

Special Issue

Semaphorins as Drivers of Inflammatory Disease: Setting Immune Cells in Motion

Message from the Guest Editor

Semaphorins serve as integral modulators of inflammation. Central signaling axes have emerged such as the Sema7A–Plexin β –C1–integrin α 1 pathway, which increases endothelial permeability and inflammatory activation—and Sema3E–Plexin β –D1, which impairs macrophage egress in atherosclerotic plaques via modulation of cytoskeletal, integrin and PI3K signaling alongside Semaphorin β 5A activation of PI3K–AKT–mTOR through Plexin β A1/B3 in synovial fibroblasts, promoting cytokine release, proliferation, migration and survival. Other family members, including various class β 3 semaphorins, regulate PI3K/Akt upstream and downstream pathways and even modulate PTEN activity depending on receptor context. These nodes underscore the therapeutic relevance of ITGA1/integrin signaling, Plexin B1 and C1 receptors, VEGF/neuropilin interfaces, PI3K–AKT axis regulation, and PTEN β mediated suppression. Built on this mechanistic foundation, this Special Issue calls for deeper dissection of semaphorin signaling at cell β type and spatially resolved levels to define how tightly regulated semaphorin–plexin/neuropilin engagement orchestrates transitions from inflammation to resolution.

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