



Editorials

Chikungunya in Italy

BMJ 2007; 335 doi: <http://dx.doi.org/10.1136/bmj.39342.563310.80> (Published 20 September 2007)

Cite this as: BMJ 2007;335:576

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Globalisation is to blame, not climate change

An epidemic of chikungunya virus has recently occurred in Italy, involving more than 190 cases.¹ The concern is that climate change will bring mosquito borne tropical diseases to Northern Europe, but is this outbreak really the result of global warming? Although such an epidemic is new in Europe, it is probably caused by globalisation rather than climate change. Increased amounts of long distance tourism, travel, and trade mean that organisms that live in and on people or goods have more opportunity to be transported across continents.

Chikungunya is an epidemic disease with many similarities to dengue—it causes fever that lasts four to seven days, sometimes with a rash. It is often accompanied by intense arthralgia.² Most infections cause noticeable disease, but haemorrhagic symptoms and other life threatening manifestations are rare.

The virus can be carried by several species of mosquito, but the vector in Italy and in recent epidemics elsewhere is *Aedes albopictus*. Its common names are Asian tiger in English and zanzara tigre in Italian. Its biology is similar to that of its cousin, *Ae aegypti*. Both evolved to breed in natural containers such as tree holes and plant axils, but now they have adapted to life with humans. They are abundant in many modern tropical and subtropical cities, and they exploit many kinds of containers made by humans. Unlike other mosquitoes their eggs can withstand desiccation, which allows them to travel around the world in a variety of containers. The international ship borne trade in used tyres has played a major role in such spread because tyres make good breeding sites and hold water no matter which way up they are stored.

Decades ago, *Ae aegypti* travelled in this way from its ancestral home of coastal East Africa to every corner of the tropical world, becoming the main dengue vector in most of the world's tropical cities. Much later, its Asian cousin *Aealbopictus* began the same process of migration, radiating throughout the Western Pacific and the Indian Ocean Islands, reaching Brazil in 1986 and Nigeria in 1991. It first arrived in the southern United States in 1983 and is now present in 26 states. It arrived in Italy in 1990, and gradually spread to scattered foci all over the country.³ The arrival of the zanzara tigre tends to be noticed because it bites during the day.

Meanwhile, an unprecedented series of chikungunya epidemics has been spreading throughout the Indian Ocean.² These epidemics are often intense and in the past few years have involved millions of cases in Comoros, Madagascar, India, and the East African coast. Travellers have also been affected, and hundreds of imported cases have been reported from all over Europe (including Italy) and the United States. In 2006, there were 133 imported cases in the United Kingdom and 774 in mainland France. In the Italian outbreak, the index case reportedly travelled from India, and a recent analysis in Italy⁴ pointed to tourism as the main reason for travel in imported cases—of 17 infected patients, 11 were tourists, two were on business, two were visiting friends or relatives, and one was a missionary.

The Italian climate has always been suitable for *Ae albopictus* to flourish. The winters in its home range of Japan and Korea are colder than Italian winters; in these conditions the adults die out and the species survives the winter in the egg form. In Italy the adult forms may be able to live through the winter.⁵ If so, this could have important epidemiological consequences, because it might allow the virus to survive the winter inside mosquitoes and to reappear in spring.

What could have been done to prevent the recent outbreak, and what can be done to prevent further outbreaks in future? It is hard to see how Italian scientists could have done more to alert local health authorities to the risks arising from the invasion of the vector,³ but perhaps more could have been done to prevent its establishment and spread.

The options for prevention are limited as no vaccine exists. Better surveillance is needed, if only to ensure that cases are given appropriate attention and care, but surveillance alone is unlikely to curb transmission. Human cases of chikungunya and dengue are viraemic and infective to mosquitoes early on in the disease course, so that prompt isolation of cases may not prevent onward transmission. In malaria, by contrast, humans are infectious to mosquitoes later in the course of the disease, and local cases in Europe are likely to be diagnosed and treated before the infection reaches this stage. That is probably one reason why the thousands of imported cases of malaria that enter Europe each year have not triggered local malaria epidemics, despite the presence of suitable vectors.

Control of transmission can probably be achieved only by measures directed against the vectors. We cannot stop people going to endemic areas, but education about the risks and methods of personal protection may help. Control of vector populations in Italy will certainly be more difficult and expensive now than it would have been in 1991 before the mosquito had spread over the whole country. The only effective long term approach is to suppress and eliminate the breeding sites, which is difficult to do thoroughly because there are many sites, which are usually small and scattered. Nevertheless, the longer we delay the harder it will be.

Finally, as well as focusing on vector control in Italy, European health authorities could consider whether European Union support for vector control efforts in areas where chikungunya is endemic might also benefit European citizens at home.

Footnotes

- This article was posted on bmj.com on 18 September 2007: <http://bmj.com/cgi/doi/10.1136/bmj.39342.563310.80>
- Competing interests: None declared.

- Provenance and peer review: Commissioned; not externally peer reviewed.

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