Formulaire de stage (sur une page maximum)
Parcours M2 GGBS 2021-22

Laboratoire : UMR1064 Centre de Recherche en Transplantation et Immunologie

Intitulé/N° d'équipe : Dendritic cells and immunoregulation in transplantation and immunopathology, Team 1 (Chiffoleau Elise/Josien Régis)

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Titre du stage : Investigate the role of the C-type lectin receptor CLEC-1 in myeloid cells in initiation and in resolution of inflammation induced by tissue damage.

Résumé du projet proposé:
C type Lectin-like Receptors (CTLR) represent subtypes of Pattern Recognition Receptors (PRRs) expressed mostly by myeloid cells that can be triggered by self-proteins to fine tune regulate homeostasis and T cell immunity during sterile inflammation. We showed that the orphan C-type Lectin receptor CLEC-1 acts in dendritic cells (DCs) as an immune checkpoint during initiation of sterile inflammation by preventing downstream CD4+ T cell activation (Lopez-Robles et al. Blood Adv 2017) (patent EP16306381). Our recent data suggest that CLEC-1 binds to DAMP(s) released by secondary necrotic cells and prevent also the cross-presentation of dead cell-associated antigens to CD8+ T cells by DC. Therefore, we aim now to investigate the role of CLEC-1 in human and mouse myeloid cells in the initiation and in the resolution of inflammation following tissue damage such as in models of liver intoxication by Acetaminophen and of E. Coli induced pneumonia (collaboration C Jacqueline/A Roquilly, EA3826) by using in vitro assays, flow cytometry analysis, in vivo experimental mouse models and high dimensional approaches (single cell RNA sequencing).

Titres et travaux :

-Cell-surface C-type lectin-like receptor CLEC-1 dampens dendritic cell activation and downstream Th17 responses. Lopez Robles MD, et al. Blood Adv. 2017 Mar 22;1(9):557-568. doi: 10.1182/bloodadvances.2016002360.

-The C-type lectin-like receptor CLEC-1, expressed by myeloid cells and endothelial cells, is up-regulated by immunoregulatory mediators and moderates T cell activation. Thebault P et al. J Immunol. 2009 Sep 1;183(5):3099-108. doi: 10.4049/jimmunol.0803767.