



The Brazilian Journal of INFECTIOUS DISEASES

www.elsevier.com/locate/bjid



Brief communication

Low seroprevalence of Zika virus in Cameroonian blood donors

Bouba Gake^{a,b}, Marie A. Vernet^b, Isabelle Leparc-Goffart^{a,c}, Jan Felix Drexler^d, Ernest A. Gould^a, Pierre Gallian^{a,e}, Xavier de Lamballerie^{a,*}

^a UMR “émergence des Pathologies Virales”, Aix-Marseille Univ, IRD 190, INSERM 1207, EHESP, Marseille, France

^b Centre Pasteur du Cameroun, Yaoundé, Cameroon

^c National Reference Laboratory for Arboviruses, Institut de Recherche Biomédicale des Armées, Marseille, France

^d University of Bonn Medical Centre Institute of Virology, Bonn, Germany

^e Établissement Français du Sang, La Plaine Saint-Denis, France

ARTICLE INFO

Article history:

Received 4 March 2017

Accepted 16 March 2017

Available online xxx

Keywords:

Zika virus

Seroprevalence

Africa

ABSTRACT

A Zika virus seroepidemiology study was performed in 1084 blood donors collected from August to October 2015 in six sites of Cameroon representing a large panel of eco-environments. Samples were tested using an anti-NS1 IgG ELISA detection kit and positives were further confirmed by seroneutralization. The observed global seroprevalence was low (around 5%, peaking at 10% and 7.7% in Douala and Bertoua, respectively) with risk factors associated with seropositivity pointing to the existence of a local (peri-)sylvatic cycle of transmission. These results call attention to the potential introduction and subsequent spread in African urban areas of Asian genotype Zika virus currently circulating in the Americas and adapted to transmission by peri-domestic mosquitoes. They should leverage reinforced surveillance efforts in Africa.

© 2017 Sociedade Brasileira de Infectologia. Published by Elsevier Editora Ltda. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

The Asian genotype of Zika virus (ZIKV) has been responsible for recent outbreaks in the Pacific islands, the Caribbean and South/Central America where severe and formerly undescribed fetal and neurological complications of the disease,^{1,2} as well as non-vectorized routes of transmission,³ have been observed. According to phylogenetic analyses, the Asian genotype of ZIKV emerged out of Africa ~180 years ago.⁴ A striking observation is that the recent Asian genotype Pacific and New

World circulating ZIKV strains are adapted to transmission by the vector *Aedes aegypti*⁵ and that this phenotypic trait is most probably crucial to understand their epidemic potential. By contrast, ZIKV strains belonging to the original African genotype have never been implicated in large outbreaks and the vector competence of African *Aedes aegypti* is low.⁶ Hence, there is considerable need for improving our knowledge about the ecology and epidemiology of the African genotype and in particular estimating the herd immunity of African populations against ZIKV. Although human cases of ZIKV infection have been reported in Africa since the early 1950s, this basic information remains essentially unavailable.

* Corresponding author.

E-mail address: [\(X. de Lamballerie\).](mailto:xavier.de-lamballerie@univ-amu.fr)

<http://dx.doi.org/10.1016/j.bjid.2017.03.018>

1413-8670/© 2017 Sociedade Brasileira de Infectologia. Published by Elsevier Editora Ltda. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

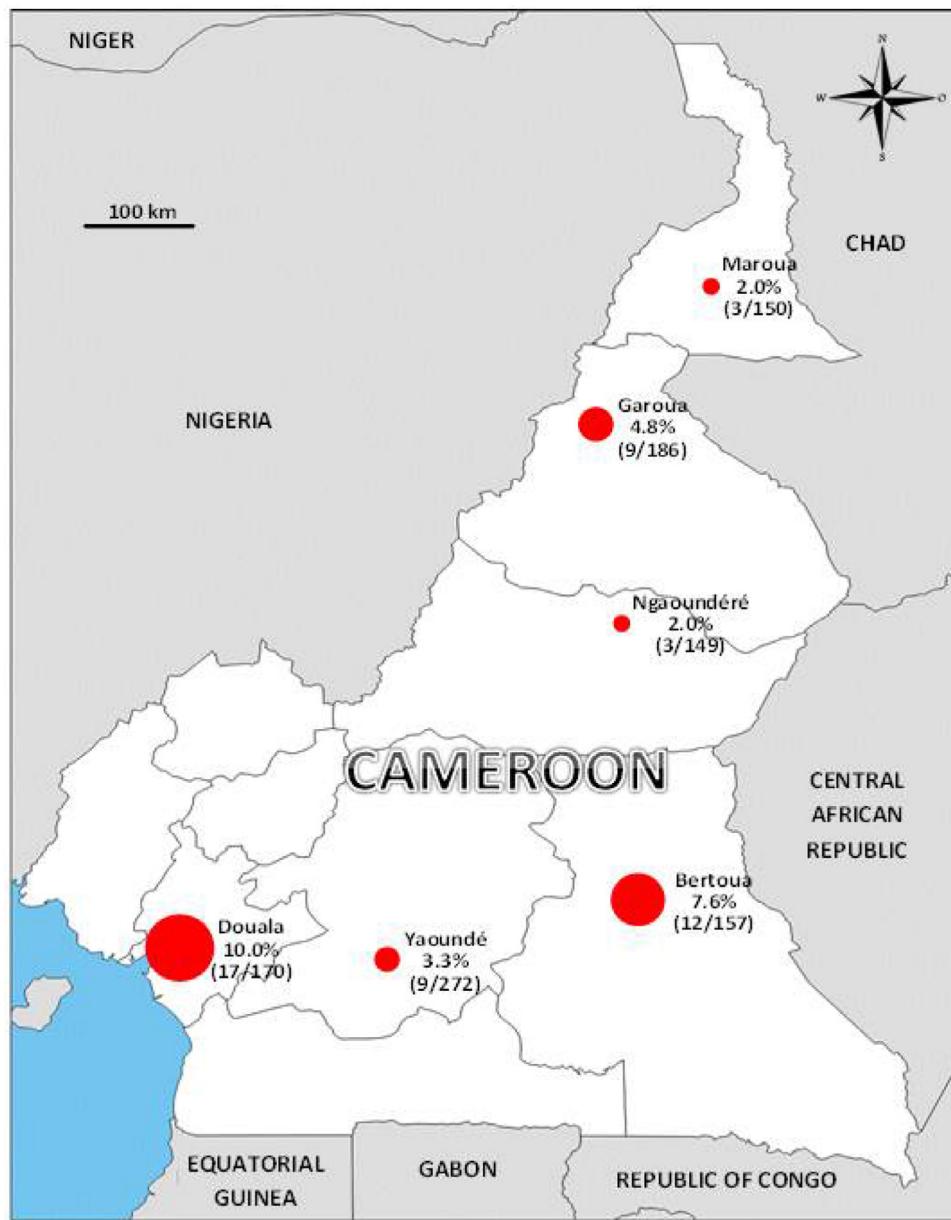


Fig. 1 – Seroprevalence of Zika virus in blood donors from six regions of Cameroon.

In this respect, 1084 blood donors from six sites of Cameroon representing a large panel of eco-environments were enrolled in a ZIKV seroepidemiology study from August to October 2015. They were administered an epidemiological questionnaire and serum samples were tested for the presence of IgG to ZIKV using the EuroImmun anti-NS1 IgG ELISA detection kit and a seroneutralization assay for confirmation of positives, as previously described.⁷ The observed global seroprevalence was low (~5%), peaking at 10% and 7.7% in Douala and Bertoua, respectively, and as low as 2% in Maroua and Ngaoundéré (Fig. 1). In multivariate analysis, the most significant risk factors associated with ZIKV seropositivity were “to be a soldier” ($p=0.021$), a “high distance to the nearest house/shop” ($p=0.042$), and a “previous familial case of

Yellow Fever” ($p=0.001$). Together with the low vector capacity of the African peri-domestic mosquito *Aedes aegypti*,⁶ these risk factors point to the existence of a (peri-)sylvatic cycle of transmission of ZIKV in Cameroon, similar to that of yellow fever virus, rather than to an urban “dengue-like” transmission of the virus.

Altogether, our findings indicate that the immunity of the Cameroonian population against ZIKV is low and that circulation in urban populations is uncommon. Hence, the risk of epidemic spread of ZIKV does exist. The epidemic emergence of the African genotype cannot be totally excluded but it would imply a first and uncertain step of adaptation of the virus to peri-domestic *Aedes* mosquitoes. More worrisome on the short term is the potential introduction in African urban areas

of Asian genotype ZIKV currently circulating in the Americas, as previously suggested by modeling studies.⁸ The virus is likely to be imported by infected travelers coming from epidemic areas and has the potential to be transmitted by local peri-domestic mosquitoes. Our study provides biological evidence that such introduction would occur in populations that are globally immunologically naïve against ZIKV infection and live in areas where potential epidemic vectors exist.

This observation should lead to specific surveillance efforts and to a broader and more systematic mapping of at-risk populations in Africa. It also should ignite interest in investigating similar scientific and public health issues in the Asian population.

Conflicts of interest

The authors declare no conflicts of interest.

Acknowledgements

This work was partially supported by the European Union's Horizon 2020 Research and Innovation Program under ZIKAlliance Grant Agreement no. 734548.

REFERENCES

1. Li H, Saucedo-Cuevas L, Shresta S, Gleeson JG. The neurobiology of Zika virus. *Neuron*. 2016;92:949–58.
2. Schuler-Faccini L, Ribeiro EM, Feitosa IM, et al. Brazilian Medical Genetics Society-Zika Embryopathy Task Force Possible association between Zika virus infection and microcephaly – Brazil, 2015. *MMWR Morb Mortal Wkly Rep*. 2016;65:59–62.
3. Grischott F, Puhan M, Hatz C, Schlagenhauf P. Non-vector-borne transmission of Zika virus: a systematic review. *Travel Med Infect Dis*. 2016;14:313–30.
4. Pettersson JH, Eldholm V, Seligman SJ, et al. How did Zika virus emerge in the Pacific Islands and Latin America? *MBio*. 2016;7.
5. Chouin-Carneiro T, Vega-Rua A, Vazeille M, et al. Differential susceptibilities of *Aedes aegypti* and *Aedes albopictus* from the Americas to Zika virus. *PLoS Negl Trop Dis*. 2016;10:e0004543.
6. Diagne CT, Diallo D, Faye O, et al. Potential of selected Senegalese *Aedes* spp mosquitoes (Diptera: Culicidae) to transmit Zika virus. *BMC Infect Dis*. 2015;15:492.
7. Gallian P, Cabié A, Richard P, et al. Zika virus in asymptomatic blood donors Martinique. *Blood*. 2016, pii: blood-2016-2016 09 737981.
8. Bogoch II, Brady OJ, Kraemer MU, et al. Potential for Zika virus introduction and transmission in resource-limited countries in Africa and the Asia-Pacific region: a modelling study. *Lancet Infect Dis*. 2016;16:1237–45.