Investigating Neutrophil behavior during malaria

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Malaria infection is a global health concern caused by several Plasmodium species, with *Plasmodium falciparum* being the most lethal. During the blood stage of malaria extracellular vesicles (EVs) derived from Plasmodium-infected red blood cells (iRBC) are released into circulation. EVs are pro-inflammatory and immunosuppressive, thereby allowing opportunistic secondary bacterial and fungal infections in malaria patients. Invasive bacterial infections have been associated with high mortality in children with severe malaria in sub-Saharan Africa. While the susceptibility of malaria patients for co-infection with bacteria or fungi is not entirely understood, several reports have suggested that neutrophil dysfunction may play a primary role. In view of this, this thesis elucidated the biological content of iRBC-EVs and their role in dysfunctional behavior in neutrophils such as reduced coordinated migration, reduced ROS production, reduced pathogen phagocytosis, and increased NETosis during malaria. This observed dysfunctional behavior in neutrophils during malaria may perhaps explain why malaria patients remain more susceptible to bacterial and fungal co-infections and understanding this mechanism could potentially lead to more clinical management of bacterial infection during malaria.

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