Article Title

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Abstract
There's nothing here yet

Contents

1. Introduction

The apicomplexan parasite *Plasmodium falciparum* is the most virulent causative agent of malaria, and responsible for over 600,000 deaths annually [1]. Along with other members of the *Plasmodium* family, *P. falciparum* has a complex lifecycle, moving between several different tissues in both mammalian and arthropod hosts. Symptomatic disease in humans occurs when *P. falciparum* undergoes rounds of asexual reproduction inside human red blood cells (RBCs) [2].

In some respects, the intracellular environment of a red blood cell is an ideal location for parasite proliferation. The cells' lack of an MHC (Major Histocompatibility Complex) system, which would otherwise be used to identify intracellular pathogens to the host immune system, renders parasites immunologically invisible [3], whilst the vascular system allows the parasite to travel throughout the body. The highly specialised nature of RBCs, however, means that the intracellular environment also presents significant challenges to parasite survival.

Red blood cells are a nutritionally poor environment, with a proteome dominated by haemoglobin, which typically accounts for around 98% of the protein content of the cell [4] and contains only a limited amount of several amino acids.

In order to survive and proliferate inside RBCs, *P. falciparum* exports a range of proteins which radically transform the red blood cell, collectively termed the **exportome**.

Recent estimates suggest that at least 10% of the *P. falciparum* genome is exported [5]

Acknowledgments

References

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