A distinct human CD4+ T cell subset that secretes CXCL13 in rheumatoid synovitis.

Kobayashi, Shio; Murata, Koichi; Shibuya, Hideyuki; Morita, Mami; Ishikawa, Masahiro; Furu, Moritoshi; Ito, Hiromu; Ito, Juichi; Matsuda, Shuichi; Watanabe, Takeshi; Yoshitomi, Hiroyuki

Arthritis and Rheumatism (2013)

http://hdl.handle.net/2433/178763

© 2013 American College of Rheumatology; この論文は出版社版ではありません。引用の際には出版社版をご確認ご利用ください。This is not the published version. Please cite only the published version.
Supplementary Figure 1. Proposed role of iTh13 cell in ectopic formation of TLO at sites of inflammation.

Left: Follicular dendritic cells (FDCs) recruit CXCR5⁺ B cells and CXCR5⁺ Tfh cells to form the GC in the secondary lymphoid organs (SLO). B helper activity of Tfh cells promotes class switch, and establishment of long-lived memory B or plasma cells. (19, 21, 22, 27).

Right: In the peripheral sites of inflammation such as RA synovial tissues, TCR stimulation and proinflammatory cytokines induce iTh13 cells (Figure 5) and maintained the persistent production of CXCL13 (Figures 4B and C). iTh13 cells modify local chemokine environment coordinately with stromal cells of synoviocytes to form TLO by recruiting CXCR5⁺ B cells and CXCR5⁺ Tfh cells (Figure 6). Presence of TCR stimulation and proinflammatory cytokines may lead to the maintenance of TLO, to the generation of autoantibodies (36,41), and to the enlargement of TLO and surrounding tissues.