**Plasmodium malariae**

*Plasmodium malariae* is a parasitic protozoa that causes malaria in humans. It is one of several species of Plasmodium parasites that infect humans including *Plasmodium falciparum* and *Plasmodium vivax* which are responsible for most malarial infection. While found worldwide, it is a so-called “benign malaria” and is not nearly as dangerous as that produced by *P. falciparum* or *P. vivax*. It causes fevers that recur at approximately three-day intervals (a quartan fever), longer than the two-day (tertian) intervals of the other malarial parasites, hence its alternate names quartan fever and quartan malaria.

A human parasite causing quartan malaria.

Each year, approximately 500 million people will be infected with malaria worldwide. Of those infected, roughly two million will die from the disease. Malaria is caused by six Plasmodium species: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale curtisi*, *Plasmodium ovale wallikeri*, *Plasmodium malariae* and *Plasmodium knowlesi*. At any one time, an estimated 300 million people are said to be infected with at least one of these Plasmodium species and so there is a great need for the development of effective treatments for decreasing the yearly mortality and morbidity rates.

*P. malariae* is the one of the least studied of the six species that infect humans, in part because of its low prevalence and milder clinical manifestations compared to the other species. It is widespread throughout sub-Saharan Africa, much of southeast Asia, Indonesia, on many of the islands of the western Pacific and in areas of the Amazon Basin of South America. In endemic regions, prevalence ranges from less than 4% to more than 20%, but there is evidence that *P. malariae* infections are
vastly underreported.

Transmission

P. malariae can be maintained at very low infection rates among a sparse and mobile population because unlike the other Plasmodium parasites, it can remain in a human host for an extended period of time and still remain infectious to mosquitoes.

Vector

The vector of transmission of the parasite is the female Anopheles mosquito, but many different species have been shown to transmit the parasite at least experimentally. Collins and Jeffrey report over thirty different types of species, which vary by geographic region. However, there are supposedly no animal reservoirs for Plasmodium malariae.

Incubation period

Information about the prepatent period of P. malariae associated malaria is limited, but the data suggests that there is great variation, often the length of time depending on the strain of P. malariae parasite. Usually, the prepatent period ranges from 16 to 59 days.

Morphology

The ring stages that are formed by the invasion of merozoites released by rupturing liver stage schizonts are the first stages that appear in the blood. The ring stages grow slowly but soon fill one-fourth to one-third of the parasitized cell. Pigment increases rapidly and the half-grown parasite may have from 30 to 50 jet-black granules. The parasite changes various shapes as it grows and stretches across the host cell to form the band form.

Clinical presentation in humans

Plasmodium malariae causes a chronic infection that in some cases can last a lifetime. The P. malariae parasite has several differences between it and the other Plasmodium parasites, one being that maximum parasite counts are usually low compared to those in patients infected with P. falciparum or P. vivax. The reason for this can be accounted for by the lower number of merozoites produced per erythrocytic cycle, the longer 72-hour developmental cycle (compared to the 48-hour cycle of P. vivax and P. falciparum), the preference for development in older erythrocytes and the resulting earlier development of immunity by the human host. Another defining feature of P. malariae is that the fever manifestations of the parasite are more moderate relative to those of P. falciparum and P. vivax and fevers show quartan periodicity. Along with bouts of fever and more general clinical symptoms such as chills and nausea, the presence of edema and the nephrotic syndrome has been documented with some P. malariae infections. It has been suggested that immune complexes may cause structural glomerular damage and that renal disease may also occur. Although P. malariae alone has a low morbidity rate, it does contribute to the total morbidity caused by all Plasmodium species, as manifested in the incidences of anemia, low birth rate and reduced resistance to other infections.
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