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COMMENTARY

Malaria attack in Southeastern Brazil: a probable locally acquired new infection

Denise Anete Madureira de Alvarenga, MSc[1], Anielle de Pina-Costa, PhD[2,3], Patrícia Brasil, MD, PhD[3], Cristiana Ferreira Alves de Brito, PhD[1]* and Cláudio Tadeu Daniel-Ribeiro, MD, PhD[2]*

[1]Laboratório de Malária, Centro de Pesquisas René Rachou – Fiocruz, Belo Horizonte, Minas Gerais, Brazil; [2]Laboratório de Pesquisa em Malária, Instituto Oswaldo Cruz – Fiocruz, Rio de Janeiro, RJ, Brazil; [3]Laboratório de Doenças Febris Agudas, Instituto Nacional de Infectologia – Fiocruz, Rio de Janeiro, RJ, Brazil

*Correspondence to: Cristiana Ferreira Alves de Brito, Laboratório de Malária, Centro de Pesquisas René Rachou – Fiocruz, Av. Augusto de Lima 1715, CEP 30.190-002, Belo Horizonte, Minas Gerais, Brazil, Email: cristiana@cpqrr.fiocruz.br; Cláudio Tadeu Daniel-Ribeiro, Laboratório de Pesquisa em Malária, Instituto Oswaldo Cruz – Fiocruz, Av. Brasil, Brasil 4365, CEP 21.040-360, Rio de Janeiro, RJ, Brazil, Email: malaria@fiocruz.br

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Sir, we read with great interest the quite literary narrative by Woodall (*Infection Ecology & Epidemiology*, 2016; 6: 30139) reporting his own case of two possibly related episodes of tertian fever separated by 51 years on two continents (1). The first episode was parasitologically confirmed as *Plasmodium vivax* malaria in Malaysia in 1956. The second one, according to the author, which could have been caused by a long-delayed relapse, corresponds to a new infection with *P. simium*, or may not be due to malaria at all. Woodall has stated to welcome correspondences. Here, we discuss the reasons as to why we believe that, accepting the clinical and epidemiological descriptions he offered, it could have probably been an episode of a new malaria infection caused by *P. vivax* or even by the simian *P. simium* in Lumiar (municipality of Nova Friburgo) under the influence of the Atlantic Forest (AF).

Concerning the possibility of a long-delayed relapse, there are some points to consider. Relapse is a common feature of *vivax* malaria, defined as the reappearance of the disease and parasitemia after initial elimination of blood forms. It is caused by the survival of hypnozoites (dormant liver forms of *P. vivax* or *P. ovale*). It is well-known that *P. vivax* relapses can occur within months of the initial infection, with a maximum recorded time of 4 years (2–5), and the references used by Woodall to support the long-latency relapse were, in fact, about long incubation periods of *P. malariae*, which do not cause relapse. There is, therefore, no literature to support delayed *vivax* malaria relapse by 51 years, which is more than 12 times the maximum timeframe ever recorded.

There is also no epidemiological support for such suspicion since the area where the second malaria episode occurred is a region known for 'bromeliad malaria', influenced by the AF and infested by malaria parasites (6). Although malaria transmission has been essentially eliminated in south and southeastern Brazil [where the state of Rio de Janeiro (RJ) is located], few autochthonous malaria cases and outbreaks have been reported to be associated with the AF environment, particularly in the valleys of southeast Brazil (7).

The municipality of Nova Friburgo, a mountainous tourist region that receives many visitors each year – including Woodall and his wife in 2007 – is known for autochthonous *P. vivax* malaria transmission, and it accounted for almost 40% of all (99) autochthonous malaria cases reported in RJ from 2007 to 2016. Lumiar, where Woodall would have had his second infection, is a district in Nova Friburgo where malaria cases have been reported most frequently in RJ each year since 1993 (7, 8): 21 cases from 2003 to 2013 (including 4 in 2007, when Woodall got infected); 15 in 2015, and 9 in 2016. A study conducted by Azevedo (9) found prevalences of 47.8 and 35.4% of anti *P. vivax* IgG antibodies among residents in this region in 1993 and 1996, respectively. This study also reported the presence of *Anopheles (Kerteszia) cruzii*

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mosquitoes in this area (9). Therefore, if the second set of Woodall's recurrent febrile episodes was due to malaria, it would have probably been among the locally acquired cases.

One other aspect pointed out by Woodall that deserves attention is the possibility of an infection by *P. simium*, a parasite found in simians. Deane and others have suggested that the transmission of simian *plasmodia* to humans could be associated with the 'bromeliad malaria' ecology because of the acrodendrophilic behavior and vertical dispersal of *Anopheles (Kerteszia) cruzii* that breeds in water accumulated in the axils of shaded and epiphyte bromeliads (6, 7, 10, 11). In 1966, Deane described the first human natural infection with *P. simium* (Horto Florestal, São Paulo, Brazil) proving that simian malaria was actually being transmitted in AF areas (12). Therefore, at least some of the human malaria cases recorded in the AF could correspond to a zoonosis with non-human primates acting as reservoirs. Two species of *Plasmodium,* namely *P. brasilianum* and *P. simium*, are responsible for simian malaria in the forests of Brazil. These species appear to be morphologically, genetically, and immunologically indistinguishable from the human malaria parasites *P. malariae* and *P. vivax*, respectively (6), and both *P. brasilianum* and *P. simium* infect humans (12). Then, if the febrile accesses presented by Woodall in Lumiar do really correspond to a new malaria episode, it could be a case of simian malaria.

Another aspect mentioned by Woodall concerns the use of chemoprophylaxis that is neither necessary nor indicated by the Brazilian Ministry of Health for Rio de Janeiro that, as the other 17 states of the Extra-Amazon, is not considered an endemic area, or even for the Amazonian endemic region, where more than 85% of the cases are due to *P. vivax*, and where a huge free parasitological diagnostic network does exist.

In conclusion, in view of: 1) the typical tertian characteristics of his febrile illness; 2) the non-existence of reports of vivax relapse 51 years after a primary infection; 3) the existence of intermittent reports of autochthonous malaria cases in Lumiar, the area visited by Dr Woodall; 4) his positive serology for both IgM and IgG; and 5) our 10-year experience on the clinical and epidemiological aspects of malaria in this region, we believe that the intriguing febrile illness presented by Dr Woodall resulted probably from a new malaria infection, caused either by *P. vivax* or even *P. simium*.

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