



[24120361](http://www.uniprot.org/citations/24120361), PubMed: [8676080](http://www.uniprot.org/citations/8676080)). Plays an important role in connecting T cell-mediated adaptive immunity and acute inflammatory response to destroy extracellular bacteria and fungi. As a signature effector cytokine of T-helper 17 cells (Th17), primarily induces neutrophil activation and recruitment at infection and inflammatory sites (By similarity). In airway epithelium, mediates neutrophil chemotaxis via induction of CXCL1 and CXCL5 chemokines (By similarity). In secondary lymphoid organs, contributes to germinal center formation by regulating the chemotactic response of B cells to CXCL12 and CXCL13, enhancing retention of B cells within the germinal centers, B cell somatic hypermutation rate and selection toward plasma cells (By similarity). Effector cytokine of a subset of gamma-delta T cells that functions as part of an inflammatory circuit downstream IL1B, TLR2 and IL23A-IL12B to promote neutrophil recruitment for efficient bacterial clearance (By similarity). Effector cytokine of innate immune cells including invariant natural killer cell (iNKT) and group 3 innate lymphoid cells that mediate initial neutrophilic inflammation (By similarity). Involved in the maintenance of the integrity of epithelial barriers during homeostasis and pathogen infection (PubMed: [21350122](http://www.uniprot.org/citations/21350122)). Upon acute injury, has a direct role in epithelial barrier formation by regulating OCLN localization and tight junction biogenesis (By similarity). As part of the mucosal immune response induced by commensal bacteria, enhances host's ability to resist pathogenic bacterial and fungal infections by promoting neutrophil recruitment and antimicrobial peptides release (By similarity). In synergy with IL17F, mediates the production of antimicrobial beta-defensins DEFB1, DEFB103A, and DEFB104A by mucosal epithelial cells, limiting the entry of microbes through the epithelial barriers (By similarity). Involved in antiviral host defense through various mechanisms (By similarity). Enhances immunity against West Nile virus by promoting T cell cytotoxicity (By similarity). May play a beneficial role in influenza A virus (H5N1) infection by enhancing B cell recruitment and immune response in the lung (By similarity). Contributes to influenza A virus (H1N1) clearance by driving the differentiation of B-1a B cells, providing for production of virus-specific IgM antibodies at first line of host defense (By similarity).

**Cellular Location**

Secreted

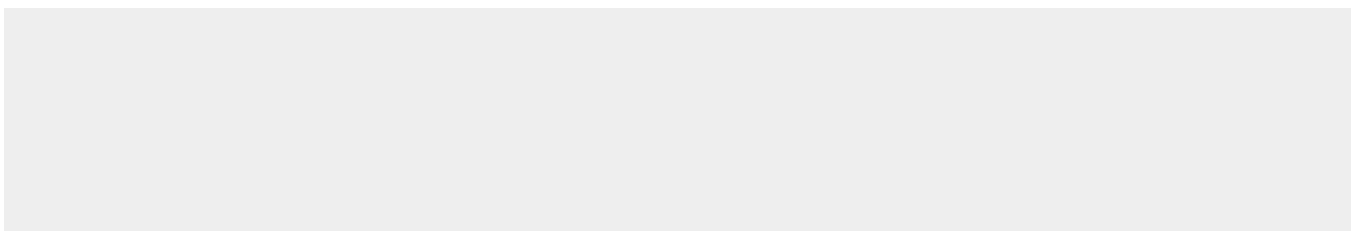
**Tissue Location**

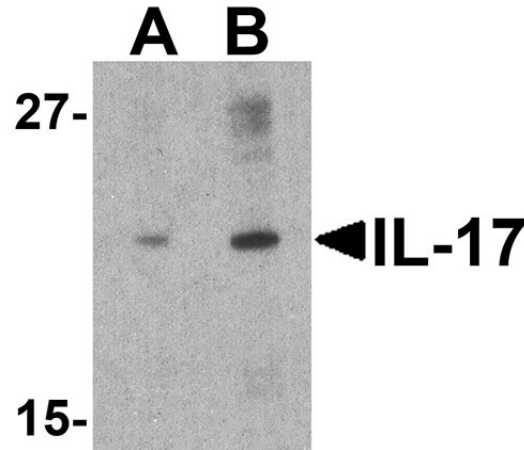
Expressed in memory Th17 cells (at protein level).

**IL-17 Antibody - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**IL-17 Antibody - Images**



Western blot analysis of IL-17 in THP-1 cell lysate with IL-17 antibody at (A) 1 and (B) 2 µg/mL. g/ml.

### IL-17 Antibody - Background

IL-17 Antibody: Interleukin 17 (IL-17) is a family of pro-inflammatory cytokines produced by activated T cells and is thought to have a major role in the initiation and perpetuation of rheumatoid arthritis. IL-17 regulates the activities of NF-κB and mitogen-activated protein kinases such as ERK and JNK. In addition, IL-17 stimulates the expression of IL-6 and cyclooxygenase-2 and enhances the production of nitric oxide. IL-17-producing T helper cells (TH-17 cells) have been the subject of much attention due to the importance of IL-17 in the pathogenesis of autoimmune inflammation. Because of its role in autoimmune diseases, it is thought that targeting the production and action of IL-17 would be beneficial therapeutically in these diseases.

### IL-17 Antibody - References

- Miossec P. Are T cells in rheumatoid synovium aggressors or bystanders? *Curr. Opin. Rheumatol.*2000; 12:181-5.
- Paunovic V, Carroll HP, Vandenbroeck K, et al. Crossed signals: the role of interleukin (IL)-12, -17, -23, and -27 in autoimmunity. *Rheumatol.*2008; 47:771-6.
- Steinman L. A brief history of TH17, the first major revision in the TH1/TH2 hypothesis of T cell-mediated tissue damage. *Nat. Med.*2007; 13:139-145.