Th17 cells were recently identified as a cell source of IL-17. They turned up to be a T cell lineage independent of previously described Th1 and Th2. The differentiation of naive CD4+ T cells towards Th17 requires the combination of TGFβ (a cytokine essential for the development of anti-inflammatory regulatory T cells) plus IL-6 or IL-21. IL-23 is required for *in vivo* function and phenotype maintenance of Th17. STAT3 and RORγt were identified as pivotal transcription factors in Th17 differentiation program. Th17 proved to have pro-inflammatory effects and are characterized by the production of IL-17A, IL-17F and IL-22 – cytokines implicated in host defense against certain extracellular pathogens. The cytokine products of Th17 cells act on wide range of cell types. They induce cytokines, chemokines and metalloproteinases and they also mediate neutrophil recruitment and production of antimicrobial peptides. Autoreactive Th17 are highly pathogenic and the production of IL-17 has been detected in several autoimmune diseases such as rheumatoid arthritis, multiple sclerosis, psoriasis, Crohn´s disease and type 1 diabetes. These diseases were thought to be mediated by Th1 cells, but it is becoming increasingly clear that the regulation of autoimmunity is influenced at least in some diseases by Th17 cells as well.