Behcet’s disease is a rare form of vasculitis that may have systemic multi-organ involvement. Behcet’s disease was first defined by Hulusi Behcet, a Turkish Professor of Dermatology, in 1937 as a triad of recurrent aphthous stomatitis, genital aphthae, and relapsing uveitis. As this disease can be fatal, an immediate medical treatment is mandatory. BD is currently thought to be an autoimmune/auto-inflammatory syndrome that is possibly induced by infections or other environmental factors. Several studies also showed that polarization of CD41 T cells into TH1 cells and their cytokines, such as IFN-γ and IL-12, play a central role in the pathogenesis of BD. Levels of IL-12, which drives the TH1 response in naïve T cells, are increased in sera from patients with BD in correlation with the presence of TH1 lymphocytes. Tissue studies demonstrated TH1-type intestinal and cutaneous lesions, possibly driven with IL-12 and IL-23. The novel T-cell subsets TH9, TH17, TH22, and regulatory T (Treg) cells and their cytokines are intricately involved in inflammatory disorders.

TH17 cells represent a new subset of TH cells, which mainly produce IL-17A-F, IL-22, and TNF-a. IL-6 and TGF-b induce the differentiation of TH17 cells from naive T cells. Geri et al demonstrated that IL-21 is the major increased cytokine in active patients’ sera compared with IL-6, IL-23, and TGF-b. IL-21 is produced by central memory CD41 T cells and correlated with the TH17 response and decreased fork-head box protein 3 expressions. Finally, the authors also studied local responses and demonstrated the presence of IL-21– and IL-17A–producing T cells within cerebrospinal fluid, brain parenchymal inflammatory infiltrates, and intracerebral blood vessels of patients with active BD with central nervous system involvement. These findings result in a remarkable increase in our current knowledge and might shed light on the treatment of BD.