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Editorial

Reports of recent events for *Plasmodium falciparum*

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EDITORIAL NOTE

Plasmodium falciparum is a unicellular protozoan parasite of humans, and the deadliest species of *Plasmodium* that causes malaria in humans. The parasite is transmitted through the bite of a female Anopheles mosquito and causes the disease's most dangerous form *falciparum*, malaria. It is responsible for around 50% of all malaria cases. *P. falciparum* is therefore regarded as the deadliest parasite in humans. It is also associated with the development of blood cancer (Burkitt's lymphoma) and is classified as Group 2A carcinogen.

The species originated from the malarial parasite Laverania found in gorillas, around 10,000 years ago. Alphonse Laveran was the first to identify the parasite in 1880, and named it Oscillaria malariae. Ronald Ross discovered its transmission by mosquito in 1897. Giovanni Battista Grassi elucidated the complete transmission from a female anopheline mosquito to humans in 1898. In 1897, William H. Welch created the name *Plasmodium falciparum*, which ICZN formally adopted in 1954. *P. falciparum* assumes several different forms during its life cycle. The human-infective stage are sporozoites from the salivary gland of a mosquito. The sporozoites grow and multiply in the liver to become merozoites. These merozoites invade the erythrocytes (RBCs) to form trophozoites, schizonts and gametocytes, during which the symptoms of malaria are produced. In the mosquito, the gametocytes undergo sexual reproduction to a zygote, which turns into ookinete. Ookinete forms oocytes from which sporozoites are formed.

As of the World Health Organization World Malaria Report 2020, there were 229 million cases of malaria worldwide in 2019, resulting in an estimated 409,000 deaths. Nearly all malarial deaths are caused by *P. falciparum*, and 94% of such cases occur in Africa. Children under five years of age are most affected, accounting for 67% of the total deaths. In Sub-Saharan Africa, almost 100% of cases were due to P. falciparum, whereas in most other malarial countries, other, less virulent plasmodial species predominate.

Although *P. falciparum* is easily recognized by human immune system while in the bloodstream, it evades immunity by producing over 2,000 cell membrane antigens The initial infective stage sporozoites produce circumsporozoite protein (CSP), which binds to hepatocytes. Binding to and entry into the hepatocytes is aided by another protein, thrombospondin-related anonymous protein (TRAP). TRAP and other secretory proteins (including sporozoite microneme protein essential for cell traversal 1, SPECT1 and SPECT2) from microneme allow the sporozoite to move through the blood, avoiding immune cells and penetrating hepatocytes.

During erythrocyte invasion, merozoites release merozoite cap protein-1 (MCP1), apical membrane antigen 1 (AMA1), erythrocyte-binding antigens (EBA), myosin A tail domain interacting protein (MTIP), and merozoite surface proteins (MSPs). Of these MSPs, MSP1 and MSP2 are primarily responsible for avoiding immune cells. The virulence of P. falciparum is mediated by erythrocyte membrane proteins, which are produced by the schizonts and trophozoites inside the erythrocytes and are displayed on the erythrocyte membrane. PfEMP1 is the most important, capable of acting as both an antigen and an adhesion molecule.

According to WHO guidelines 2010, artemisinin-based combination therapies (ACTs) are the recommended first-line antimalarial treatments for uncomplicated malaria caused by *P. falciparum* . WHO recommends combinations such as artemether/lumefantrine, artesunate/amodiaquine, artesunate/mefloquine, artesunate/sulfadoxine-pyrimethamine, and dihydroartemisinin/piperaquine.

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