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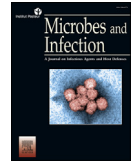


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An overview of mosquito vectors of Zika virus

Sébastien Boyer^a, Elodie Calvez^b, Thais Chouin-Carneiro^c, Diawo Diallo^d, Anna-Bella Failloux^{e,*}

^a Institut Pasteur of Cambodia, Unit of Medical Entomology, Phnom Penh, Cambodia

^b Institut Pasteur of New Caledonia, URE Dengue and Other Arboviruses, Nouméa, New Caledonia

^c Instituto Oswaldo Cruz – Fiocruz, Laboratório de Transmissores de Hematozoários, Rio de Janeiro, Brazil

^d Institut Pasteur of Dakar, Unit of Medical Entomology, Dakar, Senegal

^e Institut Pasteur, URE Arboviruses and Insect Vectors, Paris, France

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ABSTRACT

The mosquito-borne arbovirus Zika virus (ZIKV, *Flavivirus*, Flaviviridae), has caused an outbreak impressive by its magnitude and rapid spread. First detected in Uganda in Africa in 1947, from where it spread to Asia in the 1960s, it emerged in 2007 on the Yap Island in Micronesia and hit most islands in the Pacific region in 2013. Subsequently, ZIKV was detected in the Caribbean, and Central and South America in 2015, and reached North America in 2016. Although ZIKV infections are in general asymptomatic or causing mild self-limiting illness, severe symptoms have been described including neurological disorders and microcephaly in newborns. To face such an alarming health situation, WHO has declared Zika as an emerging global health threat. This review summarizes the literature on the main vectors of ZIKV (sylvatic and urban) across all the five continents with special focus on vector competence studies.

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1. Introduction

Zika virus (ZIKV) belongs to the genus *Flavivirus* in the family Flaviviridae. Within the *Flavivirus* genus, ZIKV is a mosquito-borne virus phylogenetically close to Japanese encephalitis (JEV), West Nile (WNV), dengue (DENV), and yellow fever (YFV) viruses [1]. Discovered in Uganda in 1947 [2], ZIKV emerged outside Africa in Asia after 1960. It caused the first major outbreak in Yap Island in 2007 [3,4], spread to French Polynesia [5] and other Pacific islands in 2013–2014 [6–8], reached Latin America in 2013–2015 [9,10], and ended up affecting more than 30 American countries (<https://wwwnc.cdc.gov/travel/page/world-map-areas-with-zika>). Thus, with the exception of Europe, ZIKV has circulated on all continents (Fig. 1).

ZIKV strains can be grouped into three main lineages: East African, West African, and Asian [11]. It is a 50-nm enveloped virus with an inner nucleocapsid and an outer lipid bilayer. The inner nucleocapsid is composed of a linear positive-sense, single-stranded RNA virus of 10,794-nucleotides (nt) and multiple copies

of the viral capsid (C) protein. The outer lipid bilayer derived from the host cell is covered by 180 copies of two proteins: the viral membrane M protein and the envelope (E) protein [12]. The genomic RNA comprises a single open reading frame (ORF) flanked by 3' and 5' non-coding regions. The ORF encodes a large polyprotein cleaved into 10 proteins: 3 structural proteins (C, prM, E) and 7 non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5). The viral RNA replication cycle occurs in the cell cytoplasm. The Asian lineage has been responsible for the current global expansion of ZIKV [4,11,13].

ZIKV is transmitted to humans mainly through the bite of infected mosquitoes. The two main transmission cycles are (Fig. 2): (i) a sylvatic cycle between non-human primates and arboreal canopy-dwelling mosquitoes (*Ae. africanus*, *Ae. bromeliae*, *Ae. dalzielii*, *Ae. furcifer*, *Ae. luteocapitalus*, *Ae. opok*, *Ae. taylori*, *Ae. unilineatus*, *Ae. vittatus* ...) and (ii) an urban cycle with humans as both reservoir and amplification hosts, and anthropophilic mosquitoes as vectors (primarily, *Aedes aegypti* and secondarily, *Aedes albopictus*; Fig. 1). The implication of *Ae. aegypti* as the main vector is supported by repeated isolation of ZIKV from field-collected mosquitoes [14–18] (Table 1) and experimental evidence of ability to transmit ZIKV [19–29] (Table 2). *Ae. albopictus* has been suggested to be involved in transmission as ZIKV has been

* Corresponding author.

E-mail address: anna-bella.failloux@pasteur.fr (A.-B. Failloux).

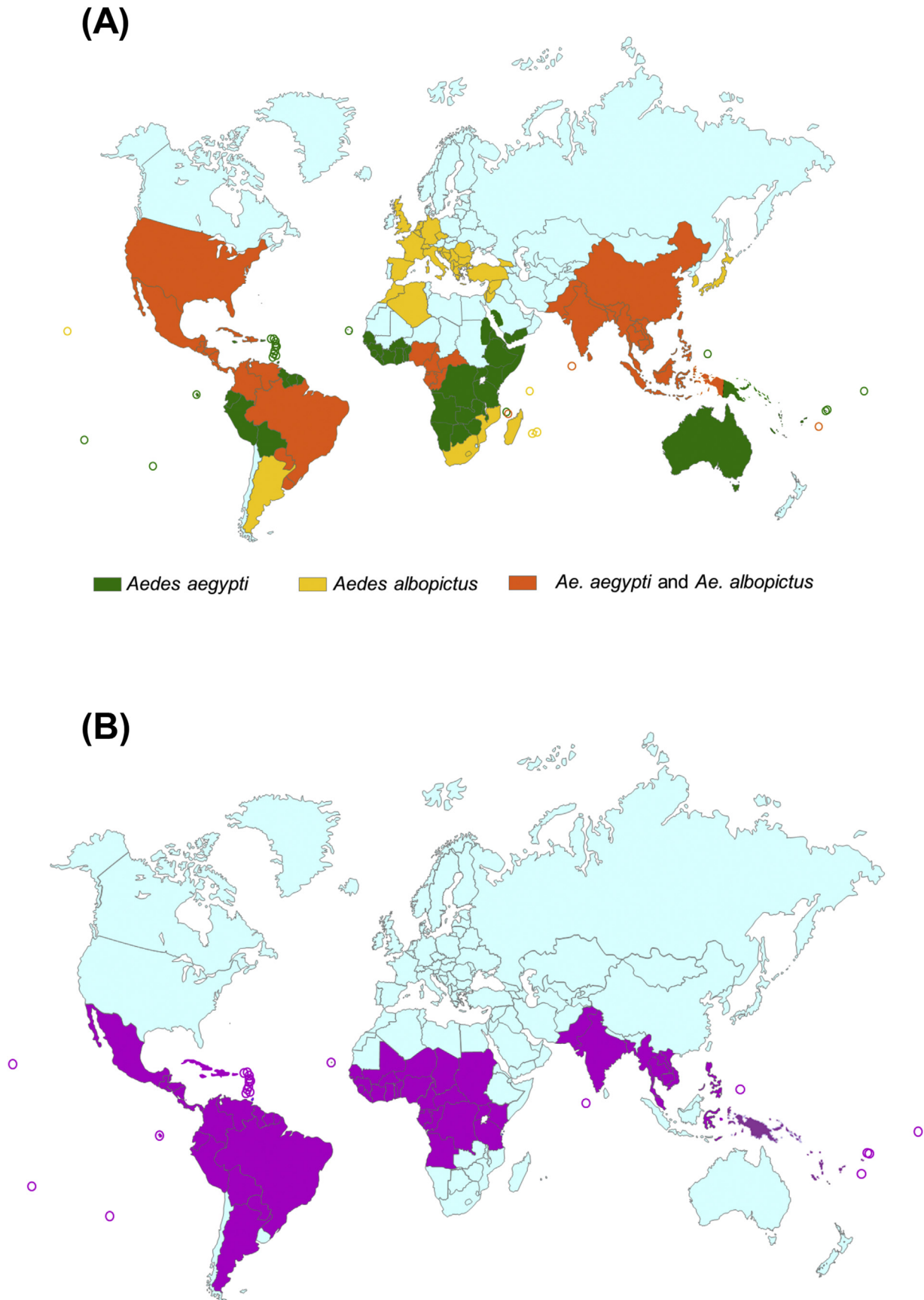


Fig. 1. Countries with reporting symptomatic ZIKV cases and geographic distribution of *Ae. aegypti* and *Ae. albopictus*.

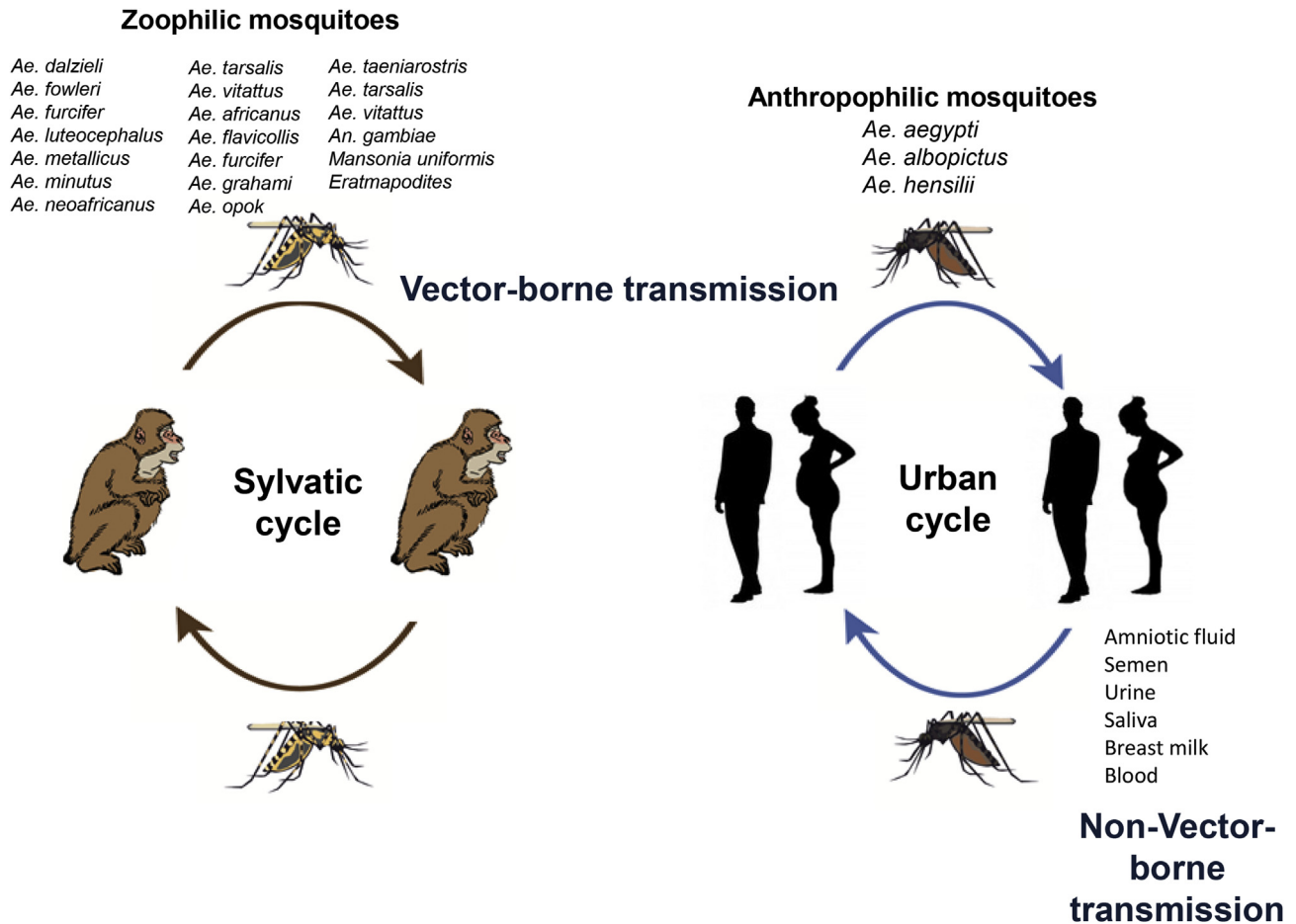


Fig. 2. Vector- and Non-vector-borne transmissions of ZIKV. Vector-borne cycles comprise a sylvatic cycle with a viral circulation between non-human primates and zoophilic mosquitoes, and an urban cycle with humans as reservoir and amplification hosts and anthropophilic mosquitoes as vectors. Non-vector borne mode involves direct human-to-human transmission by contacts of fluids.

Table 1
Mosquito species found naturally infected with ZIKV.

	Country	Sites/Localities	Years	Mosquito species	Reference
AFRICA	Senegal	<i>Saboya, Bandia, Kedougou</i>	1968–69, 1972, 1974, 77, 1980–81, 1985–89, 1991–92, 1994, 1997–99, 2001–03, 2011 and 2015	<i>Ae. aegypti, Ae. africanus, An. coustani, An. gambiae, Cx. perfuscus, Ma. uniformis, Ae. dalzieli, Ae. fowleri, Ae. furcifer, Ae. hirsutus, Ae. luteocephalus, Ae. metallicus, Ae. neoafricanus, Ae. taylori, Ae. unilineatus, Ae. vittatus, An. coustani, An. gambiae, Cx. perfuscus, Ma. uniformis</i>	[112] [14]
	Ivory coast	<i>Dabakala, Kong, Touba, Odiemé, Taï, Kakpin</i>	1973, 1975, 1979, 1980–82 and 1999	<i>Ae. aegypti, Ae. africanus, Ae. flavicollis, Ae. furcifer, Ae. grahami, Ae. luteocephalus, Ae. opok, Ae. taeniarostris, Ae. vittatus, Er. Quinquevittatus, Er. Inornatus</i>	[84] [194]
	Burkina Faso	<i>forest gallery of Dinderesso, Fada Ngourm, Bobodioulasso, Yabasso</i>	1978 and 1983	<i>Ae. aegypti, Ae. furcifer, Ae. jamoti, Ae. luteocephalus, Ae. opok</i>	[116] [195]
	Central African Republic	<i>Gomoka, Bouboui, Bozo</i>	1968, 1976 and 1979	<i>Ae. africanus, Ae. opok</i>	[111]
	Uganda	<i>Zika forest Lunyo forest</i>	1948, 1956, 1958, 1962–64, 1969–70	<i>Ae. africanus, Ae. apicoargenteus</i>	[62] [2] [113] [114] [115] [117]
	Nigeria	<i>Small forest near Tagwe</i>	1969	<i>Ae. luteocephalus</i>	[117]
	Gabon	<i>Libreville (Nzeng-Ayong and Alenkri suburbs)</i>	2007	<i>Ae. albopictus</i>	[30]
ASIA	Malaysia	<i>Bentong</i>	1966	<i>Ae. aegypti</i>	[15]
AMERICA	Mexico	<i>Guerrero state</i>	2015	<i>Ae. aegypti</i>	[18]
	Brazil	<i>Rio de Janeiro Bahía</i>	2015–2016 2015	<i>Ae. aegypti</i> <i>Ae. albopictus</i>	[16] [31]

Table 2
Vector competence of mosquitoes for ZIKV.

	Species	Mosquitoes tested		Titer of blood meal	Virus strain	Infection	Transmission	EIP (days)	References	
		Country	Locality							
AFRICA	<i>Aedes aegypti</i>	Senegal	Dakar	2.7 10 ⁶ –4 10 ⁷ pfu/mL	ArD128000 (Kedougou 1997)	+	–	–	[121]	
					ArD132912 (Kedougou 1998)	+	–	–		
					ArD157995 (Kedougou 2001)	+	–	–		
					ArD165522 (Kedougou 2002)	+	–	–		
					HD78788 (Dakar 1991)	+	–	–		
			MR766 (Uganda 1947)		+	–	–			
			Kedougou		ArD128000 (Kedougou 1997)	+	–	–	[121]	
					ArD132912 (Kedougou 1998)	+	–	–		
					ArD157995 (Kedougou 2001)	+	–	–		
					ArD165522 (Kedougou 2002)	+	–	–		
	HD78788 (Dakar 1991)	+		–	–					
	<i>Aedes vittatus</i>	Nigeria Senegal	Kebemer Ikeja near Lagos	Intrathoracic inoculation	ArD24280 (Kedougou 1976)	+	+	7	[112]	
					ZIKV	+	+	14	[120]	
			Kedougou	2.7 10 ⁶ –4 10 ⁷ pfu/mL	ArD128000 (Kedougou 1997)	+	–	–	[121]	
					ArD132912 (Kedougou 1998)	+	–	–		
					ArD157995 (Kedougou 2001)	+	–	–		
					ArD165522 (Kedougou 2002)	+	–	–		
					HD78788 (Dakar 1991)	+	–	–		
					MR766 (Uganda 1947)	+	–	–		
					MR766 (Uganda 1947)	+	+	15		
MR766 (Uganda 1947)					+	–	–			
<i>Aedes luteocephalus</i>	Senegal	Kedougou	2.7 10 ⁶ –4 10 ⁷ pfu/mL	ArD128000 (Kedougou 1997)	+	–	–	[121]		
				ArD132912 (Kedougou 1998)	+	–	–			
				ArD157995 (Kedougou 2001)	+	–	–			
				ArD165522 (Kedougou 2002)	+	–	–			
				HD78788 (Dakar 1991)	+	–	–			
				MR766 (Uganda 1947)	+	+	15			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
<i>Aedes unilineatus</i>	Senegal	Kedougou	2.7 10 ⁶ –4 10 ⁷ pfu/mL	ArD128000 (Kedougou 1997)	+	–	–	[121]		
				ArD132912 (Kedougou 1998)	+	–	–			
				ArD157995 (Kedougou 2001)	+	–	–			
				ArD165522 (Kedougou 2002)	+	–	–			
				HD78788 (Dakar 1991)	+	–	–			
				MR766 (Uganda 1947)	+	+	15			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
				MR766 (Uganda 1947)	+	–	–			
ASIA	<i>Ae. aegypti</i>	Singapore	Singapore ^a	10 ⁷ TCID ₅₀ /mL	MR766 (Uganda 1947)	+	+	4	[19]	
				10 ^{5–6} pfu/mL	H/PF/2013 (French Polynesia 2013)	+	+	3	[22]	
					BE H 815744 (Paraiba 2015)	+	+	3		
					MR766 (Uganda 1947)	+	+	4	[32]	
					H/PF/2013 (French Polynesia 2013)	+	+	ND	[22]	
	<i>Ae. albopictus</i>	Singapore	Singapore ^a	10 ^{7.5} TCID ₅₀ /mL 10 ^{5–6} pfu/mL	MR766 (Uganda 1947)	+	+	4	[32]	
					H/PF/2013 (French Polynesia 2013)	+	+	ND	[22]	
					BE H 815744 (Paraiba 2015)	+	+	ND		
					SZ01 (Samoa 2016)	+	+	8	[137]	
					H/PF/2013 (French Polynesia 2013)	+	–	–	[22]	
<i>Cx. quinquefasciatus</i>	China	Hainan	3 10 ⁵ pfu/mL	SZ01 (Samoa 2016)	+	+	8	[137]		
				H/PF/2013 (French Polynesia 2013)	+	–	–	[22]		
	Singapore	Singapore ^a	10 ^{5–6} pfu/mL	BE H 815744 (Paraiba 2015)	+	–	–			
				MR766 (Uganda 1947)	+	ND	ND	[145]		
				MR766 (Uganda 1947)	+	+	6	[23]		
PACIFIC ISLANDS	<i>Ae. hensilli</i>	French Polynesia	Tahiti ^a	10 ⁷ TCID ₅₀ /mL	PF13/251013–18 (French Polynesia 2013)	+	+	6	[23]	
					MR766 (Uganda 1947)	+	+	10	[24]	
	<i>Ae. aegypti</i>	Australia	Townsville	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	Cambodia 2010	+	+	14	[25]
						PF13/251013–18 (French Polynesia 2013)	+	–	–	[23]
	<i>Ae. polynesiensis</i>	French Polynesia	Tahiti ^a	10 ⁷ TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	MR766 (Uganda 1947)	+	–	–	[24]
						Cambodia 2010	+	+	14	[25]
	<i>Ae. notoscriptus</i>	Australia	Brisbane	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	MR766 (Uganda 1947)	+	–	–	[24]
						Cambodia 2010	+	+	14	[25]
	<i>Ae. vigilax</i>	Australia	Brisbane	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	MR766 (Uganda 1947)	+	–	–	[24]
						MR766 (Uganda 1947)	+	–	–	[24]
	<i>Ae. procax</i>	Australia	Brisbane	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	Cambodia 2010	+	+	14	[25]
						MR766 (Uganda 1947)	+	–	–	[24]
	<i>Ae. camptorhynchus</i>	Australia	Gippsland	10 ^{5.6} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	Cambodia 2010	+	+	14	[25]
						MR766 (Uganda 1947)	+	–	–	[24]
	<i>Ae. albopictus</i>	Australia	Torres Strait Islands	10 ^{5.6} TCID ₅₀ /mL	10 ⁷ TCID ₅₀ /mL	Cambodia 2010	+	+	14	[25]
						PF13/251013–18 (French Polynesia 2013)	+	–	–	[146]
	<i>Cx. quinquefasciatus</i>	French Polynesia	Tahiti	10 ⁷ TCID ₅₀ /mL	10 ^{6.7±0.2} TCID ₅₀ /mL	MR766 (Uganda 1947)	+	–	–	[24]
						Cambodia 2010	+	–	–	[25]
	<i>Cx. annulirostris</i>	Australia	Brisbane	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	MR766 (Uganda 1947)	–	–	–	[24]
						Cambodia 2010	–	–	–	[25]
<i>Cx. sitiens</i>	Australia	Brisbane	10 ^{6.7±0.2} TCID ₅₀ /mL	10 ^{5.6} TCID ₅₀ /mL	MR766 (Uganda 1947)	–	–	–	[24]	
					Cambodia 2010	–	–	–	[25]	

detected in pools of mosquitoes collected in Gabon and Brazil [30,31] (Table 1), and transmission demonstrated in laboratory [22,25,26,29,32–35] (Table 2).

The mosquito *Ae. aegypti* has a worldwide distribution and colonized most tropical countries. The geographic distribution of *Ae. aegypti* globally coincides with the area of dengue transmission. This mosquito, which breeds mainly in domestic containers [36], can take several blood-meals before oviposition, thereby increasing the chances of arbovirus infection and transmission [37]. On the other hand, *Ae. albopictus* is an opportunistic day-time and outdoor feeder, but generally prefers humans and can be found feeding and resting indoors. *Ae. albopictus* succeeded in colonizing temperate zones such as United States [38] and Europe [39], and invaded African countries where it now acts as a main vector of DENV and chikungunya virus (CHIKV) in urban [40] and rural settings [41]. In rare cases, *Ae. albopictus* can be involved in DENV epidemics (e.g. Mexico in 1997 [42], Hawaii in 2001 [43]). The species also has become the main vector of CHIKV in the world [44,45].

ZIKV, as most arboviruses, has the potential to persist in mosquito eggs. The virus can be acquired by the progeny by vertical transmission (VT) from infected mothers through (i) transovarial transmission when the virus infects germinal tissues in the ovaries and (ii) trans-egg transmission when infection occurs during fertilization [46]. VT has been demonstrated in *Ae. aegypti* and *Ae. albopictus* mosquitoes [14,16,31,47,48]. This possibility has already been suggested by the detection of ZIKV from field-collected *Ae. furcifer* males in southeastern Senegal [14].

Venereal transmission is another mechanism by which a virus can spread in a mosquito population. Males cannot get the virus from a blood meal but can acquire virus by VT from an infected female parent. In experimental studies, it has been shown that infected male *Ae. aegypti* can transmit the virus horizontally to non-infected adult females during mating. Thus venereal transmission in *Aedes* mosquitoes may have a role in the maintenance of ZIKV in nature (Campos et al. unpublished data).

In addition to vector-borne transmission, direct human-to-human transmission of ZIKV has been documented (Fig. 2): *in utero* from infected mothers to fetus [49], sexually through secretions predominantly from male to female [50–52], through breast feeding [53], blood transfusion [54–56], saliva [57], urine [58]. The importance of these non-vector-borne ZIKV transmission routes is difficult to measure. However, these multiple modes of transmission are unlikely to be as significant as mosquito-borne transmission, as suggested by their negligible effect during seasons not permissive for mosquito activity.

Beside mild symptoms, ZIKV can cause neurological disorders such as Guillain-Barré syndrome in adults [59,60] and microcephaly in newborns [61]. Zika has been then declared an emerging global health threat by the World Health Organization (WHO). In this review, we will describe the epidemiology of the disease and the main vectors (sylvatic and urban) with an emphasis on virus detection from field-collected mosquitoes and studies on vector competence in the five continents.

2. Africa, the cradle of Zika

The first evidence of ZIKV circulation was reported in Africa. The occurrence in humans and other vertebrates was cataloged by reports of outbreaks, sporadic cases and serological studies (Table 3).

2.1. East Africa

In East Africa, ZIKV was detected in many countries: Burundi, Djibouti, Ethiopia, Kenya, Somalia, Madagascar, Mozambique, Seychelles, Sudan, Tanzania, Uganda, and Zambia. The first ZIKV

isolation from a human occurred in Uganda in 1964 [62]. Neutralizing antibodies against ZIKV were detected in patients in Uganda (12.8% of positive samples) and in Tanzania (50%) in 1945, 1947–48 [63]. ZIKV circulation was then suspected in the Karamoja district of Uganda in 1967–69 and 1984 [64,65]. In Ethiopia, serological studies showed ZIKV in 3–60% of human sera collected in 1960 and 6–12% of sera collected in 1961–1964 [66]. In Kenya, antibodies against ZIKV were detected in Nyanza (3.3% of positive samples), Kitui (1.3%) and Malindi (52%) in 1964–66 [67]. In Somalia, a seroprevalence study revealed ZIKV circulation in 1966 [64]. In Zambia, anti-ZIKV IgG/IgM antibodies were found in 6% of samples tested in 2013 [68]. ZIKV appears to have circulated in other countries of East Africa: Mozambique (4% in 1957) [69], Burundi (1.4% in 1980–82) [70], Djibouti (2.2% in 1991–1992) [71], Sudan [72], Seychelles (0.7% in 1970) [73] and Madagascar (7.7% in 1977 and 1986) [74,75].

2.2. West and Central Africa

ZIKV was also detected in West and Central Africa: Angola, Benin, Burkina Faso, Cabo Verde, Cameroon, Central African Republic (CAR), Chad, Gabon, Guinea Bissau, Ivory Coast, Liberia, Mali, Niger, Nigeria, Republic of Congo, Senegal, Sierra Leone, and Togo. ZIKV was first detected in Senegal in 1962 by hemagglutination inhibition (HI) in 33% of human sera [76]. Antibodies against ZIKV were then detected in 1965, 1967, 1970–72, 1972–1976 [77], 1981 [78], 1988 and 1990 [79]. ZIKV was also isolated in humans in 1976 and 1990 in Kedougou where seroprevalence studies indicated viral circulation in 1984, 1995 and 2008 [80]. More recently, ZIKV infections in humans were reported in 2000 and 2013 [81], in 2011 (14 IgM-positive samples in Kedougou) and 2015 (17 samples in Kedougou and two other localities). A more recent retrospective study showed that 6.2% of blood samples collected between 1992 and 2016 from Senegal and Nigeria were IgM positive for ZIKV [81]. In Guinea Bissau, a ZIKV serosurvey reported that 11% of human samples collected in 1964–65 were positive [82]. Lately, ZIKV RNA was detected in blood samples from six febrile patients in the Bijagos Islands in 2016. In Ivory Coast, ZIKV circulation was suspected in 1963–65 (6 localities; 20% of positive samples) [83] and 1999 (one locality; 48% of positive samples) [84,85], and ZIKV-antibodies were detected in 45.3% of patients in 1997–1998 in Abidjan [85]. In Nigeria, 12.2–55.1% of human samples presented ZIKV antibodies in Ilobi in 1955 and in other localities in 1966–67 (Robin, in Ref. [83]), [86–89]. ZIKV was isolated from human blood samples at Ibadan in 1975 [90] and in the state of Igbo Ora-Oyo in 1971–1975 [87,91]. The most recent evidence of ZIKV active circulation was 2011 and 2013 in Jos [81]. In Cabo Verde which was the scene of the largest urban outbreak of Zika in Africa, 7557 human cases were reported mainly in the island of Santiago, Fogo and Maio in May 2016 [92]. In Gabon, ZIKV circulation was suspected in 2007 [30], in Libreville and Cocobeach. Libreville had already been exposed to ZIKV in 1967 (7% of positive samples), 1975 and 1979–80 (14.7%) [77,83,93]. In CAR, higher seroprevalences to ZIKV were documented in 1961–62 (5 localities; 48.8% of positive cases among tested ones), 1963–64 (3 localities; 6.9% of positive samples) and 1979 (1 locality; 26.3–27.4% of positive samples) [83,94,95]. In Cameroon, 2–10% blood donors were ZIKV-positive [96] corroborating the repeated exposures of humans to this virus in 1964–66 [97], 1984 [98,99] and 2015 [100]. In the neighboring country of Angola, ZIKV was detected in 27% of human sera collected in 14 localities in 1960 [101]. ZIKV was again detected in Angola in 1971–72 [102] and in 2016 in Luanda and Bengo province (detection of a case of microcephaly) [103]. In other countries in West and Central Africa, ZIKV circulation is suspected in Mali (2 localities in 1964 and 1967; 52% of positive samples), Burkina Faso (5

Table 3
Evidences of Zika virus infection in human and animals in Africa.

Country	Host	Years	Locations	Assays	References
Senegal	Human	1962	Dakar,	Serology	[76]
		1965	Casamance, Ferlo, Region du Fleuve	Isolation	[77]
		1967, 1970–76	Diourbel, Sangap, Saboya, Tankon, Kolda	PCR	[78]
		1981	Bandia		[79]
		1984	Sine Saloum		[197]
		1988	Panthiou-Sine, Niakhène		[198]
		1990	Saraya, Kedougou, Dindéfello, Fongolimbi,		
		1995	Tomboronkot, Salemata, Khossanto, Bandafassi		
		2000	Thies		
		2008	Mbour, Tambacounda, Velingara		
		2011			
		2013			
		2015			
		1996–2016			
			<i>Cercopithecus aethiops</i> , <i>Erythrocebus patas</i> , Wild mammals	1967–68	South-eastern Senegal
1973, 1976				Isolation	in Ref. [77]
Guinea Bissau	Human	1964–65, 2016	Inland, Coast Bubaque	Serology PCR	[82]
Mali	Human	1964	Nioro du Sahel, Yanfolila	Serology	[83]
Burkina Faso	Human	1963–64	Dori, Banfora, Bobodioulasso, Diébougou, Zignaré	Serology	[83]
		1963–65	Bouaké, Tiassalé, Korhogo, Man, Daloa, Sassandra	Serology	[83]
Côte d'Ivoire	Human	1997–99	Abidjan		[84]
			Comoe National Park		[85]
Nigeria	Human	1951	Ilaro, Iloba,	Serology	[199]
		1955	Imosan, Fugar, Iressa, Ado, Ase, Orhua, Ibadan,	Isolation	[88]
		1966–67	Igbo Ora, Oyo, Jos	PCR	[83]
		1971–75			[87]
		1996–2016			[90]
		2011, 2013			[91]
	Non-Human Primates	1969, 1971–72	Nupeko forest Southern Nigeria forest	Serology	[200]
			Freetown, Walihun, Sembahun, Sahn lionia, Pujohun		
Sierra Leone	Human	1972	Malcari and Magburalca Lalehun Labor Camp, Raoma Kangama, Kayima Bafodia, Koinadugu	Serology	[106]
Benin	Human	1967	Djougou and Savalou	Serology	[83]
Liberia	Human	1967, 1981–82	Loffa, Grand Bassa	Serology	[83]
					[105]
	<i>Pan troglodytes</i>			Serology	[111]
Togo	Human	1964–66	Trevis, Sokodé, Pagouda, Niamtougou, Dapango	Serology	[83]
Niger	Human	1965	Tera	Serology	[83]
Chad	Human	1954		Serology	[104]
Cabo Verde	Human	2015–16	Santiago, Fogo, Maio, Boa Vista	Serology, PCR	[92]
Gabon	Human	1967	Libreville, Coccobeach	PCR	[83]
		1975, 1979–80		Serology	[30]
		2007			[77]
					[93]
Central African Republic	Non-Human Primates	1979–80	Non-Human Primates	Serology	[93]
		1961–64,	Botambi, Obo, Bouar, Bangassou, Kemgribingui	Serology and	[83]
		1979	Bangui, Lobaye	Isolation	[94]
	<i>C. neglectus</i> , <i>C. nictitans</i> , <i>C. aethiops</i> , <i>Cercocebus</i> sp, <i>Galago demidovi</i> , <i>Pan troglodytes</i> , Bats, rodents (<i>Praomys</i> sp, <i>Lophuromys sikapusi</i> , <i>Anomalurus</i> sp)	1976		Serology	[108]
Cameroon	Human	1964–66, 1971–72,	Maroua, Garoua, Bertoua, Yaounde, Ngaoundere,	Serology	[96]
		1984, 2010, 2015	Douala, Buco, Limba, Tiko, Muyuka		[100]
					[97]
					[98]
					[99]
					[97]
					[93]
					[93]
					[93]
					[93]
Democratic Republic of Congo	<i>Pan troglodytes</i>			Serology	
Uganda	Human	1945, 1947–48,	Bwamba, Toro, Central, Molambigambo, Karamoja	Isolation	[62]
		1952, 1964		Serology	[63]
		1967–69, 1984			[64]
					[65]
					[65]
	<i>Macacca mulatta</i> , other monkeys	1947–48, 1956, 1962–63, 1969–70	Zika forest Entebbe	Isolation Serology	[2] [111] [112]

(continued on next page)

Table 3 (continued)

Country	Host	Years	Locations	Assays	References
Tanzania	Human		Tanga	Serology	[63]
Madagascar	Human	1984–86	Ambatomainty, Nosy-Bé	Serology	[63] [201] [74]
Seychelles	Human	1970		Serology	[73]
Ethiopia	Human	1960–64	Gora, Manera, Goya, Boreda Kocha, Wallamo Kaffa, Chouchouma Tchabera, Opa, Didessa	Serology	[66]
	<i>C. guereza</i> , <i>P. cynocephalus</i> Bats	1962, 1964		Serology	[108] [66] [110] [111]
Kenya	Human	1966–68	Nyanza, Kitui, Malindi, Northern	Serology	[64] [67]
Somalia	Human	1966	Mogadishu	Serology	[64]
Djibouti	Human	1991–92	Djibouti city	Serology	[71]
Zambia	Human	2014		Serology	[68]
Angola	Human	1960, 1971–72, 2016	Luanda, Bengo, 14 other localities	PCR, Serology	[101] [102] [103]
Mozambique	Human	1957		Serology	[69]
Egypt	Human	1950s		Serology	[107]
Morocco	Human	1968–69		Serology	[83]
	Birds			Serology	[83]

localities in 1963–64; 53%), Benin (between Djougou and Savalou in 1967; 44%), Togo (5 localities in 1964–66; 31%), Niger (Tera in 1965; 18%) [83], Chad (in 1954; 1.4%), Republic of Congo (in 1954; 0.4%) [104], Liberia [83,105] and Sierra Leone (14 localities in 1972; 5.3–14%) [106].

2.3. North Africa

Antibodies against ZIKV were found at low percentages in human samples collected in Egypt in 1950s [107] and in Morocco in 1968–69 [83].

2.4. Field-detections of ZIKV

2.4.1. In animals

ZIKV was isolated in a rhesus sentinel monkey (*Macacca mulatta*) in Uganda [2] and in two other monkey species (*Cercopithecus aethiops*, *Erythrocebus patas*) in Senegal. Antibodies against ZIKV were detected by HI test in 24% of 41 wild mammals collected in 1967–68 in Senegal (e.g. Chunicin, unpublished data) [83]. Anti-ZIKV antibodies were detected in non-human primates in Nigeria (83.3% in 1969), Ethiopia (50% in 1962 and 25% in 1964), CAR, Democratic Republic of Congo, Gabon in 1979–80 and Liberia [93,108,109,110,111]. Evidence for ZIKV epizootics were confirmed by serological surveys of monkeys in Uganda in 1947, 1948, 1956, 1962, 1963, 1969 and 1970), Senegal in 1973 and 1976 and CAR in 1976 [111,112]. Antibodies against ZIKV have also been identified in bats in Ethiopia and CAR, birds in Morocco and rodents in CAR [109,111].

2.4.2. In mosquitoes

ZIKV was isolated for the first time in 1948 from a pool of *Ae. africanus* collected in the Zika forest in Uganda [2]. The virus was later isolated from the same vector species in 1956, 1962–64 and 1969–70 [62,113–115]. In Africa, ZIKV was detected in 26 mosquito species (Table 1) mainly belonging to the genus *Aedes*, subgenera *Diceromyia*, *Stegomyia*, and *Aedimorphus* [14]. Detailed entomological studies in southeastern Senegal allowed detection of ZIKV in 17 mosquito species during 22 years (including eight consecutive years) over more than 40 years of arbovirus surveillance. *Ae. luteocephalus*, *Ae. africanus*, *Ae. furcifer*, *Ae. taylori* and *Ae. dalzieli*

were the mosquito species the most frequently associated with ZIKV in Senegal. In 2011, ZIKV was detected in mosquitoes collected from multiple land covers, including forests, savannas, agricultural land and villages around Kedougou. Inside villages, ZIKV was mainly detected in *Ae. vittatus* and *Ae. furcifer*, which are principally zoophagic species, meaning that they can act as bridge vectors transmitting the virus from animals to humans. In addition, repeated detection of ZIKV in pools of *Ae. furcifer* males [14] and from adults obtained from field-collected eggs of *Ae. bromeliae*, *Ae. unilineatus* and *Ae. vittatus* provide evidence of vertical transmission (Diallo et al. unpublished data). ZIKV was detected in 12 mosquito species in Ivory Coast (mainly *Ae. furcifer*, *Ae. africanus* and *Ae. luteocephalus*; between 1973 and 1999), five species in Burkina Faso (mainly *Ae. luteocephalus* and *Ae. furcifer*; in 1978 and 1983) [116], two species in CAR (*Ae. africanus* and *Ae. opok* in 1968, 1976 and 1979), *Ae. albopictus* in Gabon in 2007 [30] and *Ae. luteocephalus* in Nigeria in 1969 [117].

Aedes aegypti was the only *Aedes* species found in Cabo Verde during the ZIKV epidemic in 2015–16 (Diallo et al. unpublished data). This species has been mostly found breeding in artificial containers (including water storage and discarded containers) in Africa, but some populations of the sylvatic *Ae. aegypti formosus* breed in tree holes and other natural sites [118]. *Aedes aegypti* is mainly considered an anthropophilic, indoor and day-time feeder [119]. *Aedes albopictus* was the only species found infected during the urban ZIKV epidemic in Gabon in 2007. *Aedes furcifer*, *Ae. luteocephalus*, *Ae. africanus*, *Ae. vittatus*, *Ae. dalzieli* and *Ae. taylori* are considered to be the main vectors of ZIKV in the sylvatic and rural areas in West Africa, while *Ae. africanus* plays this role in Central and Eastern Africa. These sylvatic and rural vectors breed mainly in tree holes and fruit husks in forest galleries and savannah land covers [118]. They are mainly primatophilic, crepuscular, and outdoor feeders, but can be found feeding on humans. ZIKV has also been occasionally isolated from a wide range of mosquito species of the genera *Aedes*, *Culex*, *Eretmapodites* and *Mansonia*.

2.5. Vector competence studies

Only three published studies are available on vector competence of mosquitoes from Africa for ZIKV (Table 2). The first study used a population of *Ae. aegypti* from Nigeria which, after infection using

an artificial feeding system, was able to transmit ZIKV [120]. Another population of *Ae. aegypti* from Kebemer (located at 140 km from Dakar, Senegal) infected by intrathoracic viral injection, was found to be highly competent to ZIKV with 88% of females transmitting the virus at day 7 post infection (pi) [112]. However, when they were infected orally, 50.2% of the 221 of *Ae. aegypti* from Dakar, 57.6% of the 375 *Ae. aegypti*, 18.7% of the 300 *Ae. unilineatus*, 14.4% of the 256 *Ae. vittatus* and 75.0% of the 60 *Ae. luteocephalus* from Kedougou were susceptible to six different ZIKV strains. By contrast, only a few mosquitoes of *Ae. vittatus* and *Ae. luteocephalus* were able to transmit the virus with virus detected in mosquito saliva [121].

3. Asia, the first emergence out of Africa

3.1. ZIKV circulation in Southeast Asia

In Asia, ZIKV had been suspected to circulate from 1954, in Malaysia, Philippines, Thailand and Vietnam [107,122,123]. In Malaysia in 1953, a seroprevalence survey confirmed a positive seroneutralization in 25% of patients (six ZIKV-positive sera among 24 positive patients) [107]. In 1957, anti-ZIKV IgG/IgM antibodies were detected in 13.3% of samples (143) but not in any children under 10 years, suggesting a low circulation of ZIKV in Philippines [122]. In Thailand in 1953–54, ZIKV circulation was suspected but not serologically confirmed [123].

In the early 1980s, two serological studies in Indonesia revealed evidence for “renewed” circulation of ZIKV in Asia [124,125]; a high seroprevalence of 13% was reported in Central Java. Since 2009, ZIKV cases were more frequently reported in Asia, probably in relation to the outbreak on the Yap in 2007 in Micronesia, close to Indonesia and Philippines [3]. This first outbreak in Yap outside Africa and Asia affected more than 73% of ~11,000 residents. Thus, in Asia, ZIKV circulation has been recorded in Thailand, Cambodia, Indonesia, Philippines, Malaysia, and Vietnam in the last decade. In 2010, in Cambodia, an infection of a 3 year-old child who had never traveled was reported in the central Kampong Speu province [13]. This case was confirmed by serology, PCR and sequencing, corroborating the active circulation of ZIKV in Cambodia. Moreover, a phylogenetic analysis demonstrated that ZIKV strains responsible for the epidemic in Yap Island in 2007 and Cambodia in 2010 were similar [13]. It has been suggested that these two strains came from a common ancestor from South-East Asia, isolated in Malaysia in 1966 [13]. In Indonesia, ZIKV was circulating in 2012 following local infection of an Australian tourist with an Asian viral strain [126,127]. In Philippines, ZIKV was isolated from a 15 year-old child in a prospective longitudinal study in Cebu city [128]. The phylogenetic analysis of this virus showed that it was genetically close to the virus isolated in Yap Island. In 2013, ZIKV was notified in Thailand (detection by RT-PCR in urines) [129]. Additional studies confirmed the active circulation of the Asian ZIKV lineage: in Thailand [130,131], Indonesia [132], Malaysia [133] and Thailand [134]. Lastly, a recent retrospective study reported five ZIKV-positive sera in Cambodia from 2007, 2008, 2009 and 2015 [135], all belonging to Asian lineage. ZIKV continues to circulate in Southeast Asia with cases reported in Laos PTRs (ProMed), Vietnam (ProMed) and Singapore where from August to November 2016, 455 cases of infections were confirmed due to an Asian lineage [136]. There is no mention of a potential enzootic cycle of ZIKV in Asia.

3.2. Asian ZIKV vectors and their vector competence

Unlike Africa, entomological studies in Asia concerning ZIKV vectors are sporadic. One exception is the study in Malaysia where, in July 1966, a ZIKV strain was isolated from 29 *Ae. aegypti* females

collected in Bentong, in West-Central Malaysia [15] (Table 1). The viral strain was then retrieved after inoculation into mice confirming its identity as an Asian lineage. Both *Ae. aegypti* [15] and *Ae. albopictus* were suspected as vectors [124].

The first vector competence studies dated from 2012 in Singapore [19] (Table 2). An F1 population of *Ae. aegypti* from Singapore was infected with the Ugandan MR766 ZIKV strain. Infection of midguts was detected from day 1 pi and reached 100% at day 6 pi. Dissemination of ZIKV in salivary glands was confirmed from day 4 pi and reached 100% at day 6 pi. The viral loads in salivary glands varied between 6 and 7 Log 50% Tissue Culture Infective Dose (TCID₅₀)/mL after day 10 pi [19]. When examining F1 *Ae. albopictus* from Singapore infected with the Ugandan MR766 ZIKV strain, 73% of mosquitoes were able to transmit with virus detected in mosquito saliva at day 7 pi. Viral loads in salivary glands reached 5.96 LogTCID₅₀/mL at day 10 pi [32].

In Hainan province in China, *Culex quinquefasciatus* has been incriminated as a ZIKV vector [137]. An F1 population of this species has been infected with a ZIKV strain SZ01 isolated from Samoa in 2016 [137]. Viral dissemination to salivary glands and ovaries was shown with a peak detected at day 8 pi, and the ZIKV titers reached 4.22–4.25 Log RNA copies/mL, with direct transmission of ZIKV from infected mosquitoes to mice demonstrated [137] (Table 2). This surprising result was not confirmed when using *Cx. quinquefasciatus* from Singapore [22]. Another mosquito, *Ae. albopictus* was also suspected as a vector of ZIKV. When infected with an Asian ZIKV strain (H/PF13) from French Polynesia or an American strain collected in Brazil (BE H 815744), *Ae. albopictus* was less permissive than *Ae. aegypti* that had a higher midgut infection, an earlier presence of ZIKV in saliva and a higher susceptibility to the American ZIKV strain [22] (Table 2).

4. The South Pacific islands, the start of the pandemic

4.1. The episode in the Pacific

In 2007, ZIKV emerged in the State of Yap, Federate States of Micronesia. More than 5000 people were infected in a population of 6700 [3,4]. From 2007 to 2013, ZIKV seemed to have disappeared from the region. In 2013, ZIKV belonging to the Asian lineage emerged in French Polynesia. Health authorities estimated that more than 32,000 persons were infected (11.5% of the population). In addition, a ZIKV seroprevalence study in 2014–2015 demonstrated the presence of antibodies against ZIKV in 66% of the population sampled [5,138,139]. In French Polynesia, 42 cases of Guillain-Barré and 8 cases of microcephaly in newborns were reported [60,61]. From there, the spread of ZIKV in the Pacific region was rapid; the islands composing the Pacific region are mainly connected by airplanes facilitating the spread of the virus by infected people. ZIKV outbreaks were subsequently reported in New Caledonia in 2013, in Cook islands, Vanuatu, Fiji, Samoa, Solomon islands in 2014 and in Tonga, the Marshall islands, the Federate states of Micronesia and American Samoa in 2016 [5,140,141] (<http://www.spc.int/phd/epidemics/>). In New Caledonia, between 2014 and 2015, more than 1500 cases were confirmed [6]. Moreover, ZIKV circulation was still reported in New Caledonia and American Samoa in 2017 (source DASS-NC and <http://www.spc.int/phd/epidemics/>).

4.2. Mosquito vectors in the Pacific Islands

No ZIKV was detected from field-collected mosquitoes in the Pacific region. *Ae. aegypti* could be considered as the main ZIKV vector owing to its distribution covering most of the islands except Futuna and other isolated islands [139,142]. The first description of

Ae. aegypti was recorded at the end of the 19th century, and its introduction was probably facilitated by the migration of Europeans and Asians [143]. Since then, the presence of *Ae. aegypti* and the occurrence of a dengue epidemic were often associated. The genetic characterization of this vector demonstrated a significant population genetic structure at the Pacific scale [144]. Other *Aedes* mosquitoes, potential ZIKV vectors, were reported in this part of the world. Indeed, *Ae. albopictus* is present in the North and Central Pacific, *Aedes polynesiensis*, in the eastern part and *Aedes hensilli* in Micronesia. *Ae. hensilli* was considered the main vector of the ZIKV outbreak on Yap Island in 2007 [145]. Again, the spread of these species in the region is mainly facilitated by human travel and commercial exchanges [139,142].

4.3. Vector competence

On Yap Island, densities of *Ae. aegypti* populations are low and *Ae. hensilli* predominates making it the most likely ZIKV vector on this island. *Ae. hensilli* mosquitoes were infected with ZIKV (MR 766, African lineage). Infection was detected from day 8 pi and increased with the blood-meal titer offered to mosquitoes. Viral dissemination with virus detected in mosquito general cavity reached a maximum of 22.6%, suggesting an efficient role of the midgut in limiting escape of virus into the haemocoel [145] (Table 2).

In French Polynesia, two mosquito vectors were suspected, *Ae. aegypti* and *Ae. polynesiensis*. Their vector competence was assessed for a ZIKV belonging to Asian/Pacific lineage (PF13/251013–18) isolated from a patient in 2013 in Tahiti. When providing an infectious blood meal at a titer of 10^7 TCID₅₀/mL, *Ae. aegypti* appeared to be easily infected by the virus (>85% of mosquitoes infected up to day 21 pi). By contrast, *Ae. polynesiensis* showed an infection rate lower than 36% at day 14 pi. Concerning viral dissemination, ZIKV disseminated more in *Ae. aegypti* compared to *Ae. polynesiensis*. When examining viral transmission by detecting the virus in *Ae. aegypti* saliva, it increased over time (3% at day 6 pi and 73% at day 21 pi) (Table 2). These results suggest that ZIKV barely entered into the salivary glands during the first days post-infection. For *Ae. polynesiensis*, no ZIKV infectious particles were found until day 14 pi [23]. In addition, field-collected *Cx. quinquefasciatus* were infected with the same ZIKV strain. A low infection rate and no dissemination nor transmission was detected at day 21 pi [146] (Table 2). In New Caledonia, the vector competence of *Ae. aegypti* for ZIKV belonging to the Asian/Pacific lineage showed a high infection rate of the vector, moderate viral dissemination and low transmission of ZIKV until day 21 pi (Calvez et al. unpublished data).

In Australia, *Ae. aegypti* is well established. Seven Australian mosquito populations were infected with a blood meal containing the African lineage ZIKV strain MR766 provided at a titer of $10^{6.7}$ TCID₅₀/mL. Infection of *Ae. aegypti* was moderate (57% at day 14 pi), the viral dissemination rate was moderate to high (70% at day 14 pi) but the transmission efficiency was low (27% at day 14 pi). *Ae. aegypti* is a competent vector for ZIKV with, however, a low ability to transmit the African lineage of ZIKV. Subsequently, *Aedes notoscriptus*, *Aedes procax* and *Aedes vigilax* were tested using the same ZIKV strain. The results indicated moderate infection rates for all species, low dissemination and no transmission of ZIKV until day 14 pi (Table 2). Thus, *Ae. aegypti* is probably the main ZIKV vector and the role of other *Aedes* spp. may be excluded [24]. Also, *Culex annulirostris*, *Cx. quinquefasciatus* and *Culex sitiens* were tested for vector competence to ZIKV/MR766. Low infection and no transmission were observed for the *Culex* species suggesting a barrier to viral dissemination from the midgut [24]. When using an Asian lineage of ZIKV (strain Cambodia 2010), *Ae. aegypti* was again more efficient in transmitting the virus than *Aedes camptorhynchus* and

Aedes notoscriptus, with a transmission rate of 87% at day 14 pi. Surprisingly, *Ae. albopictus* showed a high transmission rate of 76.9% whereas *Cx. annulirostris* and *Cx. quinquefasciatus* could not transmit ZIKV [25] (Table 2).

5. An unprecedented outbreak in the Americas

5.1. Zika becomes an emerging global health threat

In 2014, the first autochthonous cases of ZIKV in the Americas were detected in Easter Island, Chile, with 51 confirmed cases [7]. In Brazil, ZIKV was first detected in symptomatic patients in March 2015, in the cities of Camaçari, Bahia [10] and in Natal, Rio Grande do Norte [9], both cities located in the Northeast of Brazil. However, a retrospective study analyzing samples from exanthematic (with rash symptoms) cases diagnosed from January to March 2015 in Rio de Janeiro, identified ZIKV suggesting that the virus was already circulating in the Southeast region [49]. Some hypotheses were put forward to explain the introduction of ZIKV into Brazil: (i) during the Football World Cup competition in June and July 2014 [9] and (ii) during the Canoe Spring World championship in August 2014, in Rio de Janeiro [147]. However, phylogenetic and molecular clock analyses estimated that ZIKV was introduced in Brazil in 2013, before these two sporting events [148], and it is consistent with the estimated timing of ZIKV introduction in the Americas [149,150]. By the end of 2015, all Brazilian regions had already reported autochthonous transmission with about 0.4–1.3 million suspected cases [151].

During the ZIKV epidemic in Brazil, the number of reports of microcephaly in newborns increased by 20-fold and reached 5400 cases [152]. The health authorities in Brazil also reported an increase in the number of cases of Guillain-Barré syndrome from 2014 to 2015 [153] suggesting a possible link between ZIKV infection and neurological disorders. Based on the results of a systematic review, WHO indicated that there was a scientific consensus that ZIKV was a cause of microcephaly and Guillain-Barré syndrome [60,154]. Early in 2016, WHO stated that infection with ZIKV was a global health threat from February to November 2016 [155,156].

After the outbreak in Brazil, Colombia was the second country to report an epidemic, with an average of 5438 cases per week and a peak of 6388 cases in February 2016 [157]. ZIKV then spread rapidly to other countries in South America, Central America and in the Caribbean [158,159]. By early 2017, almost all countries in Latin America and the Caribbean reported active ZIKV circulation, with a cumulative number of >170,000 confirmed and >510,000 suspected cases [155]. ZIKV expanded its geographic range to North America where imported ZIKV cases from South and Central American countries were detected [160,161]. Autochthonous cases were reported later in 2016 in Florida, United States (US) with 211 confirmed cases in January 2017 [162,163]. In November 2016, Texas became the second US state to confirm a locally-transmitted case of ZIKV with five autochthonous cases reported in January 2017 [163]. Until August 2017, 48 countries and territories confirmed the autochthonous transmission of ZIKV [164] with an estimation of 3–4 million cases. Until now, no specific treatment and vaccine is available and ZIKV continues to spread worldwide in areas where competent vectors are present [164]. Although the number of Zika cases has declined markedly since the outbreak of 2016, virus surveillance must remain active.

5.2. Vector-borne transmission in Americas

During the recent outbreak, *Ae. aegypti* and *Ae. albopictus* were recognized as the main vectors of ZIKV in the Americas [141]. ZIKV was first detected in *Ae. aegypti* in Mexico in 2015–2016 [17,18]

(Table 1). ZIKV was also detected in *Ae. aegypti* collected in Brazil, including a male from Rio de Janeiro, which means that the vertical and/or venereal transmission of ZIKV can occur in nature [16]. ZIKV RNAs were detected in field-collected eggs of *Ae. albopictus* from Brazil [31] (Table 1). Vertical transmission was also demonstrated in the laboratory [47,48].

5.3. *Ae. aegypti* remains the most competent

In the laboratory, both *Ae. aegypti* and *Ae. albopictus* have been shown to be efficient ZIKV vectors [20] (Table 2). The vector competence of several populations of *Ae. aegypti* and *Ae. albopictus* from the Caribbean, North America and South America was evaluated for the Asian lineage of ZIKV isolated from New Caledonia, which showed 99.4% identity with ZIKV from Brazil. The results showed that although susceptible to infection, *Ae. aegypti* and *Ae. albopictus* are unexpectedly poorly competent vectors for ZIKV. In contrast, Brazilian *Ae. aegypti* populations revealed high transmission rates and efficiencies when orally challenged with ZIKV isolated from Brazil [165,166].

Likewise, studies have revealed that American *Ae. albopictus* populations are competent to transmit ZIKV, although their competence is potentially dependent on the viral strain and the geographic origin of the mosquito population [26,33]. Interestingly, Mexican *Ae. aegypti* population showed higher rates of infection, dissemination and transmission for African ZIKV strains than American strains of the Asian lineage, responsible for the current expansion of ZIKV [21]. The difference in vector competence for ZIKV may be explained by the specific association of mosquito populations and virus genotypes [167].

The flavivirus non-structural protein-1 (NS1) is abundantly secreted into the serum of an infected host. NS1 can facilitate flavivirus acquisition by vectors. It has been shown that the increased infectivity of ZIKV and its high prevalence in *Ae. aegypti* mosquitoes have been associated with a mutation at position 188 of the NS1 protein (A188V) providing a potential explanation for the success of ZIKV transmission during the recent epidemics [168]. Potential participation of other mosquito species present in Americas was also investigated. However, it has been shown that *Cx. pipiens* and *Ae. triseriatus* mosquitoes from US were not able to transmit ZIKV [34]. Furthermore, *Ae. taeniorhynchus* from the US Gulf Coast was also not susceptible [169]. Even at a high titer, *Anopheles gambiae* and *Anopheles stephensi* from US were completely refractory to ZIKV infection [170]. Notably, *Aedes vexans* mosquitoes from northern Colorado are competent vectors of ZIKV although the potential transmission appears to be low [171].

The role of mosquitoes of the *Culex pipiens* complex in ZIKV transmission remains controversial. Most experimental infections showed that various species of *Culex* were not competent to transmit ZIKV. In the Americas, it includes *Cx. pipiens* [21,34,172], *Cx. quinquefasciatus* [21,27,165,169,170,172,173] and *Cx. tarsalis* [21]. However, a single study conducted in Brazil reported that *Cx. quinquefasciatus* is a potential ZIKV vector [28] (Table 2).

6. And Europe?

Together with CHIKV and DENV, ZIKV is transmitted by *Aedes* mosquitoes, and the countries where these mosquitoes are present could be potential sites for future Zika outbreaks. These locations could include Southern Europe where *Ae. albopictus* has already been involved in the local transmission of CHIKV and DENV. Travelers returning from areas affected by CHIKV have been diagnosed in several European countries [174,175]. In 2007, a CHIKV outbreak occurred in northern Italy, affecting 240 people over a 2-month period [176]. CHIKV hit Europe again in southern France in 2010,

2014 [177]. After decades without detecting DENV, the disease is back in Europe as demonstrated by the autochthonous cases reported in Croatia in 2010 and in southern France in 2010, 2013, and 2015 [177].

Aedes aegypti was abundant in southern Europe and the Middle East at the beginning of 20th century [178,179]. The species was responsible for several *Aedes*-borne viruses outbreaks such as yellow fever (e.g. in Livorno in the central West Coast of Italy in 1804 [180]), dengue (in Greece in 1927–28 [181]). The vector disappeared after the 1950s with the development of sanitization and management of urban water collections and anti-malaria vector control with DDT. The species has recently reinvaded European territory, Madeira island in 2005 [182], and has recently been reported around the Black Sea in southern Russia, Abkhazia, and Georgia in 2004 [183].

The invasive species *Ae. albopictus* was recorded for the first time in Europe in Albania in 1979 [184], then in Italy [185] introduced by shipments of used tires from US [186]. It is now present in more than 20 European countries [187]. Long-distance spread has been associated with the transportation of eggs by the trade of used tires [188]. Other local dispersal occurs by ground transportation [189]. In Europe, *Ae. albopictus* colonizes mainly man-made containers such as flower plates, pots, tires. In France, *Ae. albopictus* succeeded to establish in southeast of France in 2004 [190] and is now in more than 33 French departments (<http://solidarites-sante.gouv.fr/sante-et-environnement/risques-microbiologiques-physiques-et-chimiques/especes-nuisibles-et-parasites/article/cartes-de-presence-du-moustique-tigre-aedes-albopictus-en-france-metropolitaine>). Given the extensive air travel between the French overseas departments of French Guyana, Guadeloupe, Martinique and France, the risk of local transmission of ZIKV in the European area where the mosquito *Ae. albopictus* is widely distributed, has been tested using mosquito experimental infections. It has been shown that *Ae. albopictus* in southern France was weakly competent for ZIKV infection requiring at least 14 days pi to be excreted in mosquito saliva [29] (Table 2). Similarly, Italian populations of *Ae. albopictus* showed susceptibility to ZIKV with virus excreted in mosquito saliva at 11 days pi [35] (Table 2). Like other *Ae. albopictus* populations, European populations presented a lower vector competence than that of *Ae. aegypti* [29]. *Ae. aegypti* from Madeira, an autonomous region of Portugal, where the species was introduced in 2005 [182] require only 9 days to transmit the virus through mosquito saliva [29] (Table 2). To date, it appears that the risk for ZIKV in Europe is minimal based on the existing vector competence studies.

7. Concluding remarks

ZIKV is transmitted among people primarily by the bite of infected mosquitoes, *Ae. aegypti* and *Ae. albopictus*, or secondarily through non-vector modes (i.e., vertical transmission, sexual transmission, and blood transfusion). ZIKV is an African virus that has emerged outside Africa after 1960 mainly in Asia, more recently it emerged in the Pacific Islands; Yap Island in 2007, French Polynesia in 2013, and then the Americas since 2015. This virus was mainly associated with mild symptoms but more severe symptoms were described after the French Polynesian and American outbreaks. To date, this emerging virus has raised questions that are still unanswered. ZIKV transmission is characterized by low efficiency of mosquito vectors, unusual importance and diversity of non-vector-borne transmission, and the occurrence of severe cases including severe congenital malformations.

High densities of human-biting mosquitoes, high human density naïve for ZIKV and suitable environmental conditions, were mentioned as factors facilitating the rapid spread of the epidemic. It

is also legitimate to hypothesize that other anthropophilic vector species may be involved in ZIKV transmission, for example *Cx. quinquefasciatus* which are predominant in urban settings [165]. However, ZIKV has never been isolated from field-collected *Cx. quinquefasciatus* and most studies reported an absence of ZIKV transmission in laboratory [21,24,25,34,165,168,170,172,173,191,192,193] except two studies [28,137]. Pending these last results are confirmed by other studies, mosquito control measures should remain focused on the mosquito *Ae. aegypti* which is also the vector of CHIKV and DENV, and much more research effort should be allocated to fill the knowledge gaps about this virus.

Conflicts of interest

We declare that we have no competing interests.

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