BBB Seminar (BMED382)

SCAN ME

Thursday, May 7. 14:30 at the BBB, Auditorium 4

Targeting the joint stroma in autoimmune arthritis: pathways, pitfalls, and prospects

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Rheumatoid arthritis (RA) is a chronic inflammatory disease and the prototype of a destructive autoimmune arthritis. While immune-mediated mechanisms have long been the primary focus of therapeutic interventions, accumulating evidence highlights a central role of the joint stroma—particularly of synovial fibroblasts (FLS)—as key driver of disease.

This seminar will discuss emerging concepts that position FLS not merely as passive responders, but as active, imprinted effector cells that integrate inflammatory and tissue-derived signals. Building on recent work, we propose that cartilage damage represents a critical event in RA, providing matrix-derived cues that induce a stable activation of FLS in RA. These changes seem to be further triggered by a “second hit” involving loss of immune tolerance and result in pathogenic FLS-phenotypes characterized by metabolic and epigenetic reprogramming.

Recent studies from our group have identified key molecular pathways underlying these processes, including the regulation of cytoskeletal dynamics, cell–cell interactions, and tissue invasion. Importantly, these insights also open new avenues for therapeutic intervention. Strategies aimed at targeting stromal pathways include selective modulation of pathogenic FLS phenotypes, disruption of tissue-driven activation loops, and exploitation of disease-specific matrix alterations for targeted drug delivery. This seminar will discuss both the opportunities and challenges associated with these approaches.

Chairperson: Donald Gullberg, Department of Biomedicine