J. Inf. Dis. 175: 828–832.

Rocheleau, J. P., J. Arsenault, L. R. Lindsay, A. DiBernardo, M. A. Kulkarni, N. Côté, and P. Michel. 2013. Eastern equine encephalitis virus: High seroprevalence in horses from Southern Quebec, Canada, 2012. Vector Borne Zoonotic Dis. 10: 712–718.

Roehrig, J. T. 2013. West Nile virus in the United States – a historical perspective. Viruses 5: 3088–3108.

Rondan Dueñas, J. C., G. Albrieu Llinares, G. M. Panzetta-Dutari, and C. N. Gardenal. 2009. Two different routes of colonization of *Aedes aegypti* in Argentina from neighboring countries. J. Med. Entomol. 46: 1344–1354.

Rosa, R., E. A. Costa, R. E. Marques, T. S. Oliveira, R. Furtini, M. R. Bomfim, M. M. Teixeira, T. A. Paixão, and R. L. Santos. 2013. Isolation of Saint Louis encephalitis virus from a horse with neurological disease in Brazil. PLoS Negl. Trop. Dis. 7: e2537.

Rossi, G. C., N. T. Pascual, and F. J. Kristicevic. 1999. First record of *Aedes albopictus* (Skuse) from Argentina. J. Am. Mosq. Contr. Assoc. 15: 422.

Russell, P. K., and S. B. Halstead. 2016. Challenges to the design of clinical trials for live-attenuated tetravalent dengue vaccines. PLoS Negl Trop Dis. 10: e0004854. doi:10.1371/journal. pntd.0004854

Sabattini, M. S., T. P. Monath, C. J. Mitchell, J. F. Daffner, G. S. Bowen, R. Pauli, and M. S. Contigiani. 1985. Arbovirus investigations in Argentina, 1977-1980. I. Historical aspects and description of study sites. Am. J. Trop. Med. Hyg. 34: 937–944.

Sabattini, M. S., A. G. Avilés, and T. P. Monath. 1998a. Historical, epidemiological and ecological aspects of arbovirus in Argentina: Flaviviridae, Bunyaviridae and Rhabdoviridae, p 113–134. *In* A.P.A. Travassos da Rosa, P. F. C. Vasconcelos and J.F.S. Travassos da Rosa (eds.), An overview of arbovirology in Brazil and neighboring countries. Instituto Evandro Chagas, Belém.

Sabattini, M. S., G. Avilés, and T. Monath. 1998b. Historical, epidemiological and ecological aspects of arboviruses in Argentina: Togaviridae, Alphavirus, pp. 135–152. *In* A.P.A. Travassos da Rosa, P. F. C. Vasconcelos and J.F.S. Travassos da Rosa (eds.), Overview of arbovirology in Brazil and neighbouring countries. Instituto Evandro Chagas, Belém.

Saeed, M. F., H. Wang, M.R.T. Nunes, P.F.C. Vasconcelos, S. C. Weaver, R. E. Shope, D. M. Watts, R. B. Tesh, and D. T. Barrett. 2000. Nucleotide sequences and phylogeny of the nucleocapsid gene of Oropouche virus. J. Gen. Virol. 81: 743–748.

Salvador, F. S., and D. M. Fujita. 2016. Entry routes for Zika virus in Brazil after 2014 World Cup: New possibilities. Travel Med. Inf. Dis. 14: 49–51.

Santamartin, C., R. MacKensie, H. Trapido, P. Barreto, C. H. Mullenax, E. Gutiérrez, and C. Lesmes. 1973. Encefalitis equina venezolana en Colombia, 1967. Bol. Oficina Sanit Panam 74: 108–144.

Sardelis, M. R., M. J. Turell, D. J. Dohm, and M. L. O'Guinn. 2001. Vector competence of selected North American *Culex* and *Coquillettidia* mosquitoes for West Nile virus. Emerg. Inf. Dis. 7: 1018–1022.

Saxton-Shaw, K. D., J. P. Ledermann, J. L. Kenney, E. Berl, A. C. Graham, J. M. Russo, A. M. Powers, and J. P. Mutebi. 2015. The first outbreak of eastern equine encephalitis in Vermont: Outbreak description and phylogenetic relationships of the virus isolate. PLoS ONE 10: e0128712.

Schaffner, F., M. Vazeille, C. Kaufmann, A. B. Failloux, and A. Mathis. 2011. Vector competence of *Aedes japonicus* for chikungunya and dengue viruses. Eur. Mosq. Bull. 29: 141–142.

Schatzmayr, H. G. 2000. Dengue situation in Brazil by year 2000. Mem. Inst. Oswaldo Cruz 95: 179–181.

Schmaljohn, C. S., and S. T. Nichol. 2006. Bunyaviridae, pp. 1741–1789. *In* D. M. Knipe and P. M. Howley (eds.). Fields virology, 5th ed. Lippincott Williams & Wilkins, Philadelphia, PA.

Scott, T. W., and S. C. Weaver. 1989. Eastern equine encephalomyelitis virus: Epidemiology and evolution of mosquito transmission. Adv. Virus Res. 37: 277–328.

Serra, O. P., B. F., Cardoso, A. L. M., Ribeiro, F. A. L., dos Santos, and R. D. Slhessarenko. 2016. Mayaro virus and dengue virus 1 and 4 natural infection in culicids from Cuiabá, state of Mato Grosso, Brazil. Mem. Inst. Oswaldo Cruz 111: 20–29.

Sexton, D. J., P. E. Rollin, E. B. Breitschwerdt, G. R. Corey, S. A. Myers, M. R. Dumais, M. D. Bowen, C. S. Goldsmith, S. R. Zaki, S. T. Nichol, et al. 1997. Life-threatening Cache Valley virus infection. N. Engl. J. Med. 336: 547–549.

Shope, R. P., O. R. Causey, A. H. Andrade, and M. Theiler. 1964. The Venezuelan Equine Encephalomyelitis complex of group A arthropod-borne viruses, including Mucambo and Pixuna from the Amazon region of Brazil. Am. J. Trop. Med. Hyg. 13: 723–727.

Silva, M. L., G. J. Galiza, A. F. Dantas, R. N. Oliveira, K. Iamamoto, S. M. Achkar, and F. Riet-Correa. 2011. Outbreaks of Eastern equine encephalitis in northeastern Brazil. J. Vet. Diagn. Invest. 23: 570–575. doi: 10.1177/1040638711403414.

Silva, J. R., M. F. Romeiro, W. M. Souza, T. D. Munhoz, G. P. Borges, O.A.B. Soares, C.H.C. Campos, R. Z. Machado, M.L.C.R. Silva, J.L.M. Faria, et al. 2014. A Saint Louis encephalitis and Rocio virus serosurvey in Brazilian horses. Rev. Soc. Bras. Med. Trop. 47: 414–417.

Simon, F., H. Savini, and P. Parola. 2008. Chikungunya: A paradigm of emergence and globalization of vector-borne diseases. Med. Clin. North Am. 92: 1323–1343. ix.

Slegers, C. A. D., M. Keuter, S. Günther, J. Schmidt-Chanasit, A. J. van der Ven, and Q. de Mast. 2014. Persisting arthralgia due to Mayaro virus infection in a traveler from Brazil: Is there a risk for attendants to the 2014 FIFA World Cup? J. Clin. Virol. 60: 317–319.

Smith, G. S., and D. B. Francy 1991. Laboratory studies of A Brazilian strain of *Aedes albopictus* as a potential vector of Mayaro and Oropouche viruses. J. Am. Mosquito Control Assoc. 7: 89–93.

Smithburn, K. C., T. P. Hughes, A. W. Burke, and J. H. Paul. 1940. A neutrophic virus isolated from the blood of a native of Uganda. Am. J. Trop. Med. Hyg. 20: 471–492.

Smithburn, K. C., A. J. Haddow, and A. F. Mahaffy. 1946. A neurotropic virus isolated from *Aedes* mosquitoes caught in the Semliki forest. Am. J. Trop. Med. Hyg. 26: 189–208.

Soper, F. L. 1963. The elimination of urban yellow fever in the Americas through the eradication of *Aedes aegypti*. Am. J. Public Health 53: 7–16.

Soper, F. L., H. E. Penna, J. Serafim, Jr., M. Frobisher, and J. Pinheiro. 1933. Yellow fever without *Aedes aegypti*. Study of a rural epidemic in the Valle do Chanaan, Espirito Santo, Brazil, 1932. Am. J. Epidemiol. 18: 555–587.

Souza Lopes, O., L. A. Sacchetta, D. B. Francy, W. L. Jakob, and C. H. Calisher. 1981. Emergence of a new arbovirus disease in Brazil. III. Isolation of Rocio virus from *Psorophora ferox* (Humboldt, 1819). Am. J. Epidemiol. 113: 122–125.

Spence, L. P. 1980. St. Louis encephalitis in tropical America, pp. 451–471 *In* T. P. Monath (ed.), St. Louis encephalitis. Am. Publ. Hlth. Assoc., Washington, DC.

Spence, L., and W. G. Downs. 1968. Virological investigations in Guyana, 1956-1966. West Indian Med. J. 17: 83–89.

Spielman, A., and M. D'Antonio. 2002. Mosquito: A natural history of our most persistent and deadly foe. Hyperion, New York, NY.

Spinsanti, L. I., A. L. Basquiera, S. Bulacio, V. Somale, S. C. Kim, V. Ré, D. Rabbat, A. Zárate, J. C. Zlocowski, C. Q. Mayor, et al. 2003. St. Louis encephalitis in Argentina: The first case reported in the last seventeen years. Emerg. Inf. Dis. 9: 271–273.

Spinsanti, L. I., L. A. Diaz, N. Glatstein, S. Arselan, M. A. Morales, A. A. Farias, C. Fabbri, J. J. Aguilar, V. Ré, M. Frías, et al. 2008. Human outbreak of St. Louis encephalitis detected in Argentina, 2005. J. Clin. Virol. 42: 27–33.

Steele, K. E., and N. A. Twenhafel. 2010. Pathology of animal models of alphavirus encephalitis. Vet. Pathol. 5: 790–805.

Stewart Ibarra, A. M., S. J. Ryan, E. Beltran, R. Mejia, M. Silva, and A. Muñoz. 2013. Dengue vector dynamics (*Aedes aegypti*) influenced by climate and social factors in Ecuador: Implications for targeted control. PLoS ONE 8: e78263. doi:10.1371/journal.pone.0078263.

Sudeep, A. B., and D. Parashar. 2008. Chikungunya: An overview. J. Biosci. 33: 443–449.

Tabachnick, W. J. 2010. Challenges in predicting climate and environmental effects on vector-borne disease episystems in a changing world. J. Exp. Biol. 213: 946–954.

Tabachnick, W. J., W. R. Harvey, J. J. Becnel, G. G. Clark, C. R. Connelly, J. F. Day, P. J. Linser, and K. J. Linthicum. 2011. Countering a bioterrorist introduction of pathogen infected mosquitoes through mosquito control. J. Am. Mosquito Control Assoc. 27: 165–167.

Takashima, I., and L. Rosen. 1989. Horizontal and vertical transmission of Japanese Encephalitis virus by *Aedes japonicus* (Diptera: Culicidae). J. Med. Entomol. 26: 454–458.

Tambyah, P. A., E.S.C. Koay, M.L.M. Poon, R.V.T.P. Lin, and B.K.C. Ong. 2008. Dengue hemorrhagic fever transmitted by blood transfusion. N. Engl. J. Med. 359: 1526–1527.

Tauro, L. B., R. F. Venezuela, L. I. Spinsanti, B. S. Konigheim, and M. S. Contigiani. 2012. First case of human infection with a Bunyamwera serogroup virus in Argentina. J. Clin. Virol. 54: 98–99.

Tauro, L. B., M. E. Rivarola, E. Lucca, B. Mariño, R. Mazzini, J. F. Cardoso, M. E. Barrandeguy, M.R.T. Nunes, and M. S. Contigiani. 2015. First isolation of Bunyamwera virus (Bunyaviridae family) from horses with neurological disease and an abortion in Argentina. Vet. J. 206: 111–114.

Teixeira, M. G., and M. L. Barreto. 1996. Por que devemos, de novo, erradicar o *Aedes aegypti*. [Why we should eradicate again *Aedes aegypti*]. Ciência Saúde Coletiva 1: 122–136. [in Portuguese].

Terzian, A.C.B., A. J. Auguste, D. Vedovello, M. U. Ferreira, M. da Silva-Nunes, M. A. Sperança, R. B. Suzuki, C. Juncansen, J. P. Araújo, Jr., S. C. Weaver, et al. 2014. Short report: Isolation and characterization of Mayaro virus from a human in Acre, Brazil. Am. J. Trop. Med. Hyg. 29: 401–404.

Thomas, S. J. 2011. The necessity and quandaries of dengue vaccine. J. Inf. Dis. 203: 299–303.

Thomas, S. M., N. B. Tjaden, S. van den Bosm, and C. Beierkuhnlein. 2014. Implementing cargo movement into climate based risk assessment of vector-borne diseases. Int. J. Environ. Res. Publ. Health 11: 3360–3374.

Tjaden, N. B., S. M. Thomas, D. Fischer, and C. Beierkuhnlein. 2013. Extrinsic incubation period of dengue: Knowledge, backlog, and applications of temperature dependence. PLoS Negl. Trop. Dis. 7: e2207. doi:10.1371/journal.pntd.0002207.

Tognarelli, J., S. Ulloa, E. Villagra, J. Lagos, C. Aguayo, R. Fasce, B. Parra, J. Mora, N. Becerra, N. Lagos, et al. 2016. A report on the outbreak of Zika virus on Easter Island, South Pacific, 2014. J. Virol. 161: 665–668. DOI 10.1007/s00705-015-2695-5.

Torres, J. R., K. L. Russel, C. Vasques, R. Barrera, R. B. Tesh, R. Salas, and D. M. Watts. 2004. Family cluster of Mayaro Fever, Venezuela. Emerg. Infect. Dis. 10: 1304–1306.

Tsetsarkin, K. A., D. L. Vanlandingham, C. E. McGee, and S. Higgs. 2007. A single mutation in chikungunya virus affects vector specificity and epidemic potential. PLoS Pathog. 3: e201. http://dx.doi.org/10.1371/journal.ppat.0030201

Turell, M. J., J. R. Beaman, and R. F. Tammariello. 1992. Susceptibility of selected strains of *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) to Chikungunya virus. J. Med. Entomol. 29: 49–63.

Turell, M. J., D. J. Dohm, M. R. Sardelis, M. L. O'Guinn, T. G. Andreadis, and J. A. Blow. 2005a. An update on the potential of North American mosquitoes (Diptera: Culicidae) to transmit West Nile Virus. J. Med. Entomol. 42: 57–62.

Turell, M. J., M. L. O'Guinn, J. W. Jones, M. R. Sardelis, D. J. Dohm, D. M. Watts, R. Fernandez, A. Travassos Da Rosa, H. Guzman, R. Tesh, et al. 2005b. Isolation of viruses from mosquitoes (Diptera: Culicidae) collected in the Amazon Basin Region of Peru. J. Med. Entomol. 42: 891–898.

Turell, M. J., M. L. O'guinn, D. Dohm, M. Zyzak, D. Watts, R. Fernandez, C. Calampa, T. A. Klein, and J. W. Jones. 2008. Susceptibility of Peruvian mosquitoes to Eastern Equine Encephalitis virus. J. Med. Entomol. 45: 720–725.

Vaidyanathan, R., and T. W. Scott. 2007. Geographic variation in vector competence for West Nile virus in the *Culex pipiens* (Diptera: Culicidae) complex in California. Vector Borne Zoonotic. Dis. 2: 193–198. doi:10.1089/vbz.2006.0589

Van der Hurk, A. F., S. A. Ritchie, and J. S. Mackenzie. 2009. Ecology and geographical expansion of Japanese Encephalitis virus. Annu. Rev. Entomol. 54: 17–35.

Vasconcelos, P.F.C. 2003. Febre amarela [Yellow feber]. Rev. Soc. Bras. Med. Trop. 36: 275–293. [in Portuguese]

Vasconcelos, P.F.C., and C. H. Calisher. 2016. Emergence of human arboviral diseases in the Americas, 2000-2016. Vector Borne Zoonotic Dis. 16: 295–301.

Vasconcelos, P.F.C., J.F.S. Travassos da Rosa, A.P.A. Travassos da Rosa, N. Degallier, F. P. Pinheiro, and G. C. Sá. 1991. Epidemiologia das encefalites por arbovírus na Amazônia brasileira [Epidemiology of encephalitis by arboviruses in the Amazon region of Brazil]. Rev. Inst. Med. Trop. 33: 465–476.

Vasconcelos, P.F.C., A.P.A. Travassos da Rosa, S. G. Rodrigues, E. S. Travassos da Rosa, N. Dégallier, and J.F.S. Travassos da Rosa. 2001. Inadequate management of natural ecosystem in the Brazilian Amazon region results in the emergence and reemergence of arboviruses. Cad. Saúde Públ. 175: 155–164.

Venkat, H., E. Krow-Lucal, M. Hennessey, J. Jones, L. Adams, M. Fischer, T. Sylvester, C. Levy, K. Smith, L. Plante, et al. 2015. Concurrent outbreaks of St. Louis Encephalitis Virus and West Nile Virus disease - Arizona, 2015. MMWR Morb. Mortal. Wkly. Rep. 64: 1349–1350.

Vezzani, D., and A. E. Carbajo. 2008. *Aedes aegypti*, *Aedes albopictus*, and dengue in Argentina: Current knowledge and future directions. Mem. Inst. Oswaldo Cruz 103: 66–74.

Vieira, M. A., A.P.M. Romano, A. S. Borba, E.V.P. Silva, J. O. Chiang, K. D. Eulálio, R.S.S. Azevedo, S. G. Rodrigues, W. S. Almeida-Neto, and P.F.C. Vasconcelos. 2015. Case report: West Nile Virus encephalitis: The first human case recorded in Brazil. Am. J. Trop. Med. Hyg. 93: 377–379.

Villar, L., G. H. Dayan, J. L. Arredondo-García, D. M. Rivera, R. Cunha, C. Deseda, H. Reynales, M. S. Costa, J. O. Morales-Ramírez, G. Carrasquilla, et al. 2014. Efficacy of a tetravalent dengue vaccine in children in Latin America. N. Engl. J. Med. 372: doi: 10.1056/NEJMoa1411037.

Virus Taxonomy. 2014. Release. (http://www.ictvonline.org/virusTaxonomy.asp)

Vittor, A. Y., B. Armien, P. Gonzalez, J. P. Carrera, C. Dominguez, A. Valderrama, G. E. Glass, D. Beltran, J. Cisneros, E. Wang, et al. 2016. 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. 10: e0004554.

Volk, S. M., R. Chen, K. A. Tsetsarkin, A. P. Adams, T. I. Garcia, A. A. Sall, F. Nasar, A. J. Schuh, E. C. Holmes, S. Higgs, et al. 2010. Genome-scale phylogenetic analyses of chikungunya virus reveal independent emergences of recent epidemics and various evolutionary rates. J. Virol. 84: 649–704.

Vorou, R. 2016. Zika virus, vectors, reservoirs, amplifying hosts, and their potential to spread worldwide: What we know and what we should investigate urgently. Intern. J. Inf. Dis. 48: 85–90. http://dx.doi.org/10.1016/j.ijid.2016.05.014

Wassermann, S., P. A. Tambyah, and P. L. Lim. 2016. Yellow fever cases in Asia: Primed for an epidemic. Intern. J. Infect. Dis. 48: 98–103.

Waterman, S. H., H. S. Margolis, and J. J. Sejvar. 2015. Surveillance for dengue and dengue-associated neurologic syndromes in the United States. Am. J. Trop. Med. Hyg. 92: 996–998.

Weaver, S. C. 2014. Arrival of chikungunya virus in the New World: Prospects for spread and impact on public health. PLoS Negl. Trop. Dis. 8: e2921.

Weaver, S. C., and A. D. Barrett. 2004. Transmission cycles, host range, evolution and emergence of arboviral disease. Nat. Rev. Microbiol. 2: 789–801.

Weaver, S. C., and M. Lecuit. 2015. Chikungunya virus and the global spread of a mosquito-borne disease. N. Engl. J. Med. 372: 13.

Weaver, S. C., and W. K. Reisen. 2010. Present and future arboviral threats. Antiviral Res. 2: 328–345.

Weaver, S. C., C. Ferro, R. Barrera, J. Boshell, and J. C. Navarro. 2004. Venezuelan equine encephalitis. Annu. Rev. Entomol. 49: 141–174.

Weaver, S. C., R. Winegar, I. D. Manger, and N. L. Forrester. 2012. Alphaviruses: Population genetics and determinants of emergence. Antiviral Res. 3: 242–257.

WHO. 2016. Dengue and severe dengue. Fact sheet N°117. (http://www.who.int/mediacentre/factsheets/fs117/en/http://www.who.int/mediacentre/factsheets/fs117/en/) (accessed April 2016)

Wieten, R. W., E.F.F. Jonker, E.M.M. van Leeuwen, E.B.M. Remmerswaal, I.J.M. ten Berge, A. W. de Visser, P.J.J. van Genderen, A. Goorhuis, L. G. Visser, M. P. Grobusch, et al. 2016. A single 17D yellow fever vaccination provides lifelong immunity; characterization of yellow-fever-specific neutralizing antibody and T-Cell responses after vaccination. PLoS ONE 11: e0149871. doi:10.1371/journal.pone.0149871

Wilkerson, R. C., Y. M. Linton, D. M. Fonseca, T. R. Schultz, D. C. Price, and D. A. Strickman. 2015. Making mosquito taxonomy useful: A stable classification of tribe Aedini that balances utility with current knowledge of evolutionary relationships. PLoS ONE 10: e0133602.

Williges, E., A. Farajollahi, J. J. Scott, L. J. McCuiston, W. C. Crans, and R. Gaugler. 2008. Laboratory colonization of *Aedes japonicus*. J. Am. Mosquito Control Assoc. 24: 591–593.

Wilson, M. E., and P. Schlagenhauf. 2016. *Aedes* and the triple threat of DENV, CHIKV, ZIKV e arboviral risks and prevention at the 2016 Rio Olympic games. Travel Med. Inf. Dis. 14: 1–4.

Wolfe, N. D., A. M. Kilbourn, W. B. Karesh, H. A. Rahman, E. J. Bosi, B. C. Cropp, M. Andaum, A. Spielman, and D. J. Gubler. 2001. Sylvatic transmission of arboviruses among Bornean orangutans. Am. J. Trop. Med. Hyg. 64: 310–316.

Woodall, J. P., and T. M. Yuill. 2016. Why is the yellow fever outbreak in Angola a 'threat to the entire world'? Intern. J. Infect. Dis. 48: 96–97.

Xiao, S. H., H. Guzman, A.P.A. Travassos da Rosa, H. B. Zhu, and R. B. Tesh. 2003. Alteration of clinical outcome and histopathology of yellow

fever virus infection in a hamster model by previous infection with heterologous flaviviruses. Am. J. Trop. Med. Hyg. 68: 695–703.

Yakob, L., and T. Walker. 2016. Zika virus outbreak in the Americas: The need for novel mosquito control methods. Lancet 4: e148–e149. http://dx.doi.org/10.1016/S2214-109X(16)00048-6.

Yan-Jang, S. H., V. B., Ayres, A. C., Lyons, I., Unlu, B. W., Alto, L. W., Cohnstaedt, S., Higg, and D. L., Vanlandingham 2016. *Culex* species and Zika virus. Vector-Borne Zoon. Dis. 20: 1–4.

Young, D. S., L. D. Kramer, J. G. Maffei, R. J. Dusek, P. B. Backenson, C. N. Mores, K. A. Bernard, and G. D. Ebel. 2008. Molecular epidemiology of eastern equine encephalitis virus, New York. Emerg. Infect. Dis. 14: 456–460.

Zacks, M. A., and S. Paessler. 2010. Encephalitic alphaviruses. Vet. Microbiol. 140: 281–286.

Zanluca, C., V.C.A. Melo, A.L.P. Mosimann, G.I.V. Santos, C.N.D. Santos, and K. Luz. 2015. First report of autochthonous transmission of Zika virus in Brazil. Mem. Inst. Oswaldo Cruz 110: 569–572.