

MICROB'UP Webinar

Wednesday, November 22nd, 2023

Unveiling the hidden life of parasites

Host Laboratory : Catherine Lavazec

(<https://institutcochin.fr/en/equipes/biology-plasmodium-transmission> ; Institut Cochin, Paris)



22nd Nov 2023



At 11 am
Paris Time (GTM +1)



ZOOM, follow the link bellow

Registration

<https://microb-up.u-paris.fr/webinar/>

11H00

Luisa Miranda Figueiredo  @luisamfigueired

Institute of Molecular Medicine (IMM), University of Lisbon, Portugal

Unveiling the impact of trypanosome infection on adipose tissue

Luisa Figueiredo is a head of laboratory at the Institute of Molecular Medicine, University of Lisbon, Portugal. Her lab studies the cellular and molecular mechanisms underlying host-parasite interactions in sleeping sickness, caused by the parasite *Trypanosoma brucei*. Luisa has been elected an EMBO member in 2023.

11H50

Pauline Formaglio

Institut Pasteur Paris

Escaping the skin: Plasmodium sporozoites' quest for the bloody Grail

Pauline is a microscopy enthusiast with a special interest for host-parasite interactions. Ever since her PhD, she has been implementing intravital imaging approaches to investigate the mechanisms of parasite dissemination and immune containment within host tissues using rodent models of malaria (Institut Pasteur, Paris) and cutaneous leishmaniasis (Otto-von-Guericke

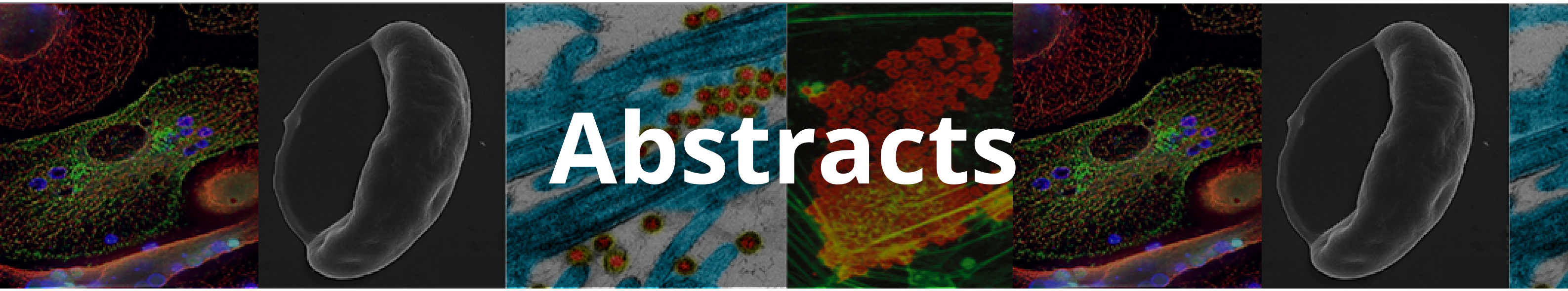
12H10

Tatyana Tavella

Institut Cochin, Paris

Characterization of erythroblast infection by Plasmodium falciparum

Tatyana is a pharmacist from the University of Sao Paulo, Brazil, with a PhD in Drug Discovery focused on antimalarials from the University of Campinas, including a sandwich period at Melbourne University. She is currently working as a postdoctoral researcher in Catherine Lavazec's lab, focusing on understanding and characterizing erythroblast infection by *Plasmodium falciparum*



Abstracts

Luisa Miranda Figueiredo

Institute of Molecular Medicine
(IMM), University of Lisbon, Portugal

*Unveiling the impact of trypanosome
infection on adipose tissue*

African trypanosomes, the causative agent of sleeping sickness, colonize several organs, including the adipose tissue. A typical sign of this disease is extreme weight loss. In this talk, we will discuss our studies in mice to understand the mechanism responsible for adipose mass loss and its impact on disease pathology.

Pauline Formaglio

Institut Pasteur

*Escaping the skin: Plasmodium
sporozoites' quest for the bloody Grail*

Malaria infection starts with the inoculation of Plasmodium sporozoites into the mammalian host skin during a mosquito bite. Within minutes, sporozoites start migrating in the dermis and actively invade blood vessels, thus gaining access to the circulation and the liver, where they infect and multiply within hepatocytes. Although crucial for the successful establishment of an infection, the cutaneous steps leading to sporozoite hematogenous dissemination have remained poorly characterized so far. Using quantitative and functional intravital imaging in a rodent malaria model in combination with statistical analysis methods, we highlight how sporozoites overcome the constraints imposed by host skin tissue and optimally find blood vessels scattered in the dermis to intravasate at hotspots delineated by the presence of pericytes.

Tatyana Tavella

Institut Cochin

*Characterization of erythroblast
infection by Plasmodium falciparum*

In Plasmodium falciparum, only mature gametocytes are found in the bloodstream, while immature gametocytes are enriched in the bone marrow. We recently showed that gametocytes can fully develop inside erythroblasts, which are nucleated, immature red blood cells (RBCs) located in the bone marrow parenchyma. This sheds light on the potential discovery of a new host cell for P. falciparum.

Albeit the remodeling of erythrocytes by P. falciparum gametocytes is well characterized, how gametocytes modify their erythroblast host is yet to be elucidated.

Erythroblasts contain more transport/channel proteins than their mature counterparts, and their nutrient-rich microenvironment might dispense the need for the parasite to develop New Permeability Pathways (NPPs) for nutrient acquisition or waste removal. Furthermore, parasites adapted to erythroblasts may not require mechanisms for mechanical retention since erythroblasts are significantly more rigid than erythrocytes and they naturally adhere to a macrophage in the bone marrow. From the drug discovery perspective, cellular host microenvironment can alter drug sensitivity. Thus, addressing drug susceptibility of gametocytes in erythroblasts in comparison with gametocytes in erythrocytes is important for the surveillance of antimalarial drug resistance.

In this study, we found evidence that trophozoites infecting erythroblasts can create NPPs and increase the rigidity of their host cell, similarly to what happens in erythrocytes. However, we did not observe any modification of host mechanical properties in erythroblasts infected with gametocytes, unlike what occurs in gametocyte-infected reticulocytes and erythrocytes. We also found that gametocytes developing in erythroblasts were ~10-fold less susceptible to dihydroartemisinin (DHA) than those developing in erythrocytes.

Altogether, our data suggest that gametocytes, rather than trophozoites, may be able to adapt to erythroblasts, providing advantages for the parasite.