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Aedes aegypti vector competence studies: A review

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Abstract

Aedes aegypti is the primary transmitter of the four viruses that have had the greatest impact on human health, the viruses causing yellow fever, dengue fever, chikungunya, and Zika fever. Because this mosquito is easy to rear in the laboratory and these viruses grow in laboratory tissue culture cells, many studies have been performed testing the relative competence of different populations of the mosquito to transmit many different strains of viruses. We review here this large literature including studies on the effect of the mosquito microbiota on competence. Because of the heterogeneity of both mosquito populations and virus strains used, as well as methods measuring potential to transmit, it is very difficult to perform detailed meta-analysis of the studies. However, a few conclusions can be drawn: (1) almost no population of *Ae. aegypti* is 100% naturally refractory to virus infection. Complete susceptibility to infection has been observed for Zika (ZIKV), dengue (DENV) and chikungunya (CHIKV), but not yellow fever viruses (YFV); (2) the dose of virus used is directly correlated to the rate of infection; (3) Brazilian populations of mosquito are particularly susceptible to DENV-2 infections; (4) the Asian lineage of ZIKV is less infective to *Ae. aegypti* populations from the American continent than is the African ZIKV lineage; (5) virus adaptation to different species of mosquitoes has been demonstrated with CHIKV; (6) co-infection with more than one virus sometimes causes displacement while in other cases has little effect; (7) the microbiota in the mosquito also has important effects on level of susceptibility to arboviral infection; (8) resistance to virus infection due to the microbiota may be direct (e.g., bacteria producing antiviral proteins) or indirect in activating the mosquito host innate immune system; (9) non-pathogenic insect specific viruses (ISVs) are also common in mosquitoes including genome insertions. These too have been shown to have an impact on the susceptibility of mosquitoes to pathogenic viruses.

One clear conclusion is that it would be a great advance in this type of research to implement standardized procedures in order to obtain comparable and reproducible results.

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1. Background

There are hundreds of known arthropod-borne-viruses (arboviruses) of which about 30 are known to cause disease in humans (Cleton et al., 2012). Despite this diversity, only four arboviruses have caused by far the most human suffering, the viruses causing yellow fever, dengue, chikungunya and Zika. Not coincidently, one mosquito, *Aedes aegypti*, has historically been the primary vector in almost all major human epidemics of these four viruses. “Not coincidently” because these viruses are native to Africa, humans are a native African primate, and *Ae. aegypti* is a native African mosquito. It has been suggested that this long history together has allowed the viruses, mosquito, and primate host to coevolve in their native Africa before spreading around the world (Powell, 2018).

These four viruses are all single-stranded RNA viruses, known to have high mutation rates, which has likely aided their rapid evolution and adaptation to replicate in different hosts (Weaver, 2006; Rückert and Ebel, 2018). Three are flaviviruses, yellow fever virus (YFV), dengue viruses (DENVs), and Zika virus (ZIKV) and one an alphavirus, chikungunya virus (CHIKV). All cause similar symptoms in humans, high fever lasting 4–14 days and joint pain. Yet each has its unique pathology with high rates of mortality for YFV and sometimes DENVs, but rarely for CHIKV or ZIKV.

Fortuitously, *Ae. aegypti* is the easiest mosquito to rear and manipulate in the laboratory. The viruses can be grown in mosquito cell tissue cultures and either injected or added to blood used to feed females. This has led to a large number of laboratory studies of the relative competence (see definition below for vector competence) of mosquitoes from diverse geographic populations to transmit these viruses. The prevalence of diseases caused by these viruses is geographically heterogeneous likely, at least partly, due to variation in competence among local populations of *Ae. aegypti*.

Here we review studies of the ability of these four viruses to be transmitted by geographically diverse populations of *Ae. aegypti* We struggle with the issue of heterogeneity in laboratory procedures and virus strains used in an attempt to detect underlying patterns. How genetic diversity that affects phenotypes, such as vector competence, varies among populations remains an open question. However, the fact that populations of *Ae. aegypti* are genetically distinct (e.g., Gloria-Soria et al., 2016) makes it more likely that they vary in vector competence compared to genetically uniform species. We also consider the contribution of microbiota in vector competence. Microbiota is a normal part of the physiology of vectors and it is clear that these microbes can affect how mosquitoes react to infection with viruses. However, details of the interactions and how these interactions vary among genetically heterogeneous mosquito populations remain to be elucidated

1.1. Quantifying the epidemiological impact of Ae. aegypti

Aedes aegypti was first identified as vector for arbovirus in 1900 in Cuba by Walter Reed, Carlos Finlay and James Carroll (Reed and Carroll, 1901). A few years later (1906), Thomas Bancroft demonstrated that *Ae. aegypti* is able to also transmit DENVs and linked frequency of transmission to the diurnal biting habits of *Ae. aegypti* (Bancroft, 1906). The

identification of the role of mosquitoes in the transmission cycle of human pathogens led scientists to the concept of vector control, that is, the control of pathogen transmission through the control of vectors. To formulate epidemiological predictions and assess the impact of vector control strategies, objective parameters have been proposed since the early 1900s that would mathematically link mosquito behaviors and their biological properties to pathogen transmission (Smith et al., 2012). The basic elements of the mathematical model of mosquito-borne disease were first conceptualized in the Ross-Mac-Donald "vectorial capacity" equation (Smith et al., 2012). Vectorial capacity defines the transmission potentials of a mosquito population and equals to $VC = [ma^2bp^n]/-\ln(p)$ where "m" is the density of vectors in relation to the host; "a" is the daily probability that the vector feeds on a host, this variable is raised to the second power because a mosquito needs to bite twice to perpetuate pathogen transmission; "b" is the intensity of transmission in relation to the initial infection rate, also called vector competence; "p" is the daily survival rate of a vector; "n" is the days it takes for a pathogen to move from the point of entry in the mosquito body (i.e. the mosquito midgut) to the point of exit (i.e. saliva), a parameter called "extrinsic incubation period" (EIP); and "1/ln(p)" is the probability of vector's surviving the EIP (Kauffman and Kramer, 2017; Rückert and Ebel, 2018).

Environmental and genetic factors of both the vector and the pathogen interact to influence the parameters of the VC equation. For instance, temperature influences EIP, the probability of mosquito survival, and may also indirectly affect adult density by impacting larval developmental time as amply discussed and reviewed elsewhere (Le Flohic et al., 2013; Gould and Higgs, 2009; Fish, 2008; Tabachnick, 2016; Kauffman and Kramer, 2017). Temperature also influences *Ae. aegypti* vector competence to DENVs (Carrington et al., 2013; Chepkorir et al., 2014; Gloria-Soria et al., 2017). Vector competence is defined as the capacity of a mosquito to acquire the pathogen and support its transmission; it is one of the most difficult parameters to compare among studies because no standardized procedures have been proposed and agreed upon by workers in the field to define viral transmission. An attempt to reduce the variability in vector competence estimates based on the genetic variability of the mosquito populations under test is to measure the heritability of viral titers in half-sibling experiments (i.e. Garcia-Luna et al., 2018; Vezzeille et al., 2016).

It has been challenging to identify a proxy for transmission given the difficulties in developing animal models for arboviral diseases that mimic pathogenesis and immunity in humans (Zompi and Harris, 2012). For instance, for DENVs, ZIKV and CHIKV various mouse models have been developed by genetically suppressing the mouse immune systems to allow viral replication and manifestation of disease symptoms (Na et al., 2017; Morrison and Diamond, 2017). However, these models are not applicable to all DENV serotypes (Na et al., 2017). YFV infects Indian crown and rhesus macaques that were used to develop early YFV vaccines (Beck and Barrett, 2015). In older literature, vector competence is often expressed in terms of infection and/or dissemination rate, that is the percentage of engorged females with virus detected in the head (as a proxy for the salivary glands, which are located at the base of the mosquito head) and/or in the whole body or legs. In more recent literature, the percentage of engorged females with viral particles in the saliva following the EIP (i.e. transmission rate) is often reported (Table 1). Viruses can be detected with various methods, primarily with RT-PCR using virus-specific primers and indirect immunofluorescent assays

on head squashes. A few studies have tested transmission by inoculating tissue cultures (*Aedes albopictus* C6/36 and *Ae. aegypti* Aeg2 are the most used) with mosquito body extracts or saliva and doing plaque assays or testing for viral particles after an incubation period (Calvez et al., 2017; Agha et al., 2017); this confirms *live* virus particles are present in saliva, rather than simply viral RNA as detected by RT-PCR. Viral detection to test for transmission is mostly pursued between 7 and 14 days after viral infection (Table 1). Shorter incubation periods are used for CHIKV as this virus has a faster dissemination rate than DENVs (Dubrulle et al., 2009; Rückert and Ebel, 2018).

1.2. Vector competence of Ae. aegypti populations for arboviruses

Despite the lack of uniformity in the procedures to test for vector competence and a focus on sampling mosquitoes in geographic areas with endemic arboviral infections or with significant epidemics (i.e. Thailand, Vietnam, New Caledonia, Mexico, Brazil, Florida, La Reunion island and Senegal), review of literature on infection, dissemination and transmission rates of arboviruses by *Ae. aegypti* mosquitoes support some general conclusions, data in Table 1. (1) Cases of complete refractoriness to arboviral infection are rare (Kay et al., 1979; Rosen et al., 1985; Diallo et al., 2008; Dickson et al., 2014; Agha et al., 2017). (2) Complete susceptibility to infection has been detected for *Ae. aegypti* populations from New Caledonia, Thailand, Australia, South Africa for DENVs; for *Ae. aegypti* populations from Dominican Republic, Brazil, China and Singapore for ZIKV; for populations from Mexico and Guadaloupe for CHIKV (Girod et al., 2011; Vega-Ruiz et al., 2014), but complete susceptibility was not observed for any population tested for YFV (Table 1); (3) Initial infection dose of virus positively correlates with infection rate. (4) Brazilian populations of *Ae. aegypti* are particularly susceptible to DENV-2 (Goncalves et al., 2014; Carvalho-Leandro et al., 2012; Lourenco-De-Oliveira et al., 2004). (5) The African lineage of ZIKV was shown to be more infective to *Ae. aegytpi* mosquitoes from the American continent than the ZIKV Asian lineage (Weger-Lucarelli et al., 2016; Roundy et al., 2017). (5) Virus adaptation to different mosquito species appears an important evolutionary force for CHIKV evolution, but its role in DENVs evolution is still controversial (Lambrechts et al., 2009; Tsetsarkin et al., 2011; Fansiri et al., 2016). The best-known example of vector-driven adaptation in an arbovirus is the emergence on La Reunion in 2005 of the A226V amino acid substitution in the E1 envelope glycoprotein of CHIKV that favors its replication in *Aedes albopictus* mosquitoes (Tsetsarkin et al., 2011). (6) Limited data are available on co-infections with different viruses or serotypes/genotypes of one viral species. Some co-infection experiments suggest competitive displacement of DENV-4 over DENV-1 (Vazeille et al., 2016) or superinfection interference (Muturi et al., 2017). Other studies indicate that *Ae. aegypti* infection with one arbovirus (i.e. CHIKV, DENV2 or ZIKV) only mildly affects infection with a subsequent infection with another (Rückert et al., 2017).

The most obvious and well accepted observation from reviewing literature on vector competence in *Ae. aegypti* is that there is great variability in susceptibility to arboviral infections across geographic populations and even for the same population with different viral species and strains; this variability includes comparisons between the domestic *Ae. aegypti aegypti* and the sylvatic *Ae. aegypti formosus* with respect to DENVs infections

(Bosio et al., 1998; Gaye et al., 2014; Dickson et al., 2014). The great variation among geographic populations of mosquito is likely due to the fact that vector competence is a complex and evolving phenotype dependent on the tri-partite interaction among the host (i.e. mosquito), the pathogen, and host symbionts (Vasilakis and Tesh, 2015; Hedge et al., 2015). The high genetic structure among *Ae. aegypti* populations is also a likely contributing factor. This variation across populations suggests that the co-evolution between *Ae. aegypti* and arboviruses did not favor a single pathway/factor in the mosquito, likely because exposure to arboviral infection is the accidental consequence of hematophagy the primary purpose of which is to support egg development. Furthermore, it is unclear how great, or even if there is, any fitness cost to mosquitoes to transmit these viruses (see e.g., Padilha et al., 2018). Selection-driven variation is more likely to be on the virus.

Specific physiological and genetic factors in mosquitoes contributing to vector competence have been thoroughly reviewed elsewhere (Franz et al., 2015; Pando-Robles and Batista, 2017; Wang et al., 2017; Palmer et al., 2018).

1.3. Microbiota and vector competence

The gut of mosquitoes is colonized by a resident microbiota which influences key physiological processes related to pathogen transmission (Guégan et al., 2018; Pike et al., 2017). In *Ae. aegypti*, DENVs replication is significantly affected by gut bacterial flora (Xi et al., 2008; Ramirez et al., 2014), the depletion of which by antibiotics renders mosquitoes more susceptible (Xi et al., 2008). Oral reintroduction of specific bacterial species into the adult mosquito midgut results in decreased viral load in the vector (Ramirez et al., 2012, 2014). Mosquito gut bacteria are presumed to exert antiviral activity through either direct or indirect mechanisms (Dennison et al., 2014; Saraiva et al., 2016; Guégan et al., 2018). While these mechanisms are not completely understood, recent studies have demonstrated that indirect mechanisms rely mainly on the basal level activation of innate antiviral responses and antimicrobial peptides (AMPs) by the gut microbiota (Xi et al., 2008; Ramirez et al., 2012). On the other hand, antiviral activity may be directly mediated by bacterial antiviral compounds (Ramirez et al., 2014). Indeed, a *Chromobacterium* sp. isolated from the *Ae. aegypti* midgut in Panama (Csp_P) produces an aminopeptidase that can bind to envelope protein of DENVs and prevent viral attachment and further invasion/replication within the host cell (Saraiva et al., 2018). Interestingly, the same bacterium has been shown to be pathogenic to both *Ae. aegypti* and *An. gambiae* (Ramirez et al., 2014) via the production of hydrogen cyanide (Short et al., 2018). Besides, it is important to consider the massive increase of bacteria in the midgut of mosquito vectors after a blood meal, and the interference with physiological processes related to the control of midgut homeostasis, such as the production of Reactive Oxygen Species (ROS) and the peritrophic matrix (Kumar et al., 2010; Oliveira et al., 2011; Rodgers et al., 2017). These processes may potentially affect mosquito vector competence and should be further investigated.

The environment, especially the larval breeding water, is pivotal in determining the mosquito gut microbiota composition (Coon et al., 2014; Duguma et al., 2015; Gimonneau et al., 2014), which varies considerably among local habitats of geographically distinct populations (Coon et al., 2016). Most of the diversity found in the *Ae. aegypti* larvae gut is also present

in the water where mosquitoes developed, with about half of it being transtadially transferred from larvae to adults (Coon et al., 2014). In addition to the environment, the mosquito genetic background also likely influences gut microbial diversity. While the mechanisms surrounding this interplay are largely unknown, concomitant decreases in both mosquito and bacterial genetic diversity have been observed in *Ae. albopictus* populations recently introduced in France (Minard et al., 2015).

It remains an open question of whether (and how) the gut microbial diversity influences mosquito competence to transmit human pathogenic arboviruses. Is the difference in vector competence among distinct mosquito populations due to their intrinsic microbiomes or genetic differences in the mosquitoes or, most likely, a combination/interaction of both factors? In this context, assessment of the gut bacteria repertoire of the genetically-selected DENV-resistant (MOYO-R) and -susceptible (MOYO-R) *Ae. aegypti* strains, identified some bacterial genera exclusively in either the resistant or in the susceptible strain (Charan et al., 2013). More recently, bacteria from the families *Rhodobacteriaceae* and *Desulfuromonadaceae* have been described as potential biomarkers of ZIKV infection in *Ae. aegypti* (Villegas et al., 2018). Exposure of germ-free *Ae. aegypti* larvae to different microbiota-derived bacterial species has been shown to result in variation in several mosquito life-history traits, including the load of DENVs disseminated to the insect head (Dickson et al., 2017). While these studies provide important insights on the interplay between mosquito microbiomes and vector competence, the relative contribution of mosquito genetics and its microbiome in the control of vector competence remains to be elucidated. This will almost certainly be key for understanding fundamental aspects of the variation in arbovirus transmission by different populations of *Ae. aegypti*.

1.4. Viriome and vector competence

The recent explosion of metagenomics studies led to the discovery of novel viral species, which are insect-specific and not able to replicate in vertebrate cells despite being phylogenetically-related to arboviruses (Vasilakis and Tesh, 2015; Bolling et al., 2015; Roundy et al., 2017). Insect-Specific Viruses (ISVs) identified so far in *Ae. aegypti* mosquitoes belong primarily to the *Flaviviridae* family, followed by the *Negoviridae* and *Bunyaviridae* families (Vasilakis and Tesh, 2015; Bolling et al., 2015; Hall et al., 2017). While the landscape of ISVs and their prevalence in natural mosquito populations vary greatly, the cell fusing agent virus (CFAV) appears to be the most common ISV in field-collected *Ae. aegypti* (Cook et al., 2006; Hall et al., 2017). Interestingly, CFAV transmits vertically and is absent in saliva and salivary glands of *Ae. aegypti* (Guegan et al., 2018). The impact of CFAV on *Ae. aegypti* vector competence has not been investigated yet, but heterologous interference was seen between Eilat virus and CHIKV in *Ae. aegypti* (Nasar et al., 2015). Eilat virus is an ISV of the *Alphavirus* genus, which was first isolated in *Anopheles constani* mosquitoes from Israel (Nasar et al., 2014). It readily infects *Ae. aegypti* (Nasar et al., 2014) and when used to infect mosquitoes prior to CHIKV infection, it delays CHIKV dissemination by 3 days (Nasar et al., 2015). Furthermore, it is possible that ISVs influence, to some extent, the mosquito's innate immune response, which could directly impact viral replication and the gut microbial diversity. These studies underscore the importance of expanding our knowledge of the viriome (the set of viruses in an organism)

and highlight its possible application for the control of arboviral infections within mosquitoes (Hall et al., 2017).

Interaction between viruses and mosquitoes may include horizontal transfer of genetic material. The genome of *Ae. aegypti* is rich in sequences with similarities to ISVs of the *Flavivirus* and *Rhabdovirus* genera and Chuviruses (Chen et al., 2015; Palatini et al., 2017; Whitfiled et al., 2017). Sequences of viral origin are statistically enriched in piRNA clusters and encode for piRNAs, suggesting that they may function analogously to transposable element fragments within the piRNA pathway (Palatini et al., 2017, Whitefiled et al., 2017). In light of this, it has been proposed that viral integrations constitute a heritable immune signal and thus could be an additional factor shaping mosquito vector competence (Olson and Bonizzoni, 2017; Palatini et al., 2017; Whitfield et al., 2017).

2. Conclusions and perspective

The recent emergence and spread of Zika, the current re-emergence of yellow fever in Brazil and Africa, the emergence of dengue in Europe, and the expansion of chikungunya to the New World brought vector-borne diseases to public attentions and fostered research. Despite great progress in the understanding of the interplay between arboviruses and vectors, the genetic and environmental elements that control vector competence in *Ae. aegypti* populations have yet to be fully elucidated. Here we reviewed historical and modern data on factors influencing vector competence in *Ae. aegypti* populations to four of the most prevalent arboviruses (i.e. DENVs, YFV, ZIKV and CHIKV). We identified no clear-cut distinctive natural factors associated with variation in vector competence among mosquito populations and/or viral species due primarily to the heterogeneity of materials (strains of mosquito and virus) and methods used in different studies. This highlights the need to standardize surveillance and laboratory procedures for assessing vector competence and to expand the range of mosquito populations and viral strains (and serotypes) tested (Fig. 1). While workers target populations and virus strains of interest to them, at the very least procedures to determine what are reported as infection rate, dissemination rate, and transmission rate should be standardized.

While there is a clear influence of the microbiota on arboviral infection, the relative importance of mosquito genetics and microbial diversity, including the interplay between these factors, on vector competence remains largely unknown and deserves attention from the scientific community.

Acquisition of arboviruses by mosquitoes is a by-product of blood-feeding, which is a necessary physiological process for egg production. Even during active arboviral epidemics, the frequency of mosquitoes infected with the pathogenic virus is usually around 1%, but can vary from 0.05% to > 10% (Chow et al., 1998; Pham Thi et al., 2017; Perez-Castro et al., 2016; Medeiros et al., 2018). In addition to these human pathogenic viruses, blood-feeding exposes mosquitoes to a broad range of entities, including bacteria, fungi and other symbionts and parasites. Considering the essential role of blood-feeding, mosquitoes must be able to withstand these microbial challenges to survive. In this context, co-evolution between mosquitoes and viruses should be viewed as a by-product of diverse and possibly

broad-range physiological processes. Some of these interactions may be deterministic and selection-driven while others may be stochastic (e.g., genetic drift) or indirect. In any case, it is clear that the genetic heterogeneity both within and among mosquito populations need to be considered in any attempts to identify genetic elements contributing to vector competence for arboviruses.

These studies have both basic science and applied importance. Unravelling the genetic components of vector competence means investigating the co-evolutionary processes between arboviruses and vectors, with the potential to identify factors that may be co-opted for genetic-based vector control strategies or identify steps in the transition from ISVs to arbovirus capable of infecting vertebraes. This should be possible in light of the fact that some ISVs are phylogeneticly ancestral to arboviruses in the same virus family (Marklewitz et al., 2015). Additionally, a better knowledge of the variability and interaction between mosquitoes and their microbiota could lead to novel vector control methods based on native and introduced mosquito symbionts (i.e. *Asaia* and *Wolbachia* spp.) (Ritchie et al., 2018).

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References

Agha SB, Chepkorir E, Mulwa F, Tigoi C, Arum S, Guarido MM, et al., 2017. Vector competence of populations of *Aedes aegypti* from three distinct cities in Kenya for chikungunya virus. PLoS Negl. Trop. Dis 11, e0005860. [PubMed: 28820881]

Alto BW, Smartt CT, Shin D, Bettinardi D, Malicoate J, Anderson SL, et al., 2014. Susceptibility of Florida *Aedes aegypti* and *Aedes albopictus* to dengue viruses from Puerto Rico. J. Vec. Ecol 39, 406–413.

Alto BW, Wiggins K, Eastmond B, Velez D, Lounibos LP, et al., 2017. Transmission risk of two chikungunya lineages by invasive mosquito vectors from Florida and the Dominican Republic. PLoS Neg. Trop. Dis 11, e005724.

Bancroft TL, 1906. On the aetiology of dengue fever. Aust. Med. Gaz 25, 17–18.

Beck AS, Barrett AD, 2015. Current status and future prospects of yellow fever vaccines. Expert Rev. Vaccines 14, 1479–1492. [PubMed: 26366673]

Bennett K, Olson K, Muno M, Fernandez-Salas I, Farfan J, et al., 2002. Variation in vector competence for dengue-2 virus among 24 collections of *Aedes aegypti* from Mexico and the United States. Am. J. Trop. Med. Hyg 67, 84–92.

Boccolini D, Toma L, Luca M, Severini F, Romi R, Remoli ME, Sabbatucci M, Venturi G, Rezza G, Fortuna C, 2016. Euro. Surveill 21, 30328.

Bolling BG, Weaver SC, Tesh RB, Vasilakis N, 2015. Insect-specific Virus Discovery: significance for the Arbovirus Community. Viruses 7, 4911–4928. [PubMed: 26378568]

Boorman JP, Porterfield JS, 1956. A simple technique for infection of mosquitoes with viruses; transmission of Zika virus. Trans. R. Soc. Trop. Med. Hyg 50, 238–242. [PubMed: 13337908]

Boromisa RD, Rai KS, Grimstad PR, 1987. Variation in the vector competence of geographic strains of the *Aedes albopictus* for Dengue 1 virus. J. Am. Mos. Cont. Ass 3, 378–386.

Bosio CF, Beaty BJ, Black WC, 1998. Quantitative genetics of vector competence for dengue-2 virus in *Aedes aegypti*. Am. J. Trop. Med. Hyg 59, 965–970. [PubMed: 9886207]

Buckner EA, Alto BW, Lounibus PL, 2013. Vertical Transmission of Key West Dengue-1 Virus by *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) Mosquitoes from Florida. J. Med. Entomol 31, 1713–1723.

Calvez E, Guillaumot L, Girault D, Richard V, O'Connor O, Paoaafaite T, et al., 2017. Dengue-1 virus and vector competence of *Aedes aegypti* (Diptera: Culicidae) populations from New Caledonia. Parasit. Vectors 10, 381. [PubMed: 28793920]

Calvez E, Mousson L, Vazeille M, O'Connor O, Cao-Lormeau VM, Mathieu-Daudé F, Pocquet N, Failloux AB, Dupont-Rouzeyrol M, 2018. Zika virus outbreak in the Pacific: Vector competence of regional vectors. PLoS Negl. Trop. Dis 12, e0006637. [PubMed: 30016372]

Carrington LB, Seifert SN, Armijos MV, Lambrechts L, Scott TW, 2013. Reduction of *Aedes aegypti* vector competence for dengue virus under large temperature fluctuations. Am J Trop Med Hyg 88, 689–697. [PubMed: 23438766]

Carvalho-Leandro D, Ayres CFJ, Guedes DRD, Suesdek L, Melo-Santos MAV, Oliveira CF, et al., 2012. Immune transcript variations among *Aedes aegypti* populations with distinct susceptibility to dengue virus serotype 2. Acta Trop. 124, 113–119. [PubMed: 22877626]

Charan SS, Pawar KD, Severson DW, Patole MS, Shouche YS, 2013. Comparative analysis of midgut bacterial communities of *Aedes aegypti* mosquito strains varying in vector competence to dengue virus. Parasitol. Res 112, 2627–2637. [PubMed: 23636307]

Chen WJ, Wei HL, Hsu EL, Chen ER, 1993. Vector competence of *Aedes albopictus* and *Ae. aegypti* (Diptera: Culicidae) to dengue 1 virus on Taiwan: development of the virus in orally and parenterally infected mosquitoes. J. Med. Entomol 30, 524–530. [PubMed: 8510112]

Chen XG, Jiang X, Gu J, Xu M, Wu Y, Deng Y, Zhang C, Bonizzoni M, Dermauw W, Vontas J, Armbruster P, Huang X, Yang Y, Zhang H, He W, Peng H, Liu Y, Wu K, Chen J, Liraki M, Topalis P, Van Leeuwen T, Hall AB, Jiang X, Thorpe C, Mueller RL, Sun C, Waterhouse RM, Yan G, Tu ZJ, Fang X, James AA, 2015. Genome sequence of the Asian Tiger mosquito, *Aedes albopictus*, reveals insights into its biology, genetics, and evolution. Proc. Natl. Acad. Sci. U. S. A 112, E5907–E5915. [PubMed: 26483478]

Chepkorir E, Lutomiah J, Mutisya J, Mulwa F, Orindi B, et al., 2014. The vector competence of *Ae. aegypti* mosquito populations from Kilifi and Nairobi for dengue-2 virus and the effect of temperature. Inter J Infect Dis 21, 3–4.

Chouin-Carneiro T, Vega-Rua A, Vazeille M, et al., 2016. Differential susceptibilities of *Aedes aegypti* and *Aedes albopictus* from the Americas to Zika virus. PLoS Negl. Trop. Dis 10.

Chow VT, Chan YC, Yong R, Lee KM, Lim LK, Chung YK, Lam-Phua SG, Tan BT, 1998. Monitoring of dengue viruses in field-caught *Aedes aegypti* and *Aedes albopictus* mosquitoes by a type-specific polymerase chain reaction and cycle sequencing. Am J Trop Med Hyg 58, 578–586. [PubMed: 9598444]

Ciota AT, Bialosuknia SM, Zink SD, et al., 2017. Effects of Zika virus strain and *Aedes* mosquito species on vector competence. Emerg. Infect. Dis 23, 1110–1117. [PubMed: 28430564]

Cleton N, Koopmans M, Reimerink J, Godeke GJ, Reusken C, 2012. Come fly with me: review of clinically important arboviruses for global travelers. J. Clin. Virol 55, 191–203. [PubMed: 22840968]

Cook S, Bennett SN, Holmes EC, De Chesse R, Moureau G, de Lamballeri X, 2006. Isolation of a new strain of the flavivirus cell fusing agent virus in a natural mosquito population from Puerto Rico. J Gen Virol 87, 735–748. [PubMed: 16528021]

Coon KL, Vogel KJ, Brown MR, Strand MR, 2014. Mosquitoes rely on their gut microbiota for development. Mol. Ecol 23, 2727–2739. [PubMed: 24766707]

Coon KL, Brown MR, Strand MR, 2016. Mosquitoes host communities of bacteria that are essential for development but vary greatly between local habitats. Mol. Ecol 25, 5806–5826. [PubMed: 27718295]

Cornet M, Robin Y, 1979. Transmission experimentale comparee du virus Zika chez *Aedes aegypti*. Ent Med Parasitol 17, 47–53.

Costa-Da-Silva AL, Ioshino RS, De Araújo HRC, Kojin BB, De Andrade Zanotto PM, et al., 2017. Laboratory strains of *Aedes aegypti* are competent to Brazilian Zika virus. PLoS One 12, 1–13.

Couto-Lima D, Madec Y, Bersot MI, Campos SS, Motta MDA, Dos Santos FB, et al., 2017. Potential risk of re-emergence of urban transmission of Yellow fever virus in Brazil facilitated by competent *Aedes* populations. Sci. Rep 7, 1–12. [PubMed: 28127051]

da Moura AJF, De Melo Santos MAV, Oliveira CMF, Guedes DRD, De Carvalho-Leandro D, et al., 2015. Vector competence of the *Aedes aegypti* population from Santiago island, Cape Verde, to different serotypes of dengue virus. Parasit. Vectors 8, 1–9. [PubMed: 25561160]

Dennison NJ, Jupatanakul N, Dimopoulos G, 2014. The mosquito microbiota influences vector competence for human pathogens. Curr Opin Insect Sci 3, 6–13. [PubMed: 25584199]

Di Luca M, Severini F, Toma L, et al., 2016. Experimental studies of susceptibility of Italian *Aedes albopictus* to Zika virus. Euro Surveill. 21.

Diagne CT, Diallo D, Faye O, et al., 2015. Potential of selected Senegalese *Aedes* spp. mosquitoes (Diptera: Culicidae) to transmit Zika virus. BMC Infect. Dis. 15, 492. [PubMed: 26527535]

Diallo M, Ba Y, Faye O, Soumare ML, Dia I, Sall AA, 2008. Vector competence of *Aedes aegypti* populations from Senegal for sylvatic and epidemic dengue 2 virus isolated in West Africa. Trans of Royal Soc Trop Med Hyg 102, 493–498.

Dickson LB, Sanchez-Vargas I, Sylla M, Fleming K, Black WC, 2014. Vector Competence in West African *Aedes aegypti* is Flavivirus Species and Genotype Dependent. PLoS Negl. Trop. Dis 8, e3153. [PubMed: 25275366]

Dickson LB, Jiolle D, Minard G, Moltini-Conclois I, Volant S, Ghozlane A, Bouchier C, Ayala D, Paupy C, Valiente Moro C, Lambrechts L, 2017. Carryover effects of larval exposure to different environmental bacteria drive adult trait variation in a mosquito vector. Sci. Adv 3, e1700585. [PubMed: 28835919]

Dodson BL, Pujhari S, Rasgon JL, 2018. Vector competence of selected north American *Anopheles* and *Culex* mosquitoes for Zika virus. PeerJ 6, e4324. [PubMed: 29472998]

Dubrulle M, Mousson L, Moutailler S, Vazeille M, Failloux AB, 2009. Chikungunya Virus and *Aedes* Mosquitoes: Saliva is Infectious as soon as two days after Oral Infection. PLoS One 4, e5895. [PubMed: 19521520]

Duguma D, Hall MW, Rugman-Jones P, Stouthamer R, Terenius O, Neufeld JD, Walton WE, 2015. Developmental succession of the microbiome of Culex mosquitoes. BMC Microbiol. 15, 140. [PubMed: 26205080]

Dupont-Rouzeyrol M, Caro V, Guillaumot L, Vazeille M, D'Ortenzio E, et al., 2012. Chikungunya Virus and the Mosquito Vector *Aedes aegypti* in New Caledonia (South Pacific Region). Vector-Borne Zoon Dis 12, 1036–1041.

Dutra HL, Rocha MN, Dias FB, Mansur SB, Caragata EP, et al., 2016. Wolbachia blocks currently circulating Zika virus isolates in Brazilian *Aedes aegypti* mosquitoes. Cell Host Microbe 19, 771–774. [PubMed: 27156023]

Ellis BR, Sang RC, Horne KM, Higgs S, Wesso DM, 2012. Yellow fever virus susceptibility of two mosquito vectors from Kenya, East Africa. Trans Royal Soc Trop Med Hyg 106, 387–389.

Fansiri T, Pongsiri A, Klungthong C, Ponlawat A, Thaisomboonsuk B, Jarman RG, et al., 2016. No evidence for local adaptation of dengue viruses to mosquito vector populations in Thailand. Evol. Appl 9, 608–618. [PubMed: 27099625]

Fernandes RS, Campos SS, Ribeiro PS, Raphael LM, Bonaldo MC, Lourenço-De-Oliveira R, 2017. *Culex quinquefasciatus* from areas with the highest incidence of microcephaly associated with Zika virus infections in the Northeast Region of Brazil are refractory to the virus. Mem. Inst. Oswaldo Cruz 112, 577–579. [PubMed: 28767975]

Fish D, 2008. Why we do not understand the ecological connections between the environment and human health: the case for vector-borne disease. Vector Borne Dis 2008, 65–69.

Franz AWE, Kantor AM, Passarelli AL, Clem RJ, 2015. Tissues barriers to arbovirus infection in mosquitoes. Viruses 7, 3741–3767. [PubMed: 26184281]

Garcia-Luna SM, Weger-Lucarelli J, Rückert C, Murrieta RA, Young MC, Byas AD, Fauver JR, Perera R, Flores-Suarez AE, Ponce-Garcia G, Rodriguez AD, Ebel GD, Black WC 4th, 2018. Variation in competence for ZIKV transmission by *Aedes aegypti* and *Aedes albopictus* in Mexico. PLoS Negl. Trop. Dis 12, e0006599. [PubMed: 29965958]

Gaye A, Faye O, Diagne CT, Faye O, Diallo D, Weaver SC, et al., 2014. Oral susceptibility of *Aedes aegypti* (Diptera: Culicidae) from Senegal for dengue serotypes 1 and 3 viruses. Tropical Med. Int. Health 19, 1355–1359.

Gimonneau G, Tchioffo MT, Abate L, Boissière A, Awono-Ambene PH, Nsango SE, Christen R, Morlais I, 2014. Composition of *Anopheles coluzzii* and *Anopheles gambiae* microbiota from larval to adult stages. Infect. Genet. Evol 28, 715–724. [PubMed: 25283802]

Girod R, Gaborit P, Marrama L, Etienne M, Ramdini C, et al., 2011. Viewpoint: High susceptibility to Chikungunya virus of *Aedes aegypti* from the French West Indies and French Guiana. Tropical Med. Int. Health 16, 134–139.

Gloria-Soria A, Ayala D, Bheecarry A, Calderon-Arguedas O, Chadee DD, Chiappero M, Coetzee M, Bin Elahee KB, Fernandez-Salas I, Kamal HA, Kamgang B, Khater EI, Kramer LD, Kramer V, Lopez-Solis A, Lutomiah J, Martins A Jr., Micieli MV, Paupy C, Ponlawat A, Rahola N, Rasheed SB, Richardson JB, Saleh AA, Sanchez-Casas RM, Seixas G, Sousa CA, Tabachnick WJ, Troyo A, Powell JR, 2016. Global genetic diversity of Aedes aegypti. Mol. Ecol 25:5377–5395. [PubMed: 27671732]

Gloria-Soria A, Armstrong PM, Powell JR, Turner PE, 2017. Infection rate of *Aedes aegypti* mosquitoes with dengue virus depends on the interaction between temperature and mosquito genotype. Proc. Biol. Sci 284 (1864).

Göertz GP, Vogels CBF, Geertsema C, Koenraadt CJM, Pijlman GP, 2017. Mosquito co-infection with Zika and chikungunya virus allows simultaneous transmission without affecting vector competence of *Aedes aegypti*. PLoS Negl Trop Dis 11, 1–22.

Gonçalves CM, Melo FF, Bezerra JMT, Chaves BA, Silva BM, Silva LD, et al., 2014. Distinct variation in vector competence among nine field populations of *Aedes aegypti* from a Brazilian dengue-endemic risk city. Parasit. Vectors 7, 1–8. [PubMed: 24411014]

Gould EA, Higgs S, 2009. Impact of climate change and other factors on emerging arbovirus diseases. Trans. R. Soc. Trop. Med. Hyg 103, 109–121. [PubMed: 18799177]

Guedes DR, Paiva MH, Donato MM, et al., 2017. Zika virus replication in the mosquito *Culex quinquefasciatus* in Brazil. Emerg Microbes Infect (6), 69.

Guégan M, Zouache K, Démichel C, Minard G, Potier P, Mavingui P, Moro CV, 2018. The mosquito holobiont: fresh insight into mosquito-microbiota interactions. Microbiome 6, 49. [PubMed: 29554951]

Guo XX, Zhu XJ, Li CX, Dong Y, De Zhang YM, Xing D, et al., 2013. Vector competence of *Aedes albopictus* and *Aedes aegypti* (Diptera: Culicidae) for DEN2–43 and New Guinea C virus strains of dengue 2 virus. Acta Trop. 128, 566–570. [PubMed: 23962388]

Hall RA, Bielefeldt-Ohmann H, McLean BJ, O'Brien CA, Colmant AM, Piyasena TB, Harrison JJ, Newton ND, Barnard RT, Prow NA, Deerain JM, Mah MG, Hobson-Peters J, 2017. Commensal Viruses of Mosquitoes: Host Restriction, Transmission, and Interaction with Arboviral Pathogens. Evol. Bioinformatics Online 12, 35–44.

Hall-Mendelin S, Pyke AT, Moore PR, et al., 2016. Assessment of local mosquito species incriminates *Aedes aegypti* as the potential vector of Zika virus in Australia. PLoS Negl. Trop. Dis 10, e004959.

Hedge S, Rasgon JL, Huges GL, 2015. The microbiome modulates arbovirus transmission in mosquitoes. Curr Opin Virol 15, 97–102. [PubMed: 26363996]

Heitmann A, Jansen S, Luhken R, et al., 2017. Experimental transmission of Zika virus by mosquitoes from Central Europe. Euro Surveill. 22.

Huber K, Le Loan L, Hoang TH, Tien TK, Rodhain F, Failloux AB, 2003. *Aedes aegypti* in South Vietnam: Ecology, genetic structure, vectorial competence and resistance to insecticides. Southeast Asian J Trop Med Public Health 34, 81–86.

Johnson BW, Chambers TV, Crabtree MB, Filippis AMB, Vilarinhos PTR, et al., 2002. Vector competence of Brazilian yellow fever virus isolate *Aedes aegypti* and *Ae. abopictus* for a Brazilian yellow fever virus isolate. Trans Royal Soc Trop Med Hyg 611–613.

Jupp PG, Kemp A, 1993. The potential for dengue in South Africa: Vector competence tests with dengue 1 and 2 viruses and 6 mosquito species. Trans Royal Soc Trop Med Hyg 87, 639–643.

Kauffman EB, Kramer LD, 2017. Zika Virus Mosquito Vectors: Competence, Biology, and Vector Control. J. Infect. Dis 216, 976–990.

Kay BH, Carley JG, Fanning ID, Fillipic C, 1979. Quantitative studies of the vector competence of *Aedes aegypti*, *Culex annulirostris* and other mosquitoes (Diptera: Culicidae) with Murray Valley. J. Med. Entomol 16, 59–60. [PubMed: 42801]

Kenney JL, Romo H, Duggal NK, et al., 2017. Transmission incompetence of *Culex quinquefasciatus* and *Culex pipiens pipiens* from North America for Zika virus. Am J Trop Med Hyg. 96, 1235–1240. [PubMed: 28500817]

Knox TB, Kay BH, Hall R, Ryan PA, 2003. Enhanced vector competence of *Aedes aegypti* (Diptera: Culicidae) from the Torres Strait compared with mainland Australia for dengue 2 and 4 viruses. J. Med. Entomol 40, 950–956. [PubMed: 14765675]

Kramer LD, Scherer WF, 1976. Vector competence of mosquitoes as a marker to distinguish central American and Mexican epizootic from enzootic strains of Venezuelan enceph. Am J Trop Med Hyg. 25, 336–346. [PubMed: 1259093]

Kumar S, Molina-Cruz A, Gupta L, Rodrigues J, Barillas-Mury C, 2010. A peroxidase/dual oxidase system modulates midgut epithelial immunity in *Anopheles gambiae*. Science 327, 1644–1648. [PubMed: 20223948]

Lambrechts L, Chevillon C, Albright RG, Thaisomboonsuk B, Richardson JH, et al., 2009. Genetic specificity and potential for local adaptation between dengue viruses and mosquito vectors. BMC Evol. Biol 9, 160. [PubMed: 19589156]

Le Flohic G, Porphyre V, Barbazan P, Gonzalez JP, 2013. Review of climate, landscape, and viral genetics as drivers of the Japanese encephalitis virus, ecology. PLoS Negl. Trop. Dis 7, e2208. [PubMed: 24069463]

Li MI, Wong PS, Ng LC, Tan CH, 2012. Oral susceptibility of Singapore *Aedes* (Stegomyia) *aegypti* (Linnaeus) to Zika virus. PLoS Negl. Trop. Dis 6, e1792. [PubMed: 22953014]

Li CX, Guo XX, Deng YQ, Xing D, Sun AJ, Liu QM, Wu Q, Dong YD, Zhang YM, Zhang HD, Cao WC, Qin CF, Zhao TY, 2017. Vector competence and transovarial transmission of two *Aedes aegypti* strains to Zika virus. Emerg Microbes Infect 6, e23. [PubMed: 28442754]

Long KC, Ziegler SA, Thangamani S, Hausser NL, Kochel TJ, et al., 2011. Experimental transmission of Mayaro virus by *Aedes aegypti*. Am J Trop Med Hyg. 85, 750–757. [PubMed: 21976583]

Lourenco-De-Oliveira R, Vazeille M, Bispo De Filippis AM, Failloux AB, 2002. Oral susceptibility to yellow fever virus of *Aedes aegypti* from Brazil. Mem. Inst. Oswaldo Cruz 97, 437–439. [PubMed: 12048581]

Lourenco-De-Oliveira R, Vazeille M, de Filippis AM, Failloux AB, 2004. *Aedes aegypti* in Brazil: genetically differentiated populations with highsusceptibility to dengue and yellow fever viruses. Trans Royal Soc Trop Med Hyg. 98, 43–44.

Lourenço-De-Oliveira R, Rua AV, Vezzani D, Willat G, Vazeille M, et al., 2013. *Aedes aegypti* from temperate regions of South America are highly competent to transmit dengue virus. BMC Infect. Dis 13, 1–8. [PubMed: 23280237]

Main BJ, Nicholson J, Winokur OC, Steiner C, Riemersma KK, Stuart J, Takeshita R, Krasnec M, Barker CM, Coffey LL, 2018. Vector competence of *Aedes aegypti, Culex tarsalis*, and *Culex quinquefasciatus* from California for Zika virus. PLoS Negl. Trop. Dis 12, e0006524. [PubMed: 29927940]

Marklewitz M, Zirkel F, Kurth A, Drosten C, Junglen S, 2015. Evolutionary and phenotypic analysis of live virus isolates suggests arthropod origin of a pathogenic RNA virus family. Proc. Natl. Acad. Sci. U. S. A 112, 7536–7541. [PubMed: 26038576]

Mbaika S, Lutomiah J, Chepkorir E, Mulwa F, Khayeka-Wandabwa C, et al., 2016. Vector competence of *Aedes aegypti* in transmitting Chikungunya virus: Effects and implications of extrinsic incubation temperature on dissemination and infection rates. Virol. J 13, 1–9. [PubMed: 26728778]

Medeiros AS, Costa DMP, Branco MSD, Sousa DMC, Monteiro JD, Galvao SPM, Azevedo PR, Fernandes JV, Araujo JMG, 2018. Dengue virus in *Aedes aegypti* and *Aedes albopictus* in urban areas in the state of Rio Granse do Norte, Brazil: Importance of virological and entomological surveillance. PLoS One 13, e0194108. [PubMed: 29534105]

Minard G, Tran FH, Van VT, Goubert C, Bellet C, Lambert G, Kim KLH, Thuy THT, Mavingui P, Valiente Moro C, 2015. French invasive Asian tiger mosquito populations harbor reduced bacterial microbiota and genetic diversity compared to Vietnamese autochthonous relatives. Front. Microbiol 6, e2836–e2845.

Mitchell CJ, Miller BR, Gubler DJ, 1987. Vector competence of *Aedes albopictus* from Houston, Texas, for dengue serotypes 1to 4, yellow fever and Ross River viruses. J Am Mosquito Cont Ass 3, 460–465.

Morrison TE, Diamond MS, 2017. Animal models of Zika virus infection pathogenesis and immunity. J Virol 91, e00009–e00017. [PubMed: 28148798]

Muturi EJ, Buckner E, Bara J, 2017. Superinfection interference between dengue-2 and dengue-4 viruses in *Aedes aegypti* mosquitoes. Tropical Med. Int. Health 22, 399–406.

Na W, Yeom M, Choi IK, Yook H, Song D, 2017. Animal models for dengue vaccine development and testing. Clin Exp Vaccine Res 6, 104–110. [PubMed: 28775974]

Nasar F, Haddow AD, Tesh RB, Weaver SC, 2014. Eilat virus displays a narrow mosquito vector range. Parasit. Vectors 7, 595. [PubMed: 25515341]

Nasar F, Erasmus JH, Haddow AD, Tesh RB, Weaver SC, 2015. Eilat virus induces both homologous and heterologous interference. Virology 484, 51–58. [PubMed: 26068885]

Ngoagouni C, Kamgang B, Kazanji M, Paupy C, Nakouné E, 2017. Potential of *Aedes aegypti* and *Aedes albopictus* populations in the Central African Republic to transmit enzootic chikungunya virus strains. Parasit. Vectors 10, 164. [PubMed: 28347325]

Oliveira JHM, Gonçalves RLS, Lara FA, Dias FA, Gandara ACP, Menna-Barreto RFS, Edwards MC, Laurindo FRM, Silva-Neto MAC, Sorgine MHF, Oliveira PL, 2011. Blood meal-derived heme decreases ROS levels in the midgut of Aedes aegypti and allows proliferation of intestinal microbiota. PLoS Pathog. 7, e1001320. [PubMed: 21445237]

Olson KE, Bonizzoni M, 2017. Nonretroviral integrated RNA viruses in arthropod vectors: an occasional event or something more? Curr Opin Insect Sci. 22, 45–53. [PubMed: 28805638]

Padilha KP, Resck MEB, Cunha OATD, Teles-de-Freitas R, Campos SS, Sorgine MHF, Lourenco-de-Oliveira R, Farnesi LC, Bruno RV, 2018. Zika infection decreases Aedes aegypti locomotor activity but does not influence egg production or viability. Mem. Inst. Oswaldo Cruz 113, e180290. [PubMed: 30156598]

Palatini U, Miesen P, Carballar-Lejarazu R, Ometto L, Rizzo E, Tu Z, van Rij RP, Bonizzoni M, 2017. Comparative genomics shows that viral integrations are abundant and express piRNAs in the arboviral vectors *Aedes aegypti* and *Aedes albopictus*. BMC Genomics 18, 512. [PubMed: 28676109]

Palmer WH, Varghese F, van Rij R, 2018. Natural variation in resistance to virus infection in Dipteran insects. Viruses 10, 118.

Pando-Robles V, Batista CV, 2017. *Aedes*-borne virus-mosquito interactions: mass spectrometry strategies and findings. Vector-borne Zoon Dis 17, 361–375.

Paupy C, Chantha N, Vazeille M, Reynes JM, Rodhain F, et al., 2003. Variation over space and time of *Aedes aegypti* in Phnom Penh (Cambodia): genetic structure and oral susceptibility to a dengue virus. Gen Res 82, 171–182.

Pérez-Castro R, Castellanos JE, Olano VA, Matiz MI, Jaramillo JF, Vargas SL, Sarmiento DM, Stenstrom TA, Overgaard HJ, 2016. Detection of all four dengue serotypes in *Aedes aegypti* female mosquitoes collected in a rural area in Colombia. Mem. Inst. Oswaldo Cruz 111, 233–240. [PubMed: 27074252]

Pesko K, Westbrook CJ, Mores CN, Lounibos LP, Reiskin MH, 2009. Effects of Infectious Virus Dose and Bloodmeal delivery Method on Susceptibility of *Aedes aegypti* and *Aedes albopictus* to Chikungunya Virus. J. Med. Entomol 46, 395–399. [PubMed: 19351094]

Pham Thi KL, Briant L, Gavotte L, Labbe L, Perriat-Sanguinet M, et al., 2017. Incidence of dengue and chikungunya viruses in mosquitoes and human patients in border provinces of Vientnam. Parasit. Vectors 10, 556. [PubMed: 29121985]

Pike A, Dong Y, Dizaji NB, Gacita A, Mongodin EF, Dimopoulos G, 2017. Changes in the microbiota cause genetically modified Anopheles to spread in a population. Science 357, 1396–1399. [PubMed: 28963254]

Pongsiri A, Ponlawat A, Thaisomboonsuk B, Jarman RG, Scott TW, et al., 2014. Differential susceptibility of two field *Aedes aegypti* populations to a low infectious dose of dengue virus. PLoS One 9, 3–8.

Poole-Smith BK, Hemme RR, Delorey M, Felix G, Gonzalez AL, et al., 2015. Comparison of Vector Competence of *Aedes mediovittatus* and *Aedes aegypti* for Dengue Virus: Implications for Dengue Control in the Caribbean. PLoS Negl. Trop. Dis 9, 1–11.

Powell JR, 2018. Mosquito-Borne Human Viral Diseases: why *Aedes aegypti*? Am J Trop Med Hyg. 98, 1563–1565. [PubMed: 29557341]

Ramirez JL, Souza-Neto J, Torres Cosme R, Rovira J, Ortiz A, Pascale JM, Dimopoulos G, 2012. Reciprocal tripartite interactions between the *Aedes aegypti* midgut microbiota, innate immune system and dengue virus influences vector competence. PLoS Negl. Trop. Dis 6, e1561. [PubMed: 22413032]

Ramirez JL, Short SM, Bahia AC, Saraiva RG, Dong Y, Kang S, Tripathi A, Mlambo G, Dimopoulos G, 2014. Chromobacterium Csp_P reduces malaria and dengue infection in vector mosquitoes and has entomopathogenic and in vitro antipathogen activities. PLoS Pathog. 10, e1004398. [PubMed: 25340821]

Reed W, Carroll J, 1901. The prevention of yellow fever. Public Health Pap Rep 27, 113–129. [PubMed: 19600973]

Richard V, Paoaafaite T, Cao-Lormeau VM, 2016a. Vector Competence of French Polynesian *Aedes aegypti* and *Aedes polynesiensis* for Zika Virus. PLoS Negl. Trop. Dis 10, e0005024. [PubMed: 27654962]

Richard V, Paoaafaite T, Cao-Lormeau VM, 2016b. Vector Competence of *Aedes aegypti* and *Aedes polynesiensis* Populations from French Polynesia for Chikungunya Virus. PLoS Negl. Trop. Dis 10, e0004694. [PubMed: 27144888]

Richards SL, Anderson SL, Alto BW, 2012. Vector competence of *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) fordengue virus in the Florida Keys. J. Med. Entomol 49, 942–946. [PubMed: 22897056]

Ritchie SA, van den Hurk AF, Smout MJ, Staunton KM, Hoffmann AA, 2018. Mission Accomplished? We need a Guide to the 'Post Release' World of Wolbachia for *Aedes*-borne Disease Control. Trends Parasitol. 34, 217–226. [PubMed: 29396201]

Rodgers FH, Gendrin M, Wyer CAS, Christophides GK, 2017. Microbiota-induced peritrophic matrix regulates midgut homeostasis and prevents systemic infection of malaria vector mosquitoes. PLoS Pathog. 13, e1006391–e1006392. [PubMed: 28545061]

Rosen L, Roseboom LE, Gubler DJ, Lien JC, Chaniotis BN, 1985. Comparative susceptibility of mosquito species and strains to oral and parenteral infection with dengue and Japanese encephalitis viruses. Am. J. Trop. Med. Hyg 34, 603–615. [PubMed: 2860816]

Roundy CM, Azar SR, Rossi SL, Huang JH, Leal G, Yun R, Fernandez-Salas I, Vitek CJ, Paploski IA, Kitron U, Ribeiro GS, Hanley KA, Weaver SC, Vasilakis N, 2017. Variation in *Aedes aegypti* Mosquito Competence for Zika Virus Transmission. Emerg. Infect. Dis 23, 625–632. [PubMed: 28287375]

Rückert C, Ebel GD, 2018. How do Virus-Mosquito Interactions Lead to Viral Emergence? Trends Parasitol. 34, 310–321. [PubMed: 29305089]

Rückert C, Weger-Lucarelli J, Garcia-Luna SM, Young MC, Byas AD, et al., 2017. Impact of simultaneous exposure to arboviruses on infection and transmission by *Aedes aegypti* mosquitoes. Nature Com 8, 1–9.

Ryckebusch F, Berthet M, Missé D, Choumet V, 2017. Infection of a French Population of *Aedes albopictus* and of *Aedes aegypti* (Paea Strain) with Zika Virus reveals Low Transmission rates to these Vectors' Saliva. J Mol Sci 18, 2384.

Saraiva RG, Kang S, Simões ML, Angleró-Rodríguez YI, Dimopoulos G, 2016. Mosquito gut antiparasitic and antiviral immunity. Dev. Comp. Immunol 64, 53–64. [PubMed: 26827888]

Saraiva RG, Fang J, Kang S, Angleró-Rodríguez YI, Dong Y, Dimopoulos G, 2018. Aminopeptidase secreted by Chromobacterium sp. Panama inhibits dengue virus infection by degrading the E protein. PLoS Negl. Trop. Dis 12, e0006443. [PubMed: 29694346]

Schneider JR, Mori A, Romero-Severson J, Chadee DD, Severson DW, 2007. Investigations of dengue-2 susceptibility and body size among *Aedes aegypti* populations. Med. Vet. Entomol 21, 370–376. [PubMed: 18092975]

Serrato IM, Caicedo PA, Orobio Y, Lowenberger C, Ocampo CB, 2017. Vector competence and innate immune responses to dengue virus infection in selected laboratory and field-collected Stegomyia aegypti (= *Aedes aegypti*). Med. Vet. Entomol 31, 312–319. [PubMed: 28407282]

Short SM, van Tol S, MacLeod HJ, Dimopoulos G, 2018. Hydrogen cyanide produced by the soil bacterium Chromobacterium sp. Panama contributes to mortality in *Anopheles gambiae* mosquito larvae. Sci. Rep 8, 8358. [PubMed: 29844510]

Sim S, Jupatanakul N, Ramirez JL, Kang S, Romero-Vivas CM, et al., 2013. Transcriptomic Profiling of Diverse *Aedes aegypti* Strains reveals increased Basal-level Immune Activation in Dengue Virus-refractory Populations and Identifies Novel Virus-vector Molecular Interactions. PLoS Negl. Trop. Dis 7, e2295. [PubMed: 23861987]

Smith DL, Battle KE, Hay SI, Barker CM, Scott TW, McKenzie FE, 2012. Ross, Macdonald, and a theory for the dynamics and control of mosquito-transmitted pathogens. PLoS Pathog. 8, e1002588. [PubMed: 22496640]

Sylla M, Bosio C, Urdaneta-Marquez L, Ndiaye M, Black IVWC, 2009. Gene flow, subspecies composition, and dengue virus-2 susceptibility among *Aedes aegypti* collections in Senegal. PLoS Negl. Trop. Dis 3, e408. [PubMed: 19365540]

Tabachnick WJ, 2016. Ecological effects on arbovirus-mosquito cycles of transmission. Curr Opin Virol. 21, 124–131. [PubMed: 27693828]

Tabachnick WJ, Wallis GP, Aitken TH, Miller BR, Amato GD, et al., 1985. Oral infection of *Aedes aegypti* with yellow fever virus: geographic variation andgenetic considerations. Am J Trop Med Hyg 34, 1219–1224. [PubMed: 3834804]

Thongrungkiat S, Jirakanjanaki N, Apiwathnasorn C, Prummongkol S, Samung Y, 2003. Comparative susceptibility to oral infection with dengue viruses among local strains of *Aedes aegypti* (Diptera : Culicidae) collected at different seasons of the year. J Vector Ecol 28, 166–170. [PubMed: 14714664]

Tran KT, Vazeille-Falcoz M, Mousson L, Tran HH, Rodhain F, et al., 1999. *Aedes aegypti* in Ho Chi Minh City (Viet Nam): susceptibility to dengue 2 virus and genetic differentiation. Trans Royal Soc Trop Med Hyg. 93, 581–586.

Tsetsarkin KA, Chen R, Sherman MB, Weaver SC, 2011. Chikungunya virus: evolution and genetic determinants of emergence. Curr Opin Virol 1, 310–317. [PubMed: 21966353]

Turell MJ, Guinn MLO, Dohm DJ, Jones JW, 2001. Vector Competence of north American Mosquitoes (Diptera: Culicidae) for West Nile Virus. J. Med. Entomol 38, 130–134. [PubMed: 11296813]

Turell MJ, Lee JS, Richardson JH, Sang RC, Kioko EN, Agawo MO, Pecor J, O'Guinn ML, 2007. Vector competence of Kenyan *Culex zombaensis* and *Culex quinquefasciatus* mosquitoes for Rift Valley fever virus. J. Am. Mosq. Control Assoc 23, 378–382. [PubMed: 18240513]

Van Den Hurk AF, McElroy K, Pyke AT, McGee CE, Hall-Mendelin S, et al., 2011. Vector competence of Australian mosquitoes for yellow fever virus. Am J Trop Med Hyg 85, 446–451. [PubMed: 21896802]

Vasilakis N, Tesh RB, 2015. Insect-specific viruses and their potential impact on arbovirus transmission. Curr Opin Virol 15, 69–74. [PubMed: 26322695]

Vazeille M, Mousson L, Rakatoarivony I, Villeret R, Rodhain F, et al., 2001. Population genetic structure and competence as a vector for dengue type 2 virus of *Aedes aegypti* and *Aedes albopictus* from Madagascar. Am J Trop Med Hyg 65, 491–497. [PubMed: 11716103]

Vazeille M, Gaborit P, Mousson L, Girod R, Failloux AB, 2016. Competitive advantage of a dengue 4 virus when co-infecting the mosquito *Aedes aegypti* with a dengue 1 virus. BMC Infect. Dis 16, 1–7. [PubMed: 26729246]

Vega-Rua A, Zouache K, Girod R, Failloux AB, Lourenco-De-Oliveira R, 2014. High Level of Vector Competence of *Aedes aegypti* and *Aedes albopictus* from ten American Countries as a crucial factor in the Spread of Chikungunya Virus. J. Virol 88, 6294–6306. [PubMed: 24672026]

Villegas LEM, Campolina TB, Barnabe NR, Orfanó AS, Chaves BA, Norris DE, et al., 2018. Zika virus infection modulates the bacterial diversity associated with Aedes aegypti as revealed by metagenomic analysis. PLoS One 13, e0190352–e0190366. [PubMed: 29293631]

Wallis GP, Aitken TH, Beaty BJ, Lorenz L, Amato GD, et al., 1985. Selection for susceptibility and refractoriness of *Aedes aegypti* to oralinfection with yellow fever vims. Am J Trop Med Hyg 34, 1225–1231. [PubMed: 3834805]

Wang Z, Zhang X, Zhang C, Xing Y, Wu DY, et al., 2012. Vector Competence of five Common Mosquito Species in the People's Republic of China for Western Equine Encephalitis Virus. Vector-Borne Zoonotic Dis 12, 605–608. [PubMed: 22276651]

Wang YH, Chang MM, Wang XL, Zheng AH, Zou Z, 2017. The immune strategies of mosquito *Aedes aegypti* against microbial infection. Dev. Comp. Immunol 1–10.

Watson TM, Kay BH, 1999. Vector competence of *Aedes notoscriptus* (Diptera: Culicidae) for Barmah Forest virus and of this species and *Aedes aegypti* (Diptera: Culicidae) for dengue 1–4 viruses in Queensland, Australia. J. Med. Entomol 36, 508–514. [PubMed: 10467781]

Weaver SC, 2006. Evolutionary influences in arboviral disease. Curr Topics Microbiol Immunol 299, 285–314.

Weger-Lucarelli J, Rückert C, Chotiwan N, Nguyen C, Garcia Luna SM, Fauver JR, Foy BD, Perera R, Black WC, Kading RC, Ebel GD, 2016. Vector Competence of American Mosquitoes for three Strains of Zika Virus. PLoS Negl. Trop. Dis 10, e0005101. [PubMed: 27783679]

Whitfield ZJ, Dolan PT, Kunitomi M, Tassetto M, Seetin MG, Oh S, Heiner C, Paxinos E, Andino R, 2017. The diversity, structure, and function of heritable adaptive immunity sequences in the Aedes aegypti genome. Curr Biol 27, 3511–3519. [PubMed: 29129531]

Wiggins K, Eastmond B, Alto BW, 2018. Transmission potential of Mayaro virus in Florida Aedes aegypti and Aedes albopictus mosquitoes. Med. Vet. Entomol 32, 436–442. [PubMed: 30006976]

Xi Z, Ramirez JL, Dimopoulos G, 2008. The *Aedes aegypti* toll pathway controls dengue virus infection. PLoS Pathog. 4, e1000098. [PubMed: 18604274]

Ye YH, Ng TS, Frentiu FD, Walker T, Van Den Hurk AF, et al., 2014. Comparative susceptibility of mosquito populations in North Queensland, Australia to oral infection with dengue virus. Am J Trop Med Hyg. 90, 422–430. [PubMed: 24420782]

Zompi S, Harris E, 2012. Animal models of dengue virus infection. Viruses 4, 62–82. [PubMed: 22355452]

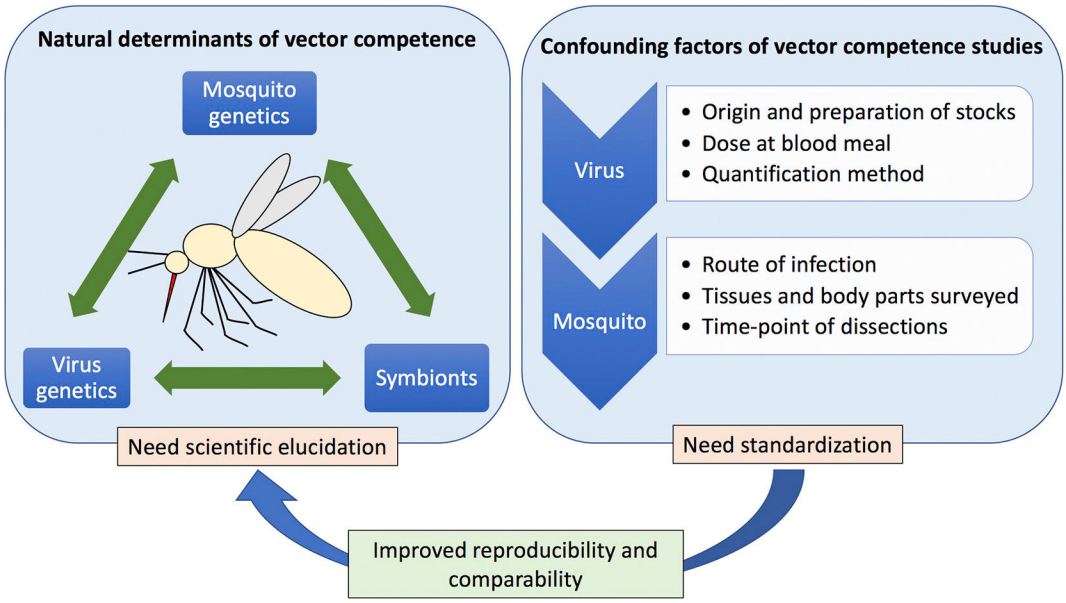

Fig. 1.

Natural and technical confounding factors related to arbovirus vector competence studies in *Aedes aegypti*. Despite progress in the understanding of the interplay between arboviruses and vectors, the genetic and environmental elements that control vector competence in *Ae. aegypti* populations have yet to be fully understood. Further elucidation is needed especially of co-evolutionary processes between arboviruses and vectors, as well as their symbionts. On the other hand, procedures used in vector competence studies should be standardized in order to improve reproducibility and comparability of scientific outputs. Together these will result in better understanding of genetic and microbial factors influencing arboviral transmission, which can lead to the development of new public health interventions.

Table 1

Summary of vector competence estimates across *Ae. aegypti* geographic populations to 1) DENVs, 2) ZIKV, 3) YFV; 4) CHIKV; 5) dual-infections and 6) infections with arboviruses other than DENVs, YFV, ZIKV and CHIKV.

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
1) DENVs				
Calvez et al., 2018	Noumea, NC	DENV-1 NC14-17022014-806	BM[2], 10^6	IR in bodies 50 at 7 dpi, 10 at 14 dpi, 8 at 21 dpi; IR in the heads 60 at 7 dpi, 100 at 14 dpi, 100 at 21 dpi; TR 3 at 7dpi, 3 at 14 dpi, 8 at 21 dpi
	Ouvea, NC	DENV-1 NC14-17022014-806	BM, 10^6	IR in bodies 53 at 7 dpi, 53 at 14 dpi, 33 at 21 dpi; IR in the heads 100 at 7 dpi, 87 at 14 dpi, 90 at 21 dpi; TR 3 at 7dpi, 13 at 14 dpi, 13 at 21 dpi
	Poindimie, NC	DENV-1 NC14-17022014-806	BM, 10^6	IR in bodies 33 at 7 dpi, 13 at 14 dpi, 17 at 21 dpi; IR in the heads 70 at 7 dpi, 100 at 14 dpi, 80 at 21 dpi; TR 0 at 7dpi, 3 at 14 dpi, 0 at 21 dpi
	Papeete, Thaiti Island	DENV-1 NC14-17022014-806	BM, 106	IR in bodies 47 at 21 dpi; IR in the heads 100 at 21 dpi; TR 3 at 7dpi, 35 at 21 dpi
Serrato et al., 2017	Valle Grande, Col	DENV-2 NG	BM, $10^{8.1}$–10^7	IR 68 at 15 dpi
	Paso del Comercio, Col	DENV-2 NG	BM, $10^{8.1}$–10^7	IR 55 at 15 dpi
	Siloe, Col	DENV-2 NG	BM, $10^{8.1}$–10^7	IR 52 at 15 dpi
	Mariano Ramos	DENV-2 NG	BM, $10^{8.1}$–10^7	IR 52 at 15 dpi
	Hanoi, Viet[8]	DENV-2 strain 6H, Hanoi Viet	BM, 2.8×10^7	IR 4.2 at 25°C; 9.1 at 27°C; 80 at 32°C
		DENV-2 strain 434S, Long An Province, Viet	BM, 3.77×10^7	IR 8.1 at 25°C; 13 at 27°C; 4.2 at 32°C
	Ho Chi Minh City, Viet	DENV-2 strain 6H, Hanoi Viet	BM, 2.8×10^7	IR 10.8 at 25°C; 2.8 at 27°C; 0 at 32°C
		DENV-2 strain 434S, Long An Province, Viet	BM, 3.77×10^7	IR 24.6 at 25°C; 9.8 at 27°C; 7.7 at 32°C
Vazeille et al., 2016[9]	Center Cayenne, FG	DENV-1 isol. from a 2009 patient living in Cayenne	BM, 10^5–10^6	IR 20 at 8 dpi, ~35 at 10 dpi, ~50 at 14 dpi; TR different from 0 only at 14 dpi, when it reached ~10
	Center Cayenne, FG	DENV-4 isol. from a 2009 patient living in Cayenne	BM, 10^5–10^6	IR ~40 at 8 dpi, ~60 at 10 dpi, ~60 at 14 dpi; TR different from 0 only at 14 dpi, when it reached ~8
	Scattered housing area, Cayenne, FG	DENV-1 isol. from a 2009 patient living in Cayenne	BM, 10^5–10^6	IR ~20 at 8 dpi, ~50 at 10 dpi, ~78 at 14 dpi; TR was always 0
	Scattered housing area, Cayenne, FG	DENV-4 isol. from a 2009 patient living in Cayenne	BM, 10^5–10^6	IR ~40 at 8 dpi, ~35 at 10 dpi, ~58 at 14 dpi; TR different from 0 only at 14dpi, when it reached ~15

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
Guo et al., 2016	Haikou strain, originally from Hainan province	DENV-2-FJ10	BM, 1.75×10^5	IR in midgut 0 up to 3 dpi; 5 from 5-7 dpi; 15 at 9 dpi, 25 at 15 dpi; IR in salivary glands 0 up to 5 dpi; 4 at 7 dpi, 15 at 9 dpi, 17 at 15 dpi
		DENV-2-FJ11	BM, 2×10^5	IR in midgut 0 up to 3 dpi; 5 at 5 dpi, 10 at 7 dpi; 25 at 9 dpi, 35 at 15 dpi; IR in salivary glands 0 up to 5 dpi; 4 at 7 dpi, 10 at 9 dpi, 25 at 15 dpi
Fansiri et al., 2016	Bangkok, Thai	14 DENV-1 Thai isol.	BM, 1.5×10^5 −8.5 10^6	IR 0 (B3 viral strain, experiment 2) - 100 (K15 and K4 viral strains experiment 1; B1, B76 and K25 viral strains experiment 2)
	Kamphaeng Phet Province, Thai	14 DENV-1 Thai isol.	BM, 1.5×10^5 −8.5 10^6	IR 0 (K1 viral strain, experiment 2) - 100 (K25 viral strain experiment 1, B76 viral strain experiment 2)
Fernandes da Moura et al., 2015	Santiago Island, Capo Verde	DENV-1 42735/BR PE	BM, 5×10^4– 2×10^5	IR 0 at 7 dpi, 74,9 at 14 dpi, 20 at 21 dpi in midguts; IR 24,3 at 7 dpi, 0 at 14 dpi, 67,5 at 21 dpi in whole body; TR 55 at 14 dpi
		DENV-2 3808/BR-PE	BM, $1,4\times10^5$– 2×10^5	IR 60 at 7 dpi, 80 at 14 dpi, 20 at 21 dpi in midguts; IR 0 at 7 dpi, 0 at 14 dpi, 92.5 at 21 dpi in whole body; TR 55 at 14 dpi
		DENV-3 85469/BR-PE	BM, 10^6	IR 12.5 at 7 dpi, 65 at 14 dpi, 75 at 21 dpi in midguts; IR 58,4 at 7dpi, 76,9 at 14 dpi, 93,8 at 21 dpi in whole body; TR 50 at 14 dpi
		DENV-4 1385 (U1842)	BM, 10^6	IR 0 at 7 dpi, 0 at 14 dpi, 9 at 21 dpi in midguts; IR 0 at 21 dpi in whole body; TR 0 at 14 dpi
Poole-Smith et al., 2015	Patillas, PR	DENV-1 Hawaii	BM, 5–6 Log10	IR 15, TR 3
		DENV-2 NG C	BM, 5–6 Log10	IR 17, TR 5
		DENV-3 H87	BM, 5–6 Log10	IR 18, TR 2
		DENV-4 H241	BM, 5–6 Log10	IR 62, TR 42
Dickson et al., 2014[10]	Fatick, S	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 61
	Bignona, S	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 29
	Richard Toll, S	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 30
	Goudiry, S	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 39
	Aedes aegypti formosus Kedougou, S, sylvatic	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 60
	Aedes aegypti formosus PK10, S, sylvatic	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 57

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Mont Rolland, S	DENV-2-75505 sylvatic genotype from S	BM, 10^7	IR 93
	Rufisque, S	DENV-2-75505 sylvatic genotype from S	BM, 1.5×10^6	IR 33
Gaye et al., 2014	Sylvatic *Aedes aegytpi formosus* from Kedoungou, S	DENV-1 IbH28328	BM[3], $5\times10^{3.3}$	IR 40 at 7 dpi, 30 at 15 dpi, 50 at 20 dpi
	Sylvatic *Ae.aegytpi formosus* from Kedoungou, S	DENV3 H87	BM[3], $5\times10^{3.3}$	IR 0 at 7 dpi, 8.3 at 15 dpi,
	Domestic *Ae.aegypti* from Dakar, S	DENV-1 IbH28328	BM[3], $5\times10^{3.3}$	IR 0 at 7 dpi, 43.7 at 15 dpi, 30.8 at 20 dpi
	Domestic *Ae. aegypti* from Dakar, S	DENV3 H87	BM[3], $5\times10^{3.3}$	IR 10 at 7 dpi, 15.2 at 15 dpi, 2.4 at 20 dpi
Alto et al., 2014	Key West, FL	DENV-1/US/BID-V852/2006	BM, 6.8±0.5 log10	IR 10 at 7 dpi and 6 at 14 dpi in midguts; 10 at 7 dpi and 88 at 14 dpi in whole body
		DENV-2/US/BID-V1041/2006	BM, 7.1±1.2 log10	IR 28 at 7 dpi, at 14 dpi, 28 at 21 dpi in midguts; IR 12 at 7 dpi, 27 at 14 dpi in whole body
Gonçalves et al., 2014[9]	Belo Horizonte, BR	DENV-2 from a hs of a patient from Belo Horizonte in 1991	BM, ntd	IR 60 and TR 58 in 2009; IR 78 and TR 55 in 2011
Pongsiri et al., 2014	Phet Province, Thai	six DENV-2 isol. from patients of the Phet Province in Thai	BM, 3.5–6 log10	IR 20.9 at 7 dpi, 31.8 at 14 dpi
Ye et al., 2014[9]	Cairns, Aus	DENV-2 92-T strain isol. during a 1992 outbreak in Townsville	BM, 10^6	IR 20-100 in midguts; 25-70 in heads
		DENV-2 ET-300 strain isol. in Timor-Leste in 2000	BM, 10^6	IR 60-100 in midguts, 38-100 in heads
	Rockhamton, Aus	DENV-2 92-T strain isol. during a 1992 outbreak in Townsville	BM, 10^6	IR 85-100 in midguts; 35-100 in heads
		DENV-2 ET-300 strain isol. in Timor-Leste in 2000	BM, 10^6	IR 80-100 in midguts; 60-100 in heads
Chepkorir et al., 2014	Nairobi, Kenya	DENV-2 from a hs (Sample N. 008/01/2012)	BM, $10^{5.08}$	mosquitoes kept at 26°C (Nairobi's average temperature) after infection, IR 12, disseminated infection 18
		DENV-2 from a hs (Sample N. 008/01/2012)	BM, $10^{5.08}$	mosquitoes kept at 30°C (Kilifi's average temperature) after infection, IR 20, disseminated infection 8
	Kifili, Kenya	DENV-2 from a hs (Sample N. 008/01/2012)	BM, $10^{5.08}$	mosquitoes kept at 26°C (Nairobi's average temperature) after infection IR 5, disseminated infection 35

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
		DENV-2 from a hs (Sample N. 008/01/2012)	BM, $10^{5.08}$	mosquitoes kept at 30°C (Kilifi's average temperature) after infection IR 10, disseminated infection 42
Guo et al., 2013	Haiku strain, Chi	DENV-2 NG C	BM[4], 7.7 log10	IR in midguts at 1 dpi is 60; TR at 15 dpi 85.7
		DENV-2 43	BM[4], 7.2 log10	IR in midguts at 1 dpi is 48.5; TR at 15 dpi 56.3
Sim et al., 2013[9]	Rockefeller strain	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 100
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 100
	Orlano strain	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 0
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 0
	Waco strain	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 15
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 10
	PR, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 30
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 25
	Saint Kitts, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 25
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 55
	Por Fin, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 28
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 10
	Puertp Triunfo, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 65
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 10
	Singapore, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 90
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 10
	Bangkok, field	DENV-2 NG C strain	BM, 10^{6-7}	IR 7 dpi in midguts, 10
		DENV4-WRAIR	BM, 10^{6-7}	IR 7 dpi in midguts, 10
Buckner et al., 2013	Key West, FL	DENV-1 (strain BOLKW010)	BM, 6.3±0.2 Log10	IR 93 in midguts, 80 in whole body
Carrington et al., 2013	Kamphaeng Phet Province, Thai	DENV-1	BM[1], 3,09–4.16×10^{5}	IR 28
Lourenço-De-Oliveira et al., 2013	Buenos Aires, Argentina	DENV-2 Thai 1974	BM, 10^{7}	IR in whole bodies 66.7 at 14 dpi and 78.1 at 21 dpi; TR 10.5 at 14 dpi and 6.7 at 21 dpi
	Corrientes, Argentina	DENV-2 Thai 1974	BM, 10^{7}	IR in whole bodies 53.3 at 14 dpi and 76.7 at 21 dpi; TR 18.5 at 14 dpi and 36.4 at 21 dpi

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Salto, Uruguay	DENV-2 Thai 1974	BM, 10^7	IR in whole bodies 53.3 at 14 dpi and 76.7 at 21 dpi; TR 20 at 14 dpi and 17.9 at 21 dpi
Richards et al., 2012	Key West, FL	DENV-1 isol. BOL-KW010	BM, 3.7 Log10	IR 89 in the abdomen, 100 in legs; TR 0 when mosquitoes were kept at 28°C
	Key West, FL	DENV-1 isol. BOL-KW010	BM, 3.7 Log10	IR 75 in the abdomen, 33 in legs; TR 0 when mosquitoes were kept at 30°C
	Stock Island, FL	DENV-1 isol. BOL-KW010	BM, 3.7 Log10	IR 75 in the abdomen, 100 in legs; TR 33 when mosquitoes were kept at 28°C
	Stock Island, FL	DENV-1 isol. BOL-KW010	BM, 3.7 Log10	IR 80 in the abdomen, 100 in legs; TR 0 when mosquitoes were kept at 30°C
Carvalho-Leandro et al., 2012[9]	Petrolina, BR	DENV-2 3808/BR-PE	BM, $10^{6–7}$	IR 25 at 3 dpi, 70 at 7 dpi, 77 at 15 dpi, 50 at 21 dpi in midguts; IR 10 at 3 dpi, 20 at 7 dpi, 58 at 15 dpi and 100 at 21 dpi in fat; TR 40 at 7 dpi, 10 at 15 dpi, 40 at 21 dpi
	Recife, BR	DENV-2 3808/BR-PE	BM, $10^{6–7}$	IR 5 at 3 dpi, 42,5 at 7 dpi, 20 at 15 dpi, 46.3 at 21 dpi in midguts; IR 0 at 3 dpi, 10 at 7 dpi, 70 at 15 dpi and 40 at 21 dpi in fat; TR 35 at 7 dpi, 60 at 15 dpi, 47.5 at 21 dpi
	Rec-L Recife Lab. strain	DENV-2 3808/BR-PE	BM, $10^{6–7}$	IR 5 at 3 dpi, 22 at 7 dpi, 20 at 15 dpi, 45 at 21 dpi in midguts; IR 0 at 3 dpi, 35 at 7 dpi, 35 at 15 dpi and 58 at 21 dpi in fat; TR 5 at 7 dpi, 20 at 15 dpi, 35 at 21 dpi
Sylla et al., 2009	D2MEB	DENV-2 JAM1409	BM, $3.1\times10^{7–8}$	IR 51.2
	D2S3	DENV-2 JAM1409	BM, $3.1\times10^{7–8}$	IR 92.3
Schneider et al., 2007	Bangkok, field	DENV-2 JaM1409	BM, ntd	IR 32.22 +/− 8.56
	DS3	DENV-2 JaM1409	BM, ntd	IR 45.95 +/− 17.76
	Form, Flavivirus refractory strC2:C83ain from Nigeria	DENV-2 JaM1409	BM, ntd	IR 48.42 +/− 6.68
	Ghana, field	DENV-2 JaM1409	BM, ntd	IR 27.44 +/− 6.03
	Ibo 11, Dengue refractory strain from Nigeria	DENV-2 JaM1409	BM, ntd	IR 31.55 +/− 2.44
	Mombasa, field	DENV-2 JaM1409	BM, ntd	IR 30.23 +/− 3.14
	MOYO-R	DENV-2 JaM1409	BM, ntd	IR 19.54 +/− 9.73
	MOYO-S, RED, mutant marker stock	DENV-2 JaM1409	BM, ntd	IR 53.60 +/− 14.16
		DENV-2 JaM1409	BM, ntd	IR 38.79 +/− 14.17
	Trinidad, field	DENV-2 JaM1409	BM, ntd	IR 34.92 +/− 29.27

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
Diallo et al., 2008[11]	Barkedji, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 7.4
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 1.74
	Dakar, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 7.8
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 0
	Ngoye, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 17.2
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 1.46
	Ndougoubene, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 9.3
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 1.57
	Kedougou, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 1.35
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 0
	Koung Koung, S	sylvatic DENV-2 AdR 140875	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 2.7
		epidemic DENV-2 ArA 6894	BM[4], 1.6×10^{7}–$10^{6.5}$	IR 1.85
Knox et al., 2003	Torres Strait, Aus	DENV-2 92T	BM[5], $10^{6.4}$	IR 96 at 8 dpi, 100 at 12 and 16 dpi; TR 0 at 8 dpi; 8 at 12 dpi, 76 at 16 dpi
		DENV-4 97B	BM[5], 10^{7}	IR 80 at 8 and 12 dpi, 84 at 16 dpi, 72 at 20 dpi; TR 0 at 8 and 12 dpi, 16 at 16 dpi, 16 at 20 dpi
	Charters Towers, Aus	DENV-2 92T	BM[5], $10^{6.4}$	IR 52 at 8 dpi, 60 at 8 dpi, 64 at 16 dpi; TR 8 at 8 dpi, 4 at 12 dpi, 24 at 16 dpi
		DENV-4 97B	BM[5], 10^{7}	IR 36 at 8 dpi, 16 at 12 dpi, 28 at 16 dpi, 32 at 20 dpi; TR 0 at 8,12 and 16 dpi, 8 at 20 dpi
	Townsville, Aus	DENV-2 92T	BM[5], $10^{6.4}$	IR 72 at 8 dpi, 90 at 8 dpi, 92 at 16 dpi; TR 0 at 8 dpi, 0 at 12 dpi, 28 at 16 dpi
		DENV-4 97B	BM[5], 10^{7}	IR 12 at 8 dpi, 28 at 12 dpi, 40 at 16 dpi, 32 at 20 dpi; TR 0 at 8, 12 and 16 dpi, 16 at 20 dpi
	Cairns, Aus	DENV-2 92T	BM[5], $10^{6.4}$	IR 80 at 8 dpi, 84 at 12 dpi, 80 at 16 dpi; 8 at 8 dpi, 4 at 12 dpi, 20 at 16 dpi
		DENV-4 97B	BM[5], 10^{7}	IR 16 at 8 dpi, 28 at 12 dpi, 36 at 16 and 20 dpi; TR 0 at 8 and 12 dpi, 4 at 16 and 20 dpi
Huber et al., 2003[12]	Ho Chi Minh City, (mosquitoes collected from 1975 to 1998)	DENV-2, strain not defined	BM, ntd	IR 94.8 +/− 3.61

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	Ho Chi Minh City (mosquitoes collected from 1975 to 1998)	DENV-2, strain not defined	BM, ntd	IR 97.7 +/− 2.39
	Paea strain, Thaiti	DENV-2, strain not defined	BM, ntd	IR 93.84 +/− 4.38
Lourenco-de-Oliveira et al., 2004	Belém, BR	DENV-2 Bangkok 1974	BM, ntd	IR 96.3
	Ananindeua, BR	DENV-2 Bangkok 1974	BM, ntd	IR 94.23
	Rio Branco, BR	DENV-2 Bangkok 1974	BM, ntd	IR 81.43
	Porto Velho	DENV-2 Bangkok 1974	BM, ntd	IR 83.19
	Boa Vista, BR	DENV-2 Bangkok 1974	BM, ntd	IR 95,75
	Salvador, BR	DENV-2 Bangkok 1974	BM, ntd	IR 81.48
	Sao Luis, BR	DENV-2 Bangkok 1974	BM, ntd	IR 97,38
	Feira de Santana, BR	DENV-2 Bangkok 1974	BM, ntd	IR 74,74
	Milha, BR	DENV-2 Bangkok 1974	BM, ntd	IR 25,79
	Pacuja, BR	DENV-2 Bangkok 1974	BM, ntd	IR 73,62
	Quixeramobin, BR	DENV-2 Bangkok 1974	BM, ntd	IR 82,10
	Represa dp Cigano, BR	DENV-2 Bangkok 1974	BM, ntd	IR 98,24
	Tingua, BR	DENV-2 Bangkok 1974	BM, ntd	IR 84,85
	Higienopolis, BR	DENV-2 Bangkok 1974	BM, ntd	IR 75,32
	Moqueta, BR	DENV-2 Bangkok 1974	BM, ntd	IR 93,40
	Rocinha, BR	DENV-2 Bangkok 1974	BM, ntd	IR 92,86
	Comendador Soares, BR	DENV-2 Bangkok 1974	BM, ntd	IR 91,15
	Cariacica, BR	DENV-2 Bangkok 1974	BM, ntd	IR 81,81
	Potim, BR	DENV-2 Bangkok 1974	BM, ntd	IR 83,62
	Leandro Ferreira, BR	DENV-2 Bangkok 1974	BM, ntd	IR 85,95
	Foz de Iguacu, BR	DENV-2 Bangkok 1974	BM, ntd	IR 62,43
	Maringa, BR	DENV-2 Bangkok 1974	BM, ntd	IR 73,6
	Campo Grande, BR	DENV-2 Bangkok 1974	BM, ntd	IR 72,73
	Paea Lab. strain	DENV-2 Bangkok 1974	BM, ntd	IR 93,34 +/− 4.63
Paupy et al., 2003[12]	Phon Penh City Center (Cambodia),	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 79,39 +/− 11,01

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	mosquitoes collected in February			
	Phon Penh City Center (Cambodia), mosquitoes collected in July	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 77,76 +/− 8,31
	Phon Penh City suburbs north (Cambodia), mosquitoes collected in February	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 90,65 +/− 8,77
	Phon Penh City suburbs west (Cambodia), mosquitoes collected in February	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 87 +/− 4,82
	Phon Penh City suburbs south (Cambodia), mosquitoes collected in February	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 95,30 +/− 0.14
	Paea strain, Thaiti	DENV-2 from a hs sample collected in Bangkok Thai in 1974	BM[3], $10^{8.2}$	IR 78.52 +/− 7.64
Thongrungkiat et al., 2003	Chiang Rai, Thai	DENV-1 16007	BM[3], $10^{8.1}$	IR 19.4
			BM[3], 10^{10}	IR 48.7
		DENV-2 16681	BM[3], $10^{8.1}$	IR 17.8
			BM[3], 10^{10}	IR 25
		DENV-3 16562	BM[3], $10^{8.1}$	IR 3.8
			BM[3], 10^{10}	IR 19.7
		DENV-4 1036	BM[3], $10^{8.1}$	IR 27.7
			BM[3], 10^{10}	IR 54.8
	Nakhon Phanom, Thai	DENV-1 16007	BM[3], $10^{8.1}$	IR 16
			BM[3], 10^{10}	IR 48.2
		DENV-2 16681	BM[3], $10^{8.1}$	IR 15
			BM[3], 10^{10}	IR 28
		DENV-3 16562	BM[3], $10^{8.1}$	IR 4.3
			BM[3], 10^{10}	IR 18.5

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
		DENV-4 1036	BM[3], $10^{8.1}$	IR 15.6
			BM[3], 10^{10}	IR 49.4
	Satun, Thai	DENV-1 16007	BM[3], $10^{8.1}$	IR 8.1
			BM[3], 10^{10}	IR 43.8
		DENV-2 16681	BM[3], $10^{8.1}$	IR 13.1
			BM[3], 10^{10}	IR 27.6
		DENV-3 16562	BM[3], $10^{8.1}$	IR 0.9
			BM[3], 10^{10}	IR 11.1
		DENV-4 1036	BM[3], $10^{8.1}$	IR 12.5
			BM[3], 10^{10}	IR 54.5
Bennett et al., 2002[9]	Hermosillo, Sonora, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	IR 45
	Guymas, Sonora, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 60
	Culiacan, Sinaloa, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 80
	Mazatlan, Sinaloa, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 65
	Puerto Valarta, Jalisco, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 30
	Manzanillo, Colima, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 55
	Lazaro Cardenas, Michoacan, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 45, with a large standard deviation
	Ixtapa Zihuatanejo, Guerrero, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 42, with a large standard deviation
	Coyuca de Benitez, Guerrero, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 70
	Puerto Excondido, Oaxaca, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 60
	Tapachula, Chiapas, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 70 (two collections from Tapachula were tested giving one TR of 60, one of 80)
	Chetumal, Quintana Roo, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 80

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	Cancun, Quintana Roo, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 70
	Merida, Yucatan, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 69
	Campeche, Campeche, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 42
	Ciudad del Carmen, Campeche, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 42
	Villahermosa, Tabasco, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 58
	Moloacan, Veracruz, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 58
	Miguel Aleman, Tamaulipas, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 60
	Nuevo Ladero, Tamaulipas, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 48
	Monterey, Nuevo Leon, MX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 56
	Huston, TX	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 40, with a great standard deviation
	Tucson, Arizona	DENV-2 JAM1409	BM[4], $10^{7.5}$ to $10^{8.5}$	TR 68
Vazeille et al., 2001	Mahaleja, Madagascar	DENV-2 Bangkok 1974	BM[3], $10^{8.2}$	IR 27.8
	Jeffreville, Madagascar	DENV-2 Bangkok 1974	BM[3], $10^{8.2}$	IR 32.5
	Paea Lab. strain	DENV-2 Bangkok 1974	BM[3], $10^{8.2}$	IR 94
Tran et al., 1999	Ho Chi Minh City	DENV-2 Bangkok 1974	BM[3], $10^{8.2}$	IR 96,16 +/− 3.35
Watson & Kay, 1999[12]	Queensland, Aus Lab. strain	DENV-1 from hs of a patent in Townaville in 1990	BM[6], 0–6–3,6 Log10	IR 31 +/− 23.34
		DENV-2 from hs of a patent in Townaville in 1992	BM[6], 1,2–4,2 Log10	IR 35.5 +/− 25.67
		DENV-3 h87	BM[6], 0,9–3,9 Log10	IR 42 +/− 27.72
		DENV-4 h241	BM[6], 0,6–3,6 Log10	IR 36 +/− 22,02
Jupp and Kemp, 1993[12]	Empangeni, SA	DENV-1 Cassim strain from Durban, SA	BM[3], 7,2 Log10	IR 100 at 8–10 dpi
	Palm Beach, SA	DENV-1 Cassim strain from Durban, SA	BM, 6.1–7.1 Log10	IR 15, TR 100 at 17–19 dpi; IR 28, TR 50 at 16–17 dpi

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
		DENV-2 BC 5007 strain from Taipei	BM[3], 7.2–7.9 Log10	IR 15.5 and TR 50 at 17–18 dpi; IR 25, TR 83 at 15 dpi
	Durban, SA	DENV-1 Cassim strain from Durban, SA	BM[3], 6.3–7.1 Log10	IR 62.8, TR 92 at 17–19 dpi; IR 43, TR 73 at 13–15 dpi
		DENV-2 BC 5007 strain from Taipei	BM, 7–7.5 Log10	IR 46, TR 75 at 14–15 dpi
	Richards Bay, SA	DENV-1 Cassim strain from Durban, SA	BM[3], 6.1–7.1 Log10	IR 38, TR 69.5 at 17–19 dpi,
		DENV-2 BC 5007 strain from Taipei	BM[3], 7.2–7.5 Log10	IR 29.5; TR 69 at 14–20 dpi
	Ndumu, SA	DENV-1 Cassim strain from Durban, SA	BM[3], 6.3–7.1 Log10	IR 36.5; TR 75 at 18–19 dpi
		DENV-2 BC 5007 strain from Taipei	BM, 7.1 Log10	IR 41.67; TR 82 at 14–18 dpi
	Skukuza, SA	DENV-1 Cassim strain from Durban, SA	BM[3], 6.9–8.4 Log10	IR 12.5; TR 100 at 14–20 dpi;
		DENV-2 BC 5007 strain from Taipei	BM[3], 7–7.9 Log10	IR 28; TR 66.5 at 16–19 dpi
Chen et al., 1993	Kaohsiung, southern Taiwan	DENV-1 from a dengue patient during the dengue epideminc in Kaohsiung in 1987–1988	IT	TR 50 at 14 dpi, 83.3 at 21 dpi
Bosio et al., 1998	San Juan, PR	DENV-2PR-159, PR	BM, ntd	IR in midguts: 61
	Aedes aegytpi formosus from Ibo village, Nigeria	DENV-2PR-159, PR	BM, ntd	IR in midguts: 25
Mitchell et al., 1987	Rexville strain from PR	DENV-1 1620, PR	BM[3], 6.6–9.2 Log10	IR 45 at 7 dpi, 605 at 14 dpi, TR 88
		DENV-2 1615, PR	BM[3], 5.6–8.4 Log10	IR 25 at 7 dpi, 28.67 at 13 dpi, 56.4 1t 14 dpi, TR 74
		DENV-3 1557, PR	BM[3], 6.3–8.4 Log10	IR 5 at 7 dpi, 58.2 at 14 dpi, TR 53
		DENV-4 1632, PR	BM[3], 6.2–9.2 Log10	IR 0 at 7 dpi, 19.67 at 13 dpi, 63 at 14 dpi, TR 42
Boromisa et al., 1987	Lab. strain from Huston, TX	DENV-1 YARU 40130, Fijii	BM[3], 8.3 Log10	IR 70 in midguts; 30 in whole body; TR 5
Rosen et al., 1985	Rockefeller strain	DENV-1 Hawaii 1944	BM[3], $10^{7.8}$	IR 16.7
	Niue strain from Niue Island	DENV-1 Hawaii 1944	BM[3], $10^{7.8}$	IR 0
		DENV-1 Malay-1 (Malaysia 1965)	BM[3], $10^{7.8}$	IR 0
		DENV-1 Malay-2 (Malaysia 1966)	BM[3], $10^{7.8}$	IR 20
		DENV-1 Thai (Bangkok, 1971)	BM[3], $10^{7.8}$	IR 25

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	Rockefeller strain	DENV-2 NG 1944	BM[3], $10^{7.8}$	IR 50
		DENV-2 Thaiti 1971	BM[3], $10^{7.8}$	IR 45
	Niue strain from Niue Island	DENV-2 Thaiti 1971	BM[3], $10^{7.8}$	IR 13.6
	Tong strain from Tonga	DENV-2 Thaiti 1971	BM[3], $10^{7.8}$	IR 23.5
	Rockefeller strain	DENV-3 H87 Manila, Phi 1956	BM[3], $10^{7.8}$	IR 26.7
		DENV-3 Manila Manila Phi 1965	BM[3], $10^{7.8}$	IR 34.6
		DENV-3 Tahiti 1964	BM[3], $10^{7.8}$	IR 30.8
		DENV-3 Thai, Bangkok Thai 1971	BM[3], $10^{7.8}$	IR 36.8
	Trinidad strain from Trinidad	DENV-3 Manila Manila Phi 1965	BM[3], $10^{7.8}$	IR 20
		DENV-3 Tahiti 1964	BM[3], $10^{7.8}$	IR 22.2
		DENV-3 Thai, Bangkok Thai 1971	BM[3], $10^{7.8}$	IR 71
	Rockefeller strain	DENV-4 H241	BM[3], $10^{7.8}$	IR 100–0 depending on viral dose inocula
2) ZIKV				
Calvez et al. (2018)	French Polynesia	NC-2014-5132, NC	BM, 107 TCID50/mL	IR: 53 at 6 dpi; 94 at 9 dpi; 97 at 14 dpi, 89 at 21 dpi; TR 0 between 6 and 9 dpi; 24 at 21 dpi
	NC			IR: 88 at 6 dpi; 73 at 9 dpi; 77 at 14 dpi, 95 at 21 dpi; TR 0 at 6dpi, 3 at 9 dpi, 0 between 14 and 21 dpi
	Samoa			IR: 33 at 6 dpi; 23 at 9 dpi; 50 at 14 dpi, 38 at 21 dpi; TR 0 between 6 and 9 dpi; 17 at 14 dpi and 30 at 21 dpi
Main et al. (2018)	Los Angeles, CA	PRVABC59, PR	BM, 5.4-6.4 log10	IR: 85 at 14 dpi; 96 at 21 dpi; DR 78 at 7-14 dpi, TR 65 at 14 dpi, 74 at 21 dpi
		MA66, P6-740, Maylasia	BM, 4.3-4.8 log10	IR: 86 at 14 dpi; 96 at 21 dpi; DR 79 at 7 dpi, 91 at 14 dpi, TR 53 at 14 dpi, 87 at 21 dpi
		BR15, SPH2015, BR	BM, 4.7 log10	IR: 90; DR: 90; TR: 75 at 14 dpi
Garcia-Luna et al. (2018)[12]	Apodaca, MX	PRVABC59, PR	BM, $1.5\text{-}1.8\times10^{6}$	IR 79 at 7 dpi; 84 at 14 dpi; DR 71 at 7 dpi, 80 at 14 dpi; TR 15 at 7 dpi; 33 at 14 dpi
	San Nicolas, MX	PRVABC59, PR	BM, $4\times10^{5}\text{-}2\times10^{7}$	IR 97 at 7 dpi; 93 at 14 dpi; DR 51 at 7 dpi, 88 at 14 dpi; TR 4 at 7 dpi; 27 at 14 dpi
	Monterey, MX	PRVABC59, PR	BM, $8\times10^{5}\text{-}4\times10^{7}$	IR 83 at 7 dpi; 63 at 14 dpi; DR 19 at 7 dpi, 45 at 14 dpi; TR 1 at 7 dpi; 14 at 14 dpi

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	Cd. Madero, MX	PRVABC59, PR	BM, 6.2-8×10^5	IR 53 at 7 dpi; 60 at 14 dpi; DR 28 at 7 dpi, 52 at 14 dpi; TR 7 at 7 dpi; 17 at 14 dpi
	Poza Rica, MX	PRVABC59, PR	BM, 1.4x10^5x1.8×10^7	IR 100 at 7-14 dpi; DR 98 at 7 dpi, 100 at 14 dpi; TR 10 at 7 dpi; 52 at 14 dpi
	Minatitlan, MX	PRVABC59, PR	BM, 6.2×10^5-1.6×10^6	IR 91 at 7dpi, 81 at 14 dpi; DR 72 at 7 dpi, 78 at 14 dpi; TR 10 at 7 dpi; 29 at 14 dpi
	Coatzacoalcos, MX	PRVABC59, PR	BM, 1.4×10^5-1.7×10^6	IR 92 at 7dpi, 98 at 14 dpi; DR 73 at 7 dpi, 95 at 14 dpi; TR 24 at 7 dpi; 51 at 14 dpi
	Merida, MX	PRVABC59, PR	BM, 8×10^5-4.4×10^7	IR 99 at 7dpi, 96 at 14 dpi; DR 74 at 7 dpi, 92 at 14 dpi; TR 10 at 7 dpi; 42 at 14 dpi
	Mazatan, MX	PRVABC59, PR	BM, 1.12-4.4×10^7	IR 100 at 7-14dpi; DR 95 at 7 dpi, 100 at 14 dpi; TR 15 at 7 dpi; 23 at 14 dpi
	Guerrero, MX	PRVABC59, PR	BM, 2×106-1.8×10^7	IR 98 at 7, 93 at 14dpi; DR 95 at 7 dpi, 93 at 14 dpi; TR 50 at 7 dpi; 42 at 14 dpi
Dodson et al. (2018)	Rockefeller strain	PRVABC59, PR	BM, 2×10^8	IR: 40.67 +/− 19; TR 2.67 +/− 4.62
Roundy et al. (2017)	Salvador, BR	DAK AR 41525, S	BM/murine[2], 10^{4-6}	IR 100; TR100
		FSS 13025, Cambodia	BM/murine[2], 10^{4-6}	IR 75; TR 0 murine: IR 100; TR 40
		MEX1-7, MX	BM, 2×10^8	IR 75; TR 0
	Dominican Republic	DAK AR 41525, S	BM, 2×10^8	IR 100; TR100
		FSS 13025, Cambodia	BM, 2×10^8	IR 100; TR 18
		MEX1-7, MX	BM, 2×10^8	IR 90; TR 20
	RioGrande Valley	DAK AR 41525, S	BM, 2×10^8	IR 100; TR 30
		FSS 13025, Cambodia	BM, 2×10^8	IR 40; TR 0
		MEX1-7, MX	BM, 2×10^8	IR 65; TR 0
Kenney et al. (2017)	Poza Rica, MX, Lab. strain	PRV ABC59	IT, 10^6	IR 100; TR 67
Heitmann et al., 2017	Bayer company, Lab. strain	FB-GWUH-2016, Central America	BM, 10^7	18 °C: IR 55; TR 0 27 °C: IR 49; TR 22
Fernandes et al. (2017)	Rio de Janeiro, BR	ZIKV strains from BR	BM, $10^{6.36}$	IR 68-100;
Guedes et al. (2017)	Fernando de Noronha, BR	BRPE 243/ 2015, BR	BM, 10^6	IR 40
	Recife, Lab. strain	BRPE 243/ 2015, BR	BM, 10^6	IR 44

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
Ciota et al. (2017)	Poza Rica, MX	CAM FSS130325, Cambodia	BM, $10^{6.6\text{-}7.7}$	IR 44; TR 33
		HND 2016-19,563, Honduras	BM, $10^{6.6\text{-}7.7}$	IR 47; TR 36
Li et al. (2017)[9]	HK strain from mosquitoes collected in Hainan province, Chi	SZ01/2016/Chi	BM, 3×10^5	IR midguts: 80 at 2dpi, 80 at 4 dpi, 85 at 6 dpi, 90 at 8 dpi, 100 at 10 dpi, 90 at 12 dpi, 100 at 16,18 and 20 dpi
				IR salivary glands: 58 at 2dpi, 78 at 4 dpi, 85 at 6 dpi, 90 at 8 dpi, 90 at 10 dpi, 100 at 12 dpi, 90 at 16,100 at 18 and 20 dpi
	RL strain from mosquitoes collected in Yunnan province, Chi	SZ01/2016/Chi	BM, 3×10^5	IR midguts 100 at 2, 4, 6, 8, 10, 12, 16,18 and 20 dpi
				IR salivary glands: 60 at 2dpi, 80 at 4 dpi, 100 at 6 dpi, 90 at 8 dpi, 100 at 10, 12, 16, 18 and 20 dpi
Ryckebusch et al. (2017)	Paea strain, Thaiti	PF-25013-18	BM[2], 2.5×107	IR midguts 100 from 3 to 10 dpi, 85 at 13 dpi
				IR in salivary glands 60 at 5, 6 and 8 dpi, 80 at 10 dpi and 7 at 14 dpi
				TR 11 at 8 dpi, 33 at 10 dpi, 16 at 14 dpi and 6.7 at 17 dpi
Costa-da-Silva et al. (2017)	Rockefeller lab. Strain	ZIKVBR Isolated from a clinical case	BM; 2.2×10^6	IR 95 in body and heads at 7 and 14 dpi; TR 10 at 7 dpi; 38 at 14 dpi
	HWE Lab. strain		BM; 2.2×10^6	IR 60 in body, 50 in heads at 7 dpi; 65 in body and head at 14 dpi; TR 0 at 7dpi, 35 at 14 dpi
	RED lab. Strain		BM; 2.2×10^6	IR 95 in body and 70 heads at 7 dpi; 95 in body and heads at 14 dpi; TR 0 at 7 dpi, 5 at 14 dpi
Weger-Lucarelli et al. (2016)	Poza Rica, MX	PRV ABC59, PR	BM, fresh $10^{6.3}$	IR 95, TR 70
		PRV ABC59, PR	BM, frozen 4 h $10^{6.3}$	IR 95, TR 65
		PRV ABC59, PR	BM, frozen 1 week $10^{6.3}$	IR 60, TR 22
		DAKAR 41525, S	BM, frozen $0^{7.2}$	IR 75, TR 55
		MR 766, Uganda	BM, frozen $10^{7.2}$	IR 58, TR 37
Richard et al. (2016a)	Tahiti 2014	PF13/2511013-18 Polynesia	BM[4], 10^7	BM: IR 85; TR 36
Hall-Mendelin et al. (2016)	Queensland, Aus	MR 766, Uganda	BM[4], $10^{6.7}$	BM: IR 57; TR 27
Di Luca et al. (2016)	MX, Lab. strain	H/PF/2013 French Polynesia	BM, $10^{6.4}$	IR 40, TR 40
Dutra et al. (2016)	Urca, Rio de Janeiro, BR	BRPE 243/2015 BR	BM, fresh 5×10^6	IR 100, TR 100
Alto et al. (2017)	Black eyed Liverpool, Lab. strain	PRV ABC59	Murine $10^{6.8}$	IR 100; TR 24

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
Boccolini et al. (2016)	Reynosa, MX, Lab. strain	H/PF/2013 French Polynesia	BM, $10^{6.46}$	IR 50; TR 38
Chouin-Carneiro et al. (2016)	FG	NC-2014-5132, NC	BM[4], 10^7	7 dpi: IR 100, TR 0
	Guadeloupe	NC-2014-5132, NC	BM[4], 10^7	7 dpi: IR 87; TR 0
	Martinique	NC-2014-5132, NC	BM[4], 10^7	7 dpi I: IR 90; TR 0
	Orlando, FL	NC-2014-5132, NC	BM[4], 10^7	7 dpi: IR 93; TR nd
	Tubiacanga, BR	NC-2014-5132, NC	BM[4], 10^7	7 dpi: IR 83; TR nd
Li et al. (2012)	Singapore	MR 766, Uganda	BM[4], 10^7	BM: IR 100; TR 100
Diagne et al. (2015)[13]	Dakar, S, domestic	ArD 128,000 and 132,912, Kedougou	BM 6.4-7.6 $\log_{10}$	IR+, DR+, TR 0
	Kedougou, S, sylvatic	ArD 128,000 and 132,912, Kedougou	BM 6.4-7.6 $\log_{10}$	IR+, DR+, TR 0
Comet and Robin (1979)	S-1971, Lab. strain	ArD 24,280, S	IT dose unknown 7-28 dpi	TR 91
Boorman and Porterfield (1956)	Nigeria, Lab. strain	MR 766, Uganda	BM, $10^{6.7}$ LD50 60 dpi	IR 100; TR 50
3)YFV				
Couto-Lima et al. (2017)[12]	Goiania, BR	74,018-1D from BR	BM, 10^6	IR 0 at 3dpi, ~ 30 at 7dpi, ~ 80 at 14 dpi, ~ 70 at 14 dpi
		4408-1E from BR	BM, 10^6	IR 0 at 3dpi, ~ 25 at 7dpi, ~ 78 at 14 dpi, ~ 10 at 14 dpi
		S-79 from Senegal	BM, 10^6	IR 0 at 3dpi, ~ 30 at 7dpi, ~ 80 at 14 dpi, 0 at 14 dpi
		74,018-1D from BR	BM, 10^6	TR 0 at 3dpi, 0 at 7dpi, ~ 18 at 14 dpi, 0 at 14 dpi
		4408-1E from BR	BM, 10^6	TR 0 at 3dpi, 0 at 7dpi, ~ 18 at 14 dpi, 58 at 14 dpi
		S-79 from S	BM, 10^6	TR 0 at 3dpi, 0 at 7dpi, 0 at 14 dpi, 0 at 14 dpi
Dickson et al. (2014)	Fatick	BA-55- West African Genyotype I, Nigeria	BM, 10^6	IR 59
	Fatick	DAK -1279- West African Genyotype II, S	BM, 7.9×10^5	IR 17
	Bignona	BA-55- West African Genyotype I, Nigeria	BM, 10^6	IR 13
	Bignona	DAK -1279- West African Genyotype II, S	BM, 6.1×10^7	IR 33
	Richard Toll	BA-55- West African Genyotype I, Nigeria	BM, 2×10^6 BM, 7.9×10^5	IR 10

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Richard Toll	DAK -1279- West African Genyotype II, S		IR 57
	Goudiry	BA-55- West African Genyotype I, Nigeria	BM, 10^6	IR 0
	Goudiry	DAK -1279- West African Genyotype II, S	BM, 7.9×10^5	IR 10
	Ae aegypti formosus PK10, S, sylvatic	BA-55- West African Genyotype I, Nigeria	BM, 2×10^5	IR 0
	Ae aegypti formosus PK10, S, sylvatic	DAK -1279- West African Genyotype II, S	BM, 7.9×10^5	IR 10
	Ae aegypti formosus PK10, S, sylvatic	BA-55- West African Genyotype I, Nigeria	BM, 10^6	IR 3
	Ae aegypti formosus PK10, S, sylvatic	DAK -1279- West African Genyotype II, S	BM, 7.9×10^5	IR 22
	Mont Rolland	BA-55- West African Genyotype I, Nigeria	BM, 2×10^6	IR 0
	Mont Rolland	DAK -1279- West African Genyotype II, S	BM, 7.9×10^5	IR 20
	Rufisque	BA-55- West African Genyotype I, Nigeria	BM, 10^6	IR 0
	Rufisque	DAK -1279- West African Genyotype II, Senegal	BM, 7.9×10^5	IR 11
Ellis et al. (2012)	Nairobi, Kenya	East African genotype (Sudan 2003)	BM, 6.7-7.5 log10	IR 7
	Mariakani, Kenya	East African genotype (Sudan 2003)	BM, 6.7-7.5 log10	IR 41
	Kerio Valley, Kenya	East African genotype (Sudan 2003)	BM, 6.7-7.5 log10	IR 11
	Kakamega, Kenya	East African genotype (Sudan 2003)	BM, 6.7-7.5 log10	IR 23
van den Hurk et al. (2011)	Cairns, Aus	African strain BA-55 (Nigeria 1955)	BM[4], $10^{7.2}$	IR 80, TR 52
		South American strain, Cinetrop 28 (OBS 7549) Bolivia 1999	BM[4], $10^{6.7}$	IR 64, TR 64
		Asibi strain	BM[4], 10^8	IR 92, TR 80
	Townsville, Aus	African strain BA-55 (Nigeria 1955)	BM[4], $10^{7.2}$	IR 72, TR 60
		South American strain, Cinetrop 28 (OBS 7549) Bolivia 1999	BM[4], $10^{6.7}$	IR 36, TR 28
		Asibi strain	BM[4], 10^8	IR 96, TR 96

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	RexD strain	African strain BA-55 (Nigeria 1955)	BM[4], $10^{7.2}$	IR 82, TR 64
		South American strain, Cinetrop 28 (OBS 7549) Bolivia 1999	BM[4], $10^{6.7}$	IR 40, TR 32
		Asibi strain	BM[4], 10^{8}	IR 76, TR 64
Johnson et al. (2002)	Santos, Brazil	no. 71528 MG2001, from BR	BM, 7-7.8 log10	IR 35, TR 25.5
Lourenco-de-Oliveira et al. (2002)	Milhã, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 0
	Comendador Soares, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 0.9
	Quixeramobim, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 1.7
	Rocinha, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 3.3
	Tinguá, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 4.9
	Pacujá, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 5.6
	Salvador, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 6.3
	Higienópolis, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 6.7
	Moquetá, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 7.6
	Feira de Santana, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 10.6
	Rio Branco, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 11.1
	Leandro Ferreira, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 12.0
	Cariacica, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 12.6
	Boa Vista, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 12.9
	Represa do Cigano, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 16.1
	São Luis, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 19.6
	Maringá, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 22.7
	Porto Velho, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 24.4
	Campo Grande, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 25
	Potim, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 27.1
	Belém, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 33.9
	Ananindeua, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 46.4

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Foz do Iguaçu, BR	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 48.6
	Phnom Penh, Cambodia	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 64.4
	Ho Chi Min	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 48.05
	Maracay, Venezuela	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 13.6
	West Palm Beach, FL	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 24.8
	Ae. aegypti formosus Boulbinet Guinea	FIOCRUZ 74018/MG/01	BM[3], $10^{8.7}$	IR 3.3
Mitchell et al. (1987)	Rexville strain from PR	788,379	BM, 5.0-6.7 Log10	IR 61 at 11 dpi, 80 at 14 dpi; TR 42 at 11 dpi, 38 at 14 dpi
Wallis et al. (1985)	Soufriere, Dominica	Asibi strain	BM, ntd	IR 17,17 +/− 13,50
Tabachnick et al. (1985)	West Africa Sylvan, Dakar S, lab. Strain	Asibi strain	BM, ntd	IR 11
	West Africa Sylvan, N'Gove S, lab. Strain	Asibi strain	BM, ntd	IR 7
	West Africa Sylvan, Gambia, lab. Strain	Asibi strain	BM, ntd	IR 27
	East Africa Sylvan, Kampala Uganda, lab. Strain	Asibi strain	BM, ntd	IR 8
	East Africa Sylvan, Kombeni, Kenya; lab. Strain	Asibi strain	BM, ntd	IR 34
	East Africa Domestic, Kwa Dzivo Kenya; isofemale lines	Asibi strain	BM, ntd	IR 57
	East Africa Domestic, Majengo Kenya; isofemale lines	Asibi strain	BM, ntd	IR 29
	Asia-Pacific Domestic Bangalore India; lab. Strain	Asibi strain	BM, ntd	IR 23
	Asia-Pacific Domestic Colombo Sri Lanka; lab. Strain	Asibi strain	BM, ntd	IR 21

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	Asia-Pacific Domestic Djakarta Java; lab. Strain	Asibi strain	BM, ntd	IR 32
	Asia-Pacific Domestic Karachi Pakistan; lab. Strain	Asibi strain	BM, ntd	IR 30
	Asia-Pacific Domestic Thai, Amphur strain	Asibi strain	BM, ntd	IR 28
	Asia-Pacific Domestic Fiji; lab. Strain	Asibi strain	BM, ntd	IR 22
	Domestic Austin, TX; isofemale lines	Asibi strain	BM, ntd	IR 29
	Domestic Galveston, TX; lab. Strain	Asibi strain	BM, ntd	IR 16
	Domestic Huston, TX; lab. Strain	Asibi strain	BM, ntd	IR 21
	Domestic Welasco, Texas USA; lab. Strain	Asibi strain	BM, ntd	IR 15
	Domestic Victoria, MX; isofemale lines	Asibi strain	BM, ntd	IR 20
	Domestic Abbeville, Luisiana USA; lab. Strain	Asibi strain	BM, ntd	IR 12
	Domestic Beamont, TX; lab. Strain	Asibi strain	BM, ntd	IR 26
	Domestic Vero Beach, FL; field	Asibi strain	BM, ntd	IR 41
	Domestic Esquintla, Guatemala; isofemale lines	Asibi strain	BM, ntd	IR 2
	Domestic Malaga, Colombia; field	Asibi strain	BM, ntd	IR 46
	Domestic Santa Cruz, Bolivia; isofemale lines	Asibi strain	BM, ntd	IR 31
	Domestic Trinidad, West Indies; isofemale lines	Asibi strain	BM, ntd	IR 42

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Domestic Arecibo, Puerto Rico; lab. Strain	Asibi strain	BM, ntd	IR 34
	Domestic Limestone Bay, Anguilla; field	Asibi strain	BM, ntd	IR 39
	Domestic Plymouth, Montserrat; field	Asibi strain	BM, ntd	IR 53
4) CHIKV[14]				
Agha et al. (2017)	Mombasa, Kenya	Lamu001 strain of and East/Central/ South Africa lineage	BM, $10^{5.6}$	IR 0 at 5-7 dpi
			BM, $10^{5.9}$	IR 6 at 5-7 dpi and 17 at 9 dpi
			BM, $10^{6.9}$	IR 62 at 5-7 dpi
			BM, $10^{7.5}$	IR 100 at 5-7 dpi and 75 at 14 dpi
	Kisumu, Kenya		BM, $10^{5.6}$	IR 0 at 5-7 dpi and 0 at 14 dpi
			BM, $10^{5.9}$	IR 20 at 5-7 dpi; 5 at 9 dpi and 6 at 14 dpi
			BM, $10^{6.9}$	IR 40 at 5-7 dpi; 50 at 9 dpi and 63 at 14 dpi
	Nairobi, Kenya		BM, $10^{5.6}$	IR 0 at 5-7 dpi and 17 at 14 dpi
			BM, $10^{5.9}$	IR 7 at 5-7 dpi and 10 at 9 dpi
			BM, $10^{6.9}$	IR 50 at 5-7 dpi and 57 at 9 dpi
			BM, $10^{7.5}$	IR 71 at 5-7 dpi and 89 at 14 dpi
Alto et al. (2017)	Indian River/ St. Lucie County, FL		BM, 8 log10	IR in legs 37 at 2dpi, 71 at 5 dpi, 28 at 12 dpi; TR 35 at 2 dpi, 66 at 5 dpi, 24 at 12 dpi
	Monroe County, FL		BM, 8 log10	IR in legs 90 at 2dpi, 20 at 5 dpi, 54 at 12 dpi; TR 83 at 2 dpi, 18 at 5 dpi, 50 at 12 dpi
	Manatee county, FL		BM, 8 log10	IR in legs 71 at 2dpi, 68 at 5 dpi, 60 at 12 dpi; TR 58 at 2 dpi, 63 at 5 dpi, 51at 12 dpi
	Dominican Repuublic		BM, 8 log10	IR in legs 35 at 2dpi, 22 at 5 dpi, 18 at 12 dpi; TR 17 at 2 dpi, 19 at 5 dpi, 15 at 12 dpi
Ngoagouni et al. (2017)	Bangui, Central African Republic	ArB10262	BM; 108	IR 50 at 7 dpi, 27 at 14 dpi, TR 0 at 7 dpi, 28 at 14 dpi
Mbaika et al. (2016)	Coastal Kenya	South/Central Africa and Indian Ocean Genotype (Group III), subgroup IIIa and b	BM; 7.9×10^{5}	IR tested in Midgut at 26 °C 26.41 7dpi; 33.96 10 dpi, 39.62 13 dpi;
				IR tested in Midgut at 32 °C 26.41 7dpi; 33.96 10 dpi, 39.62 13 dpi;

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
				IR tested in legs at 26 °C 17.9 7dpi; 25.5 10 dpi, 17 13 dpi;
				IR tested in legs at 32 °C 6.8 7dpi; 20.4 10 dpi, 29.1 13 dpi;
				IR tested in heads at 26 °C 10.4 7dpi; 2.8 10 dpi, 2.8 13 dpi;
				IR tested in heads at 32 °C 2.9 7dpi; 16.5 10 dpi, 26.2 13 dpi;
	Western Kenya	South/Central Africa and Indian Ocean Genotype (Group III), subgroup IIIa and b	BM; 7.9×10^5	IR tested in Midgut 26 °C 7.55 7dpi; 5,66 10 dpi, 18,88 13 dpi;
				IR tested in Midgut 32 °C 33,02 7dpi; 24,53 10 dpi, 24,53 13 dpi;
				IR tested in legs at 26 °C 26.5 7dpi; 11.8 10 dpi, 20.6 13 dpi;
				IR tested in legs at 32 °C 28.7 7dpi; 17.2 10 dpi, 26.4 13 dpi;
				IR tested in heads at 26 °C 26.5 7dpi; 17.6 10 dpi, 20.6 13 dpi;
				IR tested in heads at 32 °C 25.3 7dpi; 8 10 dpi, 23 13 dpi;
Richard et al. (2016b)	districts of Toahotu, Thaiti Island	PF14/300914-109	BM[4], 7 log10 TCID50/mL	IR 78 at 6 dpi, 87 at 9 dpi, 90 at 14 dpi, 80 at 21 dpi
				TR 5 at 2 dpi, 18 at 6 dpi, 34 at 9 dpi, 49 at 14 dpi abd 53 at 21 dpi
Vega-Rua et al. (2014)	Vero Beach, FL	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 dpi, 100 at 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100
	Chiapas, MX	CHIKV 06.21	BM $10^{7.5}$	IR 96.7 at 7 dpi, 93.3 at 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 96.7 at 7 dpi, 100 at 10 dpi
	Panama	CHIKV 06.21	BM $10^{7.5}$	IR 96.7 at 7 dpi, 100 at 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 96.7 at 7 and 10 dpi
		NC/2011-568	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Delta Amacuro, Venezuela	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Tumbes, Peru	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Punchana, Peru	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Manaus, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		NC/2011-568	BM $10^{7.5}$	IR 100 at 7 and 10 dpi

			Vector Competence	
Reference	**Mosquito origin**	**Virus genotype and strain**	**Infection Route, virus dose**[1]	**Results**[7]
	Santarem, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Parnamirin, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Campos Belos,BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Campos Grande, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Jurujuba, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Paqueta, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Vaz Lobo, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 dpi; 96,7 at 10 dpi
	Belford Roxo, BR	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Santos, BR	CHIKV 06.21	BM $10^{7.5}$	IR 93.3 at 7 dpi, 100 at 10 dpi
	Monteagudo, Bolivia	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Salto del Guaira, Paraguay	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Asuncion, Paraguay	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 96.7 at 7 dpi, 93.3 at 10 dpi
	Salto, Uruguay	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
	Corrientes, Argentina	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 and 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 100 at 7 dpi, 96.7 at 10 dpi
	Buenos Aires, Argentina	CHIKV 06.21	BM $10^{7.5}$	IR 100 at 7 dpi, 96.7 at 10 dpi
		CHIKV 05.115	BM $10^{7.5}$	IR 96.6 at 7 dpi, 100 at 10 dpi
		NC/2011-568	BM $10^{7.5}$	IR 96.9 at 7 dpi, 90 at 7 dpi
Dupont-Rouzeyrol et al. (2012)	Noumea, NC, mosquitoes had a 92% susceptibility to	NC/2011-568	BM $10^{7.5}$	IR 53.3 at 3 dpi; 54.5 at 8 dpi; 66.7 at 14 dpi

Reference	Mosquito origin	Virus genotype and strain	Vector Competence	
			Infection Route, virus dose[1]	Results[7]
	pyrethroids (pop 163/11)			
	Noumea, New Caledonia, mosquitoes had a 85% susceptibility to pyrethroids (pop 174/11)		BM $10^{7.5}$	IR 50 at 3 dpi; 64.3 at 8 dpi; 20 at 14 dpi
	Noumea Laboratory strain, New Caledonia (pop 282/10)		BM $10^{7.5}$	IR 40 at 3 dpi; 58.8 at 8 dpi; 50 at 14 dpi
	Noumea, NC, mosquitoes had a 92% susceptibility to pyrethroids (pop 163/11)	CHIKV-RE from Reunion Island (2005), also known as CHIKV 06.21	BM $10^{7.5}$	IR 33.3 at 3 dpi; 57.1 at 8 dpi; 75 at 14 dpi
	Noumea, NC, mosquitoes had a 85% susceptibility to pyrethroids (pop 174/11)		BM $10^{7.5}$	IR 73.3 at 3 dpi; 46.2 at 8 dpi; 90 at 14 dpi
	Noumea Lab.strain, NC(pop 282/10)		BM $10^{7.5}$	IR 40 at 3 dpi; 57.1 at 8 dpi; 66.7 at 14 dpi
Girod et al. (2011)[15]	Pointe a Pitre, Carenage, Guadaloupe	CHIKV 06.21	BM, $10^{7.5}$	IR 98 at 14 dpi in 2008; 96.6 at 7 dpi and 100 at 14 dpi in 2009
	Petit bourg, Prise d'eau, Guadalupe	CHIKV 06.21	BM, $10^{7.5}$	IR 95.8 at 14 dpi in 2008; 97.9 at 14 dpi in 2009
	Fort de France, Ermitage, Martinique	CHIKV 06.21	BM, $10^{7.5}$	IR 98.9 at 14 dpi in 2008; 100 at 7 dpi and 96.8 at 14 dpi in 2009
	Robert, Cafe, Martinique	CHIKV 06.21	BM, $10^{7.5}$	IR 97.4 at 14 dpi in 2008; 88.9 at 7 dpi and 93.4 at 14 dpi in 2009
	Cayenne, Centre Ville FG	CHIKV 06.21	BM, $10^{7.5}$	IR 100 at 14 dpi in 2008; 97.5 at 7 dpi and 95.5 at 14 dpi in 2009
	Cayenne, Madeleine, FG	CHIKV 06.21	BM, $10^{7.5}$	IR 98.8 at 14 dpi in 2008; 94.7 at 7 dpi and 98.5 at 14 dpi in 2009
Pesko et al. (2009)	Palm Beach, FL	CHICK LR2006-OPY1, La Reunion Island	BM, 6.1 log10	IR at 6 dpi 18.8 and 57.7 for mosquitoes feeding on pletdgets or water jackets membranes, respectively
			BM, 5.2 log10	IR at 6 dpi 4.5 and 23.8 for mosquitoes feeding on pletdgets or water jackets membranes, respectively

Reference	Mosquito origin	Virus genotype and strain	Vector Competence: Infection Route, virus dose[1]	Vector Competence: Results[7]
			BM, 4.4 log10	IR at 6 dpi 0 and 3.1 for mosquitoes feeding on pletdgets or water jackets membranes, respectively
			BM, 3.6 og10	IR at 6 dpi 0 and 0 for mosquitoes feeding on pletdgets or water jackets membranes, respectively results
5) dual-infections				
Rückert et al. (2017)[16]	Poza Rica, Mexico	CHIKV (strain 99,659)	BM 3.1×10^4-1.9×10^5	IR 87; TR 20 at 3dpi, 30 at 7 dpi, 60 at 14 dpi
		DENV-2 (strain Merida)	BM 3×10^3-7.4×10^5	IR 87; TR 0 at 3 dpi, 15 at 7 dpi, 20 at 14 dpi
		ZIKV (strain PRVABC59)	BM 1.7×10^4-5.4×10^5	IR 48; TR 0 at 3 dpi, 8 at 7 dpi, 40 at 14 dpi
		CHIKV (strain 99,659) + DENV-2 (strain Merida)	BM, as single	IR CHIKV 87; DENV-2 85; TR at 3 dpi CHIKV 10; DENV 0; at 7 dpi CHIKV 38; DENV 10; at 14dpi CHIKV 30, DENV 18
		CHIKV (strain 99,659) + ZIKV (strain PRVABC59)	BM, as single	IR CHIKV 90; ZIKV 45; TR at 3 dpi CHIKV 28; ZIKV 5; at 7 dpi CHIKV 45; ZIKV 8; at 14dpi CHIKV 40, ZIKV 38
		ZIKV (strain PRVABC59) + DENV-2 (strain Merida)	BM, as single	IR ZIKV 50; DENV-2 80; TR at 3 dpi DENV 028; ZIKV 0; at 7 dpi DENV 20; ZIKV 0; at 14dpi DENV 38, ZIKV 20
Göertz et al. (2017)	Rockefeller strain	CHIKV strain 37,997	BM 2×10^5	IR 47.9, TR 10.4
			BM 2×10^6	IR 66.7, TR 5.9
			BM 2×10^7	IR 81.2, TR 21.2
		ZIK Suriname strain 011 V-01621	BM 2×10^5	IR 65.3, TR 34.7
			BM 2×10^6	IR 92.2, TR 68.6
			BM 2×10^7	IR 100, TR 68.3
		CHIKV (strain 37,997) + ZIKV Suriname strain	BM, as single	IR 84.4; TR 11.5
6) infections with arboviruses other than DENVs, YFV, ZIKV and CHIKV				
Wiggins et al. (2018)[12]	Miami, FL	Mayaro virus, Tridinad strain TRVL 4675	BM 7.5 log10	IR 65 at 6 dpi; 80 at 6 dpi; 70 at 9-12 dpi; DR 44 at 3 dpi; 60 at 6 dpi; 80 at 9 dpi-12 dpi; TR < 10 at 3-9 dpi; 25 at 12 dpi
Wang et al. (2012)	Haikou strain, Chi	Western equine encephalomyelitis virus (WEEV), McMillian strain	BM, ntd	IR 25; TR 45
Long et al. (2011)	Iquitos, Peru	Maroyo virus, strain IQT4235	BM, 5.59-7.34 Log10	IR 46.67±21.13; TR 83 +/− 23.44
			BM, 5.57-3.36 Log10	IR 0.46 +/− 1.13;

Reference	Mosquito origin	Virus genotype and strain	Vector Competence: Infection Route, virus dose[1]	Vector Competence: Results[7]
Turell et al. (2007)	Kenya, collected as eggs in 1982	Rift Valley Fever (RVFV) ZH501 from an Egyptian patient	BM, ~$10^{7\text{-}7.8}$	IR 100 at 3-10 dpi; 33 at 11-16 dpi
		Rift Valley Fever ZH501 from an Egyptian patient	BM, ~$10^{>8}$	IR 85 at 3-10 dpi; 75 at 11-16 dpi
Turell et al. (2001)	Rockefeller strain	West Nile virus Crow 397-99	BM $10^{7.2}$	IR 16, TR < 16
Kay et al. (1979)	Townsville colony, from northern Queesland in 1957	Sindbis MRM39		
		Getah N544	BM, 4-6.5 Log ID50	IR 64, TR 28.5, EIP 20
		Ross River T78	BM, 4.9 Log ID50	IR 100, TR 69, EIP 12
		Murray Valley Encephalitis MRM66	BM, 5.1 Log ID50	IR 96, TR 95, EIP 7-10
		Kunji MRM16	BM, > 6.5 Log ID50	IR 46, TR 38, EIP 20-27
		Kokobera MRM32	BM, 4.2 Log ID50	IR 100, TR 100, EIP 12
		Edge Hill C281	BM, 2.7 Log ID50	IR 89, TR 80, EIP 20
		Alfuy MRM3929	BM, > 5.5 Log ID50	IR 47, TR 21, EIP 10-15
		Corriparta MRM1	BM, 2.1-2.9 Log ID50	IR 100, TR 5, EIP 10-15
		Belmont Ch9824	BM, ntd	IR 0, TR 0
		Ngaingan MRM14556	BM, ntd	IR 10, TR 0
		CHIKV BKMS 459/64	BM, 4.7 Log ID50	IR 71, TR 57, EIP 15
Kramer and Scherer (1976)	Laboratory strain	Venezuelan Encephalitis virus, epizootic strain subytoe I, variety B, 69TI597	IT or BM	TR 60 at 14 dpi, 100 at 17 dpi, 50 at 21 and 27 dpi
		Venezuelan Encephalitis virus, enzootic strain subytoe I, variety E, 63Z1	IT or BM	TR 0 at all time points

Abbreviations: BM, mosquitoes offered an infectious blood-meal; IT, mosquitoes were infected by intrathoracic inoculation; dpi, days post infection; IR, percentage of engorged females with viral particles in the head, legs and/or salivary glands; TR, transmission rate calculated as percentage of engorged females with viral particles in the saliva at 14 dpi, unless otherwise stated; PFU, plaque forming units, FFU, fluorescent focus unit, LD_{50}, 50 infectious dose; $TCID_{50}$, 50 tissue culture infectious dose; MID_{50}, mosquito infectious dose for 50 of *Ae. aegypti* individuals; EIP, extrinsic incubation period; MX, Mexico; NC, New Caledonia; Col, Colombia; Viet, Vietnam; NG, New Guinea; FG, French Guiana; Thai, Thailand; S, S; PR, PR; BR, Brazil; Aus, Australia; Chi, China; Philippines, Phi; FL, Florida; South Africa, SA; Texas, TX; California, CA; isol., isolate; human serum, hs; lab. Strain, laboratory strain.

[1] PFU/ml unless otherwise stated

[2] FFU/ml

[3] MID50/ml

[4] TCID50/mL

[5] CCID50/ml

[6] PFU ingested per mosquito

[7] expressed in unless otherwise stated

[8] mosquitoes were tested for infections within the 9th generation after laboratory colonization

[9] Infection and transmission rates reported here were extrapolated from a figure

[10] wild-caught mosquitoes were adapted to the laboratory and tested at generation F10-15

[11] Infection rates for DENV2 AdR 140,875 are mean over two infections experiments

[12] results are mean over different experiments

[13] mosquitoes were infected by all viruses strains and dissemination was studied for both strains

[14] CHIKV 06.21 is the strain with the E1-226 V mutation and CHIKV 05.115 is the strain with the E1-226A mutation

[15] experiments were carried out in two consecutive years (2008 and 2009); in 2009, two different concentrations of CHIKV were compared for infection rates at 7 dpi; only data for the highest concentration are shown here

[16] mosquitoes of the F12_F14 after laboratory colonization were used in experimental infections.