Source of Homologous Parasites in Recurrent Plasmodium vivax Malaria

To The Editor—In the group of infants with Plasmodium vivax malaria that was monitored by Imwong et al [1], most of the first recurrences were found to be caused by parasites that were genotypically similar to those that had given rise to the initial clinical symptoms. Alternatively, parasites responsible for recurrent P. vivax malaria can be genetically different from those that caused the first bout of illness [1].

Hypnozoites appear to be directly sporozoite derived [2, 3], and sporozoite inocula may contain more than 1 genotype [4]. Renewed parasitemia of a heterologous nature can therefore be explained (in theory) as a relapse resulting from hypnozoite activation. However, the point has been made that many heterologous recurrences in adults might not derive from the inoculum that gave rise to the previous clinical or parasitemic episode but rather from earlier mosquito inoculation of parasites [1]. In contrast to the heterologous relapse situation, the origin of homologous parasites in the bloodstream in recurrent P. vivax malaria is puzzling. The source in an unknown proportion of cases might be latent merozoites [3, 5] rather than hypnozoites, because the latter origin would require that some inoculated sporozoites of a particular genotype undergo early multiplication in the liver.
and that others simultaneously form hypnozoites. There is not as yet any evidence that genetically similar sporozoites do, in fact, behave in 2 different ways like this.

The pioneering work of Imwong et al [1] represents an important step toward clarification of the source of homologous P. vivax malarial recurrences.

Note

Potential conflict of interest. Author certifies no potential conflicts of interest.

The author has submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Miles B. Markus
University of Witwatersrand, Johannesburg, South Africa, and Biomedical Analysis International, London, United Kingdom

References


Received 4 January 2012; accepted 20 April 2012; electronically published 13 June 2012.
Correspondence: Miles B. Markus, Biomedical Analysis International, 27 Old Gloucester Street, London WC1N 3XX, UK (medsynth@yahoo.co.uk).

The Journal of Infectious Diseases 2012;206:622–3 © The Author 2012. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.
DOI: 10.1093/infdis/jis393