'HUMAN PATHOLOGICAL MECHANISMS IN DROSOPHILA' AXIS.

- In collaboration with Maxime Breban's team (IRIS, U1173 INSERM/UVSQ), we are using the *Drosophila* model system to study the activities of the HLA-B27 antigen, the main genetic predisposing factor for ankylosing spondylitis, an inflammatory rheumatic disease (Breban et al. 2021). This study identified a specific HLA-B27 interactor in *Drosophila*, which turned out to have a human homolog also capable of interacting with HLA-B27, a receptor in the TGF/Activin/BMP pathways. Functional studies were able to show the ability of HLA-B27 to deregulate these signaling pathways in patient cells, which could explain aspects of ankylosing spondylitis (Grandon et al. 2019). This project continues with the aim of further characterizing these deregulations.

- In collaboration with Jean-Louis Herrmann's team (EPIM , U1173 INSERM/UVSQ), we are using *Drosophila* as a model organism for infection by *Mycobacterium abscessus*, in particular, to identify factors enabling Mycobacterium abscessus to resist the innate immune response and the humoral (antimicrobial peptides) or cellular (plasmatocytes)

immune response during infection (Touré *et al*. 2023a and 2023b).

- In collaboration with Eugénie Huillet (UMR Micalis, INRAE de Jouy-en-Josas), we use the Drosophila model to study the protective effect of bacterial supernatants and lysates on intestinal physiology and immune response in the context of inflammation and dysbiosis following infection.