

Genetic manipulate: separation of host protein participation in multiple parts of malaria parasite pathology

Ms. Madhu Mishra, Dept. of Bioscience
Rabindranath Tagore University, Bhopal

Abstract

This paper investigating the role of host erythrocyte proteins play in malarial infection by genetic intractability of anucleate cell. Reticulocytes derived from enucleating-competent immortalized erythroblast cell line support both successful invasion and intracellular development of malarial parasite *Plasmodium falciparum*. An essential role for the erythrocyte receptor basigin in *P.falciparum* attack and determine rescue of hostile susceptibility by receptor reexpression using CRISPR-method. Reticulocytes use resultant from preserved erythrocytes as a model to discover theories regarding host receptor necessities for *P.falciparum* attack.

Keywords

Erythrocytes, *P.falciparum*, malaria

Introduction

Malaria is an infectious enormous economic and health burden disease caused by *Plasmodium* parasites. This disease is caused by attack of parasite into development within red blood cells. An interaction between a merozoite and the RBC surface begins the attack which results diffusion and intracellular vacuole formation via mechanisms that is partly understood. It is very difficult to investigate host protein participation in red blood cells attack is the complexity of anucleate cell as a system for genetic manipulation. The identification of blood donors provide awareness is incompetent and stops hypothesis-driven investigation of protein involvement in attack. It is possible to make enucleated reticulocytes with unusual or new phenotypes to study host cell protein necessities and association in attack by lentiviral transduction of undeveloped nucleated erythroblast precursors prior to diversity. Generation of preserved erythroid cells capable to boom indefinitely in an undistinguishable state whilst keeping the capacity to feel diversity to make reticulocytes has been a major goal of the erythroid biology field for years. The characterization ability of orthochromatic erythroblasts is done by their compact nuclei to sustenance malarial parasite entry which lead to investigation of cell lines incapable to complete distinction as a model for attack. These cells show a nucleated polychromatic erythroblast-like morphology and notwithstanding associate parasite attack were not able to support further

parasite development. These cells can provide into the requirement of receptors such as entry, basigin for addition and entry significant membrane remodeling and decline of membrane protein profusion that occur previous to and during erythroblast enucleation means that explanations made using this model may not conclude well to enucleate red blood cells. Sustain attack and growth of *P.falciparum* by variation of the BEL-A cell line to generate.

Different malaria species have very complex lifecycle. An infected female *Anopheles* mosquito bites the vertebrate host and injects parasite forms which are known as sporozoites inside the dermis. Some of the parasites allow intense movement in the dermis to encounter blood and lymphatic vessels that causes a decrease in movement. The parasites that enter the lymphatic system attack the nearby lymph node and convert into a stage alike to the hepatic step. To thorough the process of division or reach cell maturity they are doubtful. Recently by Amino et al. (2006) the extraerythrocytic stage was described, but its importance in founding infection and the hosts' immune response is still unidentified. The blood vessels are carried parasites in the blood and reach the hepatocytes after invading these cells. They are improved to a more smoothed form that starts the process of asexual reproduction after forming the hepatic schizont that contains thousands of merozoites which are enlightened into the host circulation. In *P. vivax*, they can differentiate into a dormant stage after the sporozoites invaded in the hepatocytes called a hypnozoite. These can re-initiate replication and lie inactive for months when the original disease has already been healed and leading to another infection, known as relapse. In this way, disease can develop a new infection after cured of the disease. Independence of this particular life stage of *P. vivax*, once merozoites are unconstrained into the bloodstream, they quickly enter red blood cells and start the asexual intraerythrocytic cycle, which causes the disease pathology. It develops into the first stage when the parasite forms a parasitophorous vacuole known as the ring stage. This nourishes on the contents of the red blood cell and converts into a trophozoite. The process of cell division stage is known as schizogony and transforms into the schizont. The red blood cell contains the new merozoites that rupture and released into the bloodstream to infect new cells after continuing the disease cycle. The process of cell division do not begin some parasites and instead, differentiate into the sexual form of the parasites i.e., the male and female gametocytes that will be consumed by a mosquito during its blood meal. The red blood cells are digested and released the male and female gametes once it reached in the digestive tract. The male gametes undergo three rounds of mitosis which give rise to eight cells and phenomenon known as exflagellation and transformed into individual male gametes. The only diploid stage of the parasite forming a zygote after fertilization of the gametes. This differentiates into the ookinete and lodges in the internal wall of the digestive tract between the epithelium, an amoeboid form able to cross the peritrophic membrane and the basal lamina. It forms a protective packet and converts into an oocyst. This undergoes the first dropping meiosis and followed by many stages of mitosis, releasing haploid sporozoites into the haemolymph of the insect. Sporozoites migrate, a new blood meal is done after some of them actively penetrate the salivary gland and after that another individual may be infected.

Results/ Conclusion

Attack of *P.falciparum* and development in BEL-Reticulocytes. Disruption ability and functionally complement phenotypes through exogenous gene expression and genetic knockout is an approach in genetics and cell biology. This technique is directly applicable to red blood cells by their anuclear nature. This paper inaugurates reticulocytes imitative through diversity of the memorialized erythroblast cell line BEL-A as a healthy model system for the investigation of host protein participation in malaria attack

Reference

1. Snow RW, Guerra CA, Noor AM, Myint HY, Hay SI . The global distribution of clinical episodes of *Plasmodium falciparum* malaria. *Nature*. 2005;434(7030):214–217. [PMC free article] [PubMed]
2. Miller LH, Mason SJ, Clyde DF, McGinniss MH. The resistance factor to *Plasmodium vivax* in blacks. The Duffy-blood-group genotype, FyFy. *N Engl J Med*. 1976;295(6):302–304. [PubMed]. Barnwell JW, Wertheimer SP. *Plasmodium vivax*: merozoite antigens, the Duffy blood group, and erythrocyte invasion. *Prog Clin Biol Res*. 1989; 313:1–11.
3. Baum J, Maier AG, Good RT, Simpson KM, Cowman AF. Invasion by *P. falciparum* merozoites suggests a hierarchy of molecular interactions. *PLoS Pathog*. 2005; 1(4):e37.
4. Amino R, Thiberge S, Martin B, Celli S, Shorte S, Frischknecht F, Menard R. Quantitative imaging of *Plasmodium* transmission from mosquito to mammal. *Nat Med*. 2006;12(2):220–224. del Portillo HA, Lanzer M, Rodriguez-Malaga S, Zavala F, Fernandez-Becerra C. Variant genes and the spleen in *Plasmodium vivax* malaria. *Int J Parasitol*. 2004;34(13-14):1547–1554.