Session V. Vector insects and transmission of diseases

21 Do we have to get rid of mosquitoes to eliminate malaria?

Markus Gildenhardt, Elena Levashina* Vector Biology Unit, Max Planck Institute for Infection Biology, Berlin. Germany Corresponding author.

E-mail address: levashina@mpiib-berlin.mpg.de (E. Levashina)

Malaria is a local disease with global impact. The fitness of vector-borne Plasmodium parasites, the causative agents of malaria, is closely linked to the ecology and evolution of its mosquito vector. Ongoing adaptive radiation and introgression diversify mosquito populations in Africa. However, whether the genetic structure of vector populations impacts malaria transmission remains unknown.

We discuss below new approaches that gauge the contribution of mosquito species to Plasmodium abundance in nature, with a particular focus on time-series analyses in the context of population genetics and epidemiology [1]. Our data highlighted the importance of focusing vector control strategies on mosquito species that drive malaria dynamics.

Using time-series collections and the econometric approach Granger causality, we demonstrated that the abundance of Plasmodium-infected mosquitoes in a field site in Mali was driven by only one of the two sympatric vectors (Fig. 1). This mosquito species carried a susceptible allele of the known antiparasitic gene TEP1 [2,3], and until now it was resistant to colonization efforts and, therefore, is not the target of current gene drive applications.

Extending such studies to other key components of vectorial capacity and epidemiological and parasitological surveys should ultimately identify patterns, tipping points, and general laws that describe dynamics, emergence, and resurgence of mosquito-borne diseases.

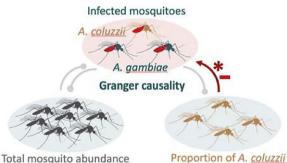


Fig. 1 Granger causality.

Disclosure of interest The authors declare that they have no competing interest.

References

- [1] M. Gildenhard, E.K. Rono, A. Diarra, et al., Mosquito microevolution drives Plasmodium falciparum dynamics, Nat. Microbiol. 4 (6) (2018) 941–947.
- [2] S. Blandin, S.-H. Shiao, L.F. Moita, C.J. Janse, A.P. Waters, F.C. Kafatos, et al., Complement-like protein TEP1 is a determinant of vectorial capacity in the malaria vector Anopheles gambiae, Cell 116 (2004) 661-670.

[3] S.A. Blandin, R. Wang-Sattler, M. Lamacchia, J. Gagneur, G. Lycett, Y. Ning, et al., Dissecting the genetic basis of resistance to malaria parasites in Anopheles gambiae, Science 326 (2009) 147-150.

https://doi.org/10.1016/j.crvi.2019.09.022

22

Human activities and climate change in the emergence of vector-borne diseases Anna-Bella Failloux

Arboviruses and Insect Vectors Laboratory, Department of Virology, Institut Pasteur, Paris, France E-mail address: anna-bella.failloux@pasteur.fr

Because of human population expansion and activities, arthropod-borne viruses (arboviruses) have increased in importance during these last decades. Arboviruses are maintained by alternate replication in both vertebrate hosts and arthropod vectors. Successful transmission relies on a complex life cycle in the vector, which starts when a competent arthropod ingests an infectious blood meal from a viremic vertebrate host. Following an extrinsic incubation time during which the virus replicates in the vector midgut, followed by systemic viral dissemination to the salivary glands, the vector can transmit the virus to a new naïve host. Whereas they typically cause selflimiting, acute infections in their vertebrate hosts, arboviruses establish persistent infections in their vectors.

Arboviruses are typically maintained within an enzootic cycle between wild animals and vectors. As human populations encroach on regions where these diseases are endemic, spillover transmission to humans and domestic animals can lead to large-scale disease outbreaks affecting millions of people. Dengue, chikungunya, Zika, and yellow fever mainly use humans as amplification hosts. Extensive urbanization combined with increased commerce and travel give rise to the mosquitoes Aedes aegypti and Aedes albopictus, both highly adapted to the human environment. The high densities of these human-biting mosquitoes that proliferate in highly populated cities made the bed to explosive outbreaks of arboviral diseases. As insects are ectothermic organisms, climate change may affect the geographical distribution of vectors, with consequences on the transmission of arboviruses.

Yellow fever (YF) is a good example of an emerging arbovirus, as it illustrates three main steps in the emergence: (i) introduction of a new pathogen in a new environment causing urban outbreaks, (ii) spillback of this pathogen into the wild initiating an enzootic cycle, and (iii) the spillover of this pathogen from an enzootic cycle to initiate an urban cycle (Fig. 1).

YF (YFV, Flavivirus, Flaviviridae) is a disease endemic to tropical regions of Africa and South America. There are seven lineages: five in Africa and two in America. Each year, 200,000 cases and 30,000 deaths were reported. In 2016, YFV emerged in Angola and imported cases were detected outside Africa, posing the threat of emergence of this virus outside Africa and Americas (Amraoui et al. Euro Surveill 2016).

YFV was introduced into the New World during the slave trade, causing devastating outbreaks in several American countries, including cities like New York City and Boston. Once Carlos Finlay and Walter Reed had demonstrated that the YFV was transmitted by a mosquito, Ae. aegypti, eradication campaigns of the vector were initiated, leading to the control of YF.

In Brazil, Ae. aegypti was eradicated in 1954. YF disappeared from cities and only persisted in a sylvatic cycle where YFV circulated between zoophilic mosquitoes (Hemagogus and Sabethes) and non-human primates. Using experimental infections, we showed that the zoophilic mosquitoes (Hemagogus leucocelaenus and Sabethes albiprivus) were able to transmit YFV at very high rates (Couto-Lima et al. Sci Rep 2017). With the relaxation of control measures in Brazil, Ae. aegypti was reintroduced in 1967 and Ae. albopictus in 1986. We showed that Ae. aegypti and Ae. albopictus were highly susceptible to YFV. We also demonstrated that using a protocol of experi-