Aedes albopictus as an epidemic vector of chikungunya virus: another emerging problem?

A major epidemic of chikungunya fever on the island of Reunion (population 770 000) has resulted in 265 000 clinical cases (34% of the population) and 237 deaths.[1] Surprisingly, *Aedes (Stegomyia) aegypti*, the mosquito usually implicated in such outbreaks, is virtually absent.

Chikungunya is an arbovirus of the family Togaviridae. The natural vectors of the virus are African forest mosquitoes of the subgenera *Diceromyia*, *Stegomyia*, and *Aedimorphus* that feed preferentially on primates.[2] The "domestic" form of *A aegypti* is closely associated with human habitation, readily enters houses, feeds almost exclusively on human beings, and is ubiquitous throughout the tropics. By contrast, *Aedes albopictus*, the species implicated in the Reunion outbreak, is of Asian origin, is often abundant far from human habitation, and feeds readily on many species of mammals and birds. In the laboratory, many *A albopictus* strains have a high vector competence (ie, they are readily infected by chikungunya virus),[3] but in nature, they are assumed to have a low vectorial capacity (ie, efficacy as a vector) because blood meals taken from non-susceptible hosts do not contribute to the transmission cycle.[4]

In the Hawaiian islands, major epidemics of dengue were frequent from 1840 until the 1940s, but ceased after an energetic mosquito control campaign. *A aegypti* was virtually eliminated, but *A albopictus* remained widespread and abundant.[5] Since then, despite a high rate of imported cases, there was no evidence of autochthonous transmission until a small outbreak (122 confirmed cases, 0·01% of the total population) in 2001–02. After nearly 60 years, the human herd immunity was minimal, yet there was no repeat of past epidemics. *A aegypti* was rare and restricted to one island, but *A albopictus* was ubiquitous, and super-abundant in the focus of transmission.[6] By contrast, in French Polynesia, where *A aegypti* remains abundant, there have been ten major outbreaks of dengue since World War II, many with high morbidity and substantial mortality.[7] Indeed, there is persuasive evidence that the 2001 outbreak in Hawaii was initiated by infected people arriving from Tahiti, where a major epidemic (about 33 000 cases, 14% of the total population) was under way.

The history of transmission in Hawaii versus Tahiti seems to confirm that *A albopictus* has a low vectorial capacity for dengue viruses compared with *A aegypti*, yet in 1977 there was a major outbreak of dengue 2 on Reunion, with an estimated 160 000 cases—ie, 30% of the population[8]—and laboratory studies confirmed high vector competence in the local *A albopictus*.[9] As in Hawaii, the species was super-abundant, whereas *A aegypti* had remained rare after an effective control campaign in the 1950s.[10,11] Thus, the 1977 epidemic and the current epidemic of chikungunya confirm that *A albopictus* can have a high vectorial capacity, at least on Reunion. The local *A albopictus* population may be more anthropophilic than in other parts of the world; a recent study in Thailand found some evidence of a preference for human blood.[12] Alternatively, the relative abundance of human beings in the peridomestic environment could oblige the mosquito to feed on human beings. Other species could conceivably be involved in the transmission cycle. Whatever, the reasons, it is clear that the role of *A albopictus* as a vector should be re-assessed, both on Reunion and in other parts of the world.

Mosquito control is the sole available method for reducing transmission of chikungunya; no vaccines are available. As already mentioned, large-scale campaigns (using DDT) have been highly effective against *A aegypti*, but not *A albopictus*. Moreover, in the past three decades, even control of *A aegypti* has rarely been achieved and never sustained.[13] Therefore, if we assume that it is possible to prevent future epidemics, we must explore new and innovative approaches, such as novel methods of using insecticides or the introduction of genetically modified strains.[14]

It is ironic to reflect that *A aegypti*, yellow fever, dengue, *A albopictus*, chikungunya, and West Nile virus all have a common vector: mankind. *A aegypti* and yellow fever were transported to the New World during the slave trade, *A albopictus* achieved worldwide distribution via containerised shipments of used tyres,[15,16] West Nile virus almost certainly arrived in the New World in imported birds, and there is a well-documented global traffic of human viruses in aircraft passengers.[17] Thus, modern transportation has produced a quantum leap in the mobility of vectors and pathogens, and the consequences of this globalisation will continue to surprise us.

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