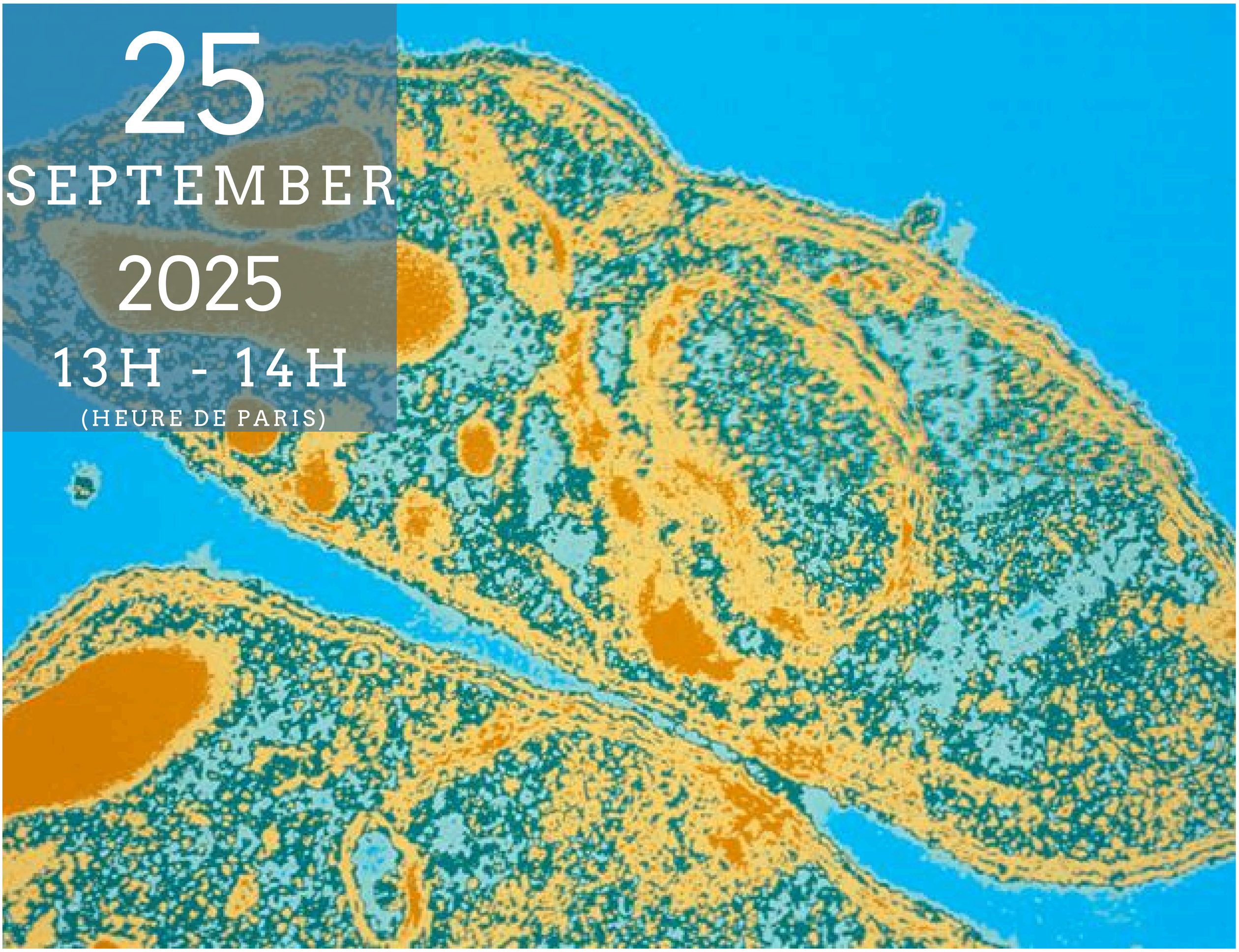

SAVE THE DATE !

Webinaire

Understanding cell cycle checkpoints in malaria parasites

-

Ana Rita Gomes

Inserm
La science pour la santé
From science to health

Inserm Institut thématique Immunologie, inflammation, infectiologie et microbiologie

Ana Rita Gomes

Ana Rita Gomes is a research leader at the Laboratory of Pathogens and Host Immunity (CNRS / University of Montpellier). Trained as a microbiologist, she specialized during her career in parasitology. During her PhD at the Wellcome Sanger Institute in the UK, she focused on functional genetics, developing large-scale reverse genetics screening technologies that enable the study of gene function. These approaches paved the way for high-throughput research on the malaria parasite, allowing the simultaneous analysis of dozens of genes in parallel.

After joining CNRS in 2018, she centered her research on DNA biology in the deadliest malaria parasite, Plasmodium falciparum. Unlike model organisms such as yeast or mammalian cells, malaria parasites divide in an unconventional manner, producing not two but dozens of daughter cells in a single cell cycle. Her goal is to unravel how this atypical multiplication process takes place and how the parasite replicates its DNA, with the aim of identifying vulnerabilities that could be exploited as new therapeutic strategies.

WEBINAIRE

Understanding cell cycle checkpoints in malaria parasites

Unlike model organisms, malaria parasites divide in unconventional ways producing not just two but dozens of daughter cells, in a single cell cycle round. This points to a yet-to-be-explored original and divergent cell cycle architecture where conventional rules likely do not apply. Our team’s focus is to elucidate how these parasites undergo and control cell cycle progression. Specifically, we aim to determine whether malaria parasites deploy checkpoint responses when faced with generalised DNA stress, during active DNA replication. Using a combination of phosphoproteomics, scRNA-seq and live cell imaging, we have defined a stalled state that is triggered by DNA stress. Notably, this state is reversible when the stress is resolved much alike canonical checkpoint responses. In the absence of conserved cell cycle regulators, we now seek to dissect the underlying regulatory networks and identify key factors driving this response in the atypical cell cycle of malaria parasites.