Global risk of Zika virus depends critically on vector status of Aedes albopictus

Constância F J Ayres recently pointed out that Zika virus has been collected from several mosquito species including those from the genera, Anopheles, Culex, and Mansonia besides Aedes. Moreover, at least ten Aedes species are known to harbour Zika virus. However, the presence of the virus does not automatically make the species an efficient vector for the disease. It is, therefore, unfortunate that a recent risk map published in The Lancet considers Aedes aegypti and Aedes albopictus together. On the same basis, WHO has predicted that the virus will establish itself in all countries in the Americas except Canada and Chile. However, while the vectorial competence of A aegypti is well established, that of A albopictus is not. Although there is evidence of the potential role of A albopictus, there is no quantitative estimate of its efficiency. It is, therefore, useful to conduct a risk analysis that considers two cases: spread driven by A aegypti presence alone and by both species.

In the context of dengue we have previously modelled the habitat suitability for both species globally and integrated the results with air transport data. We quantified the relative risk of Zika virus spread from infected travellers arriving at airports in new regions by aggregating incoming air travel from infected areas with vector habitat suitability at the destination. The habitat suitability models used a standard maximum entropy algorithm. Source airports were defined as those in areas with autochthonous Zika virus transmission as of Feb 15, 2016, according to the US Centers for Disease Control and Prevention and with vector suitability greater than 0·5 (on a 0–1 scale). Travel statistics from the International Air Transport Association were used to generate relative passenger travel volumes, which include direct and indirect routes (with stopovers). Habitat suitability at each airport was aggregated to circles with a radius of 50 km.

The following inferences can be drawn from the resulting relative risk map (figure). First, if A aegypti is the only competent Zika virus vector, then risk is geographically restricted; in North America to Florida, Louisiana, and Texas. Second, if A albopictus is a competent vector, then there is risk of autochthonous transmission cycles in Canada, Chile, much of western Europe, as well as south and east Asia. Third, for all these areas, the risk compounds that from flights originating in other areas historically endemic for Zika virus. These results underscore Ayres’ point that the vector competence of the various potential mosquito species should be a matter of immediate quantitative assessment.

We declare no competing interests.

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Clinical management of pregnant women exposed to Zika virus

We read with interest the recent work about Zika virus in The Lancet Infectious Diseases and The Lancet. Even if still yet to be confirmed, the association between infection during pregnancy and birth defects is alarming. Recommendations for management of pregnant women at risk of Zika virus infection (ie, those living in endemic areas or who travelled to an area with active virus circulation), are urgently needed. In this Correspondence, we, as perinatal and infectious diseases specialists suggest a detailed management algorithm to help health-care providers (appendix). These recommendations should be adapted to local guidelines, as well as to any further updates on Zika virus.

Since 80% of patients infected with Zika virus are asymptomatic, we propose screening is offered to all pregnant women potentially exposed to the virus. The testing method should be carefully chosen according to the presence and timing of symptoms and done in reference centers. Unlike closely related infections (dengue, chikungunya), there is no abrupt onset of symptoms in Zika fever and determination of the timing of illness is challenging. Therefore, we recommend testing of multiple samples for Zika virus RNA and assaying of serum samples. In addition to blood, molecular detection of Zika virus in saliva can increase the detection rate of the virus in the acute phase of the disease, and urine can increase the window of detection. Serological cross-reaction with other flavivirus is frequently observed, especially in secondary flavivirus infections (ie, past infections with another flavivirus), in both IgM detection and neutralisation tests. In endemic countries, laboratory screening might be difficult due to the number of suspected cases, and testing can exceed laboratories’ capabilities.

Ultrasound monitoring of at-risk pregnancies is required independently of maternal Zika virus status and subsequent management needs to be based on the presence of ultrasound anomalies only. Amniocentesis should be done after 6 weeks from exposure and not before 21 weeks’ gestation. Correlation between head circumference in-utero and microcephaly at birth is more accurately measured in the third trimester, although it is still not optimal. Therefore, at least one ultrasound should be done after 28 weeks’ gestation.

In asymptomatic newborn babies from mothers with confirmed Zika virus infection during pregnancy, latent anomalies should be carefully evaluated. Long-term complications of congenital infections with Zika virus are still not known, but as for congenital cytomegalovirus and toxoplasmosis infections, neurological development, eye fundus, and hearing should be assessed. In the presence of birth defects, an alternative diagnosis needs to be excluded, especially other congenital infections, genetic or syndromic anomalies, and perinatal injuries.

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