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Malaria's Hidden Weapon: How *Plasmodium* Transforms Red Blood Cells to Evade and Invade

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Editorial

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Abbreviations

PC: Phosphatidylcholine; PE: Phosphatidylethanolamine; PS: Phsphatidylserine; PfEMP1: *Plasmodium falciparum* Erythrocyte Membrane Protein 1; RBC: Red Blood Cell.

Editorial

Plasmodium, the malaria-causing parasite, alters host red blood cell (RBC) membranes for survival. After entering the RBC, it forms a parasitophorous vacuole and secretes proteins that help transport nutrients and evade immune detection. The parasite also changes the RBC membrane's lipid composition, making it more rigid and prone to vascular blockages, leading to symptoms like anemia. It exports proteins that cause infected RBCs to stick to blood vessel walls, helping them evade spleen clearance and contributing to severe complications like cerebral malaria. These membrane changes allow the parasite to avoid immune detection. Targeting these alterations, such as blocking RBC adhesions or restoring membrane flexibility, could improve malaria treatment. Understanding these processes helps to develop better therapies (Figure 1).

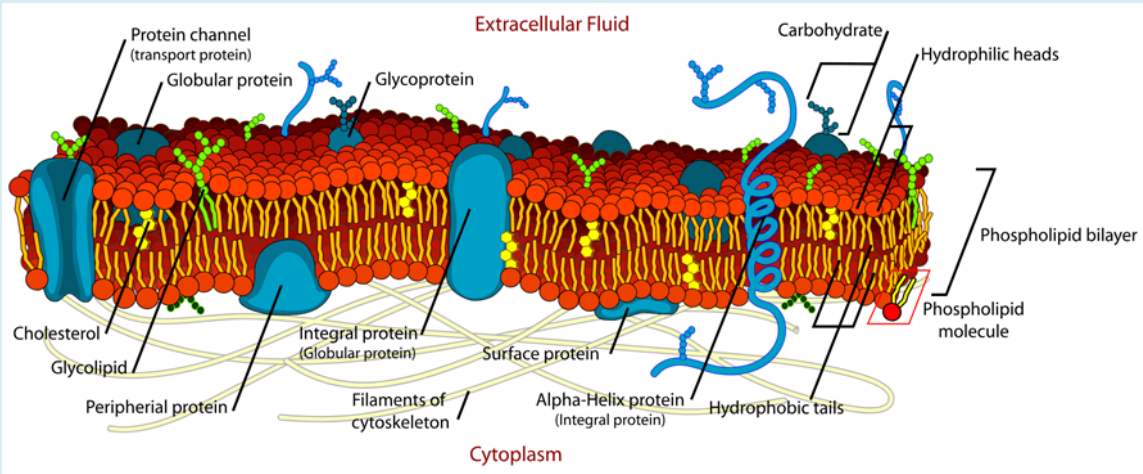

Figure 1: Cell membrane. This image is licensed under creative commons attribution.

Plasmodium infection significantly alters the lipids composition and structural integrity of cell host membranes, impacting red blood cells, hepatocytes, and endothelial cells. Changes in lipids like phospholipids, sphingolipids, and

cholesterol affect membrane fluidity, stability, and flexibility, helping the parasite evade the immune system and enhance its survival. The infection also produces lipid metabolites that trigger inflammatory and immune evasion pathways. These lipid changes disrupt normal cellular functions and facilitate parasite replication, making them potential targets for developing new malaria treatments [1,2].

In *Plasmodium*-infected erythrocytes, lipid composition changes, increasing phosphatidylcholine (PC) and phosphatidylethanolamine (PE), are essential for membrane integrity and parasite growth. They are reducing Phosphatidylserine (PS). The parasite acquires lipids through scavenging from the host and synthesizing them internally, ensuring it meets its metabolic needs. The parasite's membrane, composed of lipids and proteins, supports nutrient exchange, signal transduction, and immune evasion. Lipids also serve as energy sources and signaling molecules, with the parasite manipulating these processes to survive. Disrupting these lipid pathways offers potential for antimalarial therapies [3-7].

Plasmodium, the parasite responsible for malaria, disrupts key processes in red blood cells by altering membrane proteins and transporters. These changes lead to ion imbalances, nutrient deprivation, and weakened immune responses, damaging the host cells. Early infection in liver cells also contributes to symptoms like fever, anemia, and organ dysfunction. *Plasmodium*'s manipulation of membrane proteins can lead to drug resistance by affecting how antimalarial drugs are processed. Understanding these disruptions helps to develop treatments that restore cellular balance, block nutrient access, and combat drug resistance, especially in resistant strains [8-11].

Plasmodium parasites, responsible for malaria, modify red blood cell (RBC) membranes to promote their survival and growth. They change the membrane's lipid composition, increasing its rigidity and permeability, leading to RBC fragility and anemia. The altered permeability aids nutrient absorption and waste removal by the parasite. *Plasmodium* also disrupts membrane proteins, affecting ion exchange, cell signaling, and vesicular trafficking, further weakening the RBC. Insights into these manipulations could help develop treatments targeting these disruptions to fight malaria [12,13].

During *Plasmodium* infection, red blood cell (RBC) membranes undergo several critical changes contributing to malaria pathogenesis. The parasite alters RBC lipid composition, making the membrane more rigid and unstable, leading to the sequestration of infected cells in blood vessels, which worsens the disease. *Plasmodium* modifies host membrane proteins, such as PfEMP1, to avoid immune detection and disrupt nutrient transport, affecting cellular functions. Changing membrane fluidity and permeability help the parasite survive, evade the immune system, and contribute to inflammation. Modified membranes make infected cells adhere to tissues, blocking blood flow and causing severe symptoms like cerebral malaria. Targeting these membrane alterations could help restore normal cell function, disrupt parasite-host interactions, and lead to better treatments for malaria [14-17].

Plasmodium extensively modifies host red blood cell (RBC) membranes to ensure its survival and replication, causing significant alterations in lipid composition, membrane rigidity, and protein function. These modifications enhance nutrient uptake and immune evasion while also driving the development of severe malaria symptoms, including anemia and vascular blockages. Understanding how *Plasmodium* manipulates host cell membranes and disrupts cellular functions helps identify new therapeutic targets. Potential treatments could focus on reversing these membrane alterations, blocking cell adhesions, or inhibiting lipid pathways critical for the parasite's survival. This approach holds promise for enhancing malaria treatment and managing drug-resistant strains, paving the way for more effective interventions against this life-threatening disease [18-43].

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