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Identification of Zika virus vectors and implications for control



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Zika virus is an emerging pathogen that has recently been causing serious epidemics around the world. Cases of Zika virus disease were reported in Micronesia in 2007¹ and then in French Polynesia in 2013.² In French Polynesia, Guillain-Barré syndrome was reported for the first time in a few patients following Zika virus infection. In Brazil, Zika virus was introduced in 2014,³ and was subsequently associated with cases of microcephaly. So far, an estimated minimum of 400 000 cases of Zika virus disease have been reported in 24 states in Brazil, although the number of cases could be far higher. Most cases are concentrated in the Pernambuco state, in the northeast region. Currently, many countries in South and Central America, besides Brazil, are reporting a high number of Zika virus disease cases. Before 2007, very few human cases had been reported, and as a result, this virus has been poorly studied. It is important to highlight that other diseases caused by Zika virus infection might be identified in the future. This arbovirus has only just begun to spread and could become endemic in some areas in a very rapid manner.

Zika virus disease is a vector-borne disease, but sexual transmission⁴ and congenital cases have now been reported. The first isolation of Zika virus from mosquito samples was made in 1948 from *Aedes africanus*.⁵ In 1956, Weinbren and Williams⁶ isolated two other strains from the same mosquito species. These investigators collected about 1355 *A africanus* specimens, and Zika virus was isolated from two pools, containing 206 and 127 specimens, respectively. Interestingly, other mosquito species that were collected at that time were

all discarded; thus, no other species were tested for the presence of Zika virus by these investigators. Lately, many other *Aedes* species have been surveyed for the detection of Zika virus, and thus far, Zika virus has been detected by RT-PCR or isolated from many mosquito species, human beings, and non-human primates.

Surprisingly, previous studies that have investigated the vector competence for Zika virus have neglected other mosquito species,^{7–9} such as *Culex* species, which are very abundant in the tropical areas where Zika virus has spread and have also transmitted arboviruses that are closely related to Zika virus, such as West Nile virus. Faye and colleagues¹⁰ reported a long list of mosquito species from which Zika virus strains were isolated, including several species of *Aedes* and *Anopheles coustani*. Diallo and colleagues¹¹ surveyed mosquitoes from different environments from Senegal and detected by RT-PCR the presence of Zika virus in ten species from the genus *Aedes*, and *Mansonia uniformis*, *Anopheles coustani*, and *Culex perfuscus*. These mosquito species probably contribute to the zoonotic cycle of Zika virus transmission. However, the simple detection of a virus in a mosquito sample does not incriminate it as a vector. It is important to prove in laboratory conditions that an organism is able to acquire the pathogen and maintain and transmit it to other hosts. Additionally, even if the ability of a given species to transmit a pathogen is proven in laboratory conditions, that species is not necessarily the primary vector. A good example involves *Aedes aegypti* and *Aedes albopictus* in Brazil. Both species are known

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as good dengue vectors; however, *A aegypti* plays a major part in dengue transmission in the country due to its vectorial capacity, whereas *A albopictus* does not because its level of infestation is low and it prefers sylvatic environments.

In this respect, the urban transmission of Zika virus could involve other mosquito species, especially considering the adaptability of this virus,¹² and this issue deserves urgent attention. Vector control strategies must be directed at all potential vectors. To assume that the main vector is *A aegypti* in areas in which other mosquito species coexist is naive, and could be catastrophic if other species are found to have important roles in Zika virus transmission. Therefore, researchers from different institutions who are working on vector–pathogen interactions must attempt to answer this important question as soon as possible, to direct control actions towards the correct target and to help to minimise the drastic effects of Zika virus disease outbreaks.

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Corrections

Malhotra-Kumar S, Xavier BB, Das AJ, Lammens C, Butaye P, Goossens H. *Colistin resistance gene mcr-1 harboured on a multidrug resistant plasmid*. *Lancet Infect Dis* 2016; **16**: 283–84—In the author list of this Correspondence, Surbi Malhotra-Kumar should be listed as Surbhi Malhotra-Kumar. This correction has been made to the online version as of Feb 22, 2016, and the printed version is correct.

Yates TA, Khan PY, Knight GM, et al. *The transmission of Mycobacterium tuberculosis in high burden settings*. *Lancet Infect Dis* 2016; **16**: 227–38—For the sentence on page 229 in this Review, “Although age assortative mixing might mean paediatric infections do not fully reflect M tuberculosis transmission between adults...”, the citation should be reference 68 and not 67. Additionally, for the sentence in the fifth row of the table, “...whether differences in prevalence are a result of variation in transmission or in progression from infection to disease is not clear”, the word prevalence should be changed to incidence. An addition to the acknowledgments should read as follows: “The research was partially funded by the National Institute for Health Research Health Protection Research Unit in Healthcare Associated Infection and Antimicrobial Resistance at Imperial College London in partnership with Public Health England. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR, the Department of Health, or Public Health England.” These corrections have been made to the online version as of Feb 22, 2016.