

**Mouse IL27 Antibody (Center) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP19313c****Specification**

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**Mouse IL27 Antibody (Center) Blocking Peptide - Product Information**Primary Accession [Q8K3I6](#)**Mouse IL27 Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 246779**Other Names**

Interleukin-27 subunit alpha, IL-27 subunit alpha, IL-27-A, IL27-A, p28, IL27, IL27a

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**Mouse IL27 Antibody (Center) Blocking Peptide - Protein Information****Name** IL27**Synonyms** IL27a**Function**

Associates with EBI3 to form the IL-27 interleukin, a heterodimeric cytokine which functions in innate immunity. IL-27 has pro- and anti-inflammatory properties, that can regulate T-helper cell development, suppress T-cell proliferation, stimulate cytotoxic T-cell activity, induce isotype switching in B-cells, and that has diverse effects on innate immune cells. Among its target cells are CD4 T-helper cells which can differentiate in type 1 effector cells (TH1), type 2 effector cells (TH2) and IL17 producing helper T-cells (TH17). It drives rapid clonal expansion of naive but not memory CD4 T-cells. It also strongly synergizes with IL-12 to trigger interferon-gamma/IFN- gamma production of naive CD4 T-cells, binds to the cytokine receptor WSX-1/TCCR which appears to be required but not sufficient for IL-27- mediated signal transduction. IL-27 potentiate the early phase of TH1 response and suppress TH2 and TH17 differentiation. It induces the differentiation of TH1 cells via two distinct pathways, p38 MAPK/TBX21- and ICAM1/ITGAL/ERK-dependent pathways. It also induces STAT1, STAT3, STAT4 and STAT5 phosphorylation and activates TBX21/T-Bet via STAT1 with resulting IL12RB2 up-regulation, an event crucial to TH1 cell commitment. It suppresses the expression of GATA3, the inhibitor TH1 cells development. In CD8 T-cells, it activates STATs as well as GZMB. IL-27 reveals to be a potent inhibitor of TH17 cell development and of IL-17 production. Indeed IL27 alone is also able to inhibit the production of IL17 by CD4 and

CD8 T-cells. While IL-27 suppressed the development of pro-inflammatory Th17 cells via STAT1, it inhibits the development of anti-inflammatory inducible regulatory T-cells, iTreg, independently of STAT1. IL-27 has also an effect on cytokine production, it suppresses pro-inflammatory cytokine production such as IL2, IL4, IL5 and IL6 and activates suppressors of cytokine signaling such as SOCS1 and SOCS3. Apart from suppression of cytokine production, IL-27 also antagonizes the effects of some cytokines such as IL6 through direct effects on T-cells. Another important role of IL-27 is its antitumor activity as well as its antiangiogenic activity with activation of production of antiangiogenic chemokines such as IP-10/CXCL10 and MIG/CXCL9.

#### **Cellular Location**

Secreted. Note=Poorly secreted without coexpression of EB13

#### **Tissue Location**

Expressed in macrophages and dendritic cells.

### **Mouse IL27 Antibody (Center) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

### **Mouse IL27 Antibody (Center) Blocking Peptide - Images**

### **Mouse IL27 Antibody (Center) Blocking Peptide - Background**

Cytokine with pro-and anti-inflammatory properties, that can regulate T helper cell development, suppress T-cell proliferation, stimulate cytotoxic T cell activity, induce isotype switching in B-cells, and that has diverse effects on innate immune cells. Among its target cells are CD4 T helper cells which can differentiate in type 1 effector cells (TH1), type 2 effector cells (TH2) and IL17 producing helper T-cells (TH17). It drives rapid clonal expansion of naive but not memory CD4 T-cells. It also strongly synergizes with IL-12 to trigger interferon-gamma/IFN-gamma production of naive CD4 T-cells, binds to the cytokine receptor WSX-1/TCCR which appears to be required but not sufficient for IL-27-mediated signal transduction. IL-27 potentiates the early phase of TH1 response and suppresses TH2 and TH17 differentiation. It induces the differentiation of TH1 cells via two distinct pathways, p38 MAPK/TBX21-and ICAM1/ITGAL/ERK-dependent pathways. It also induces STAT1, STAT3, STAT4 and STAT5 phosphorylation and activates TBX21/T-Bet via STAT1 with resulting IL12RB2 up-regulation, an event crucial to TH1 cell commitment. It suppresses the expression of GATA3, the inhibitor of TH1 cell development. In CD8 T-cells, it activates STATs as well as GZMB. IL-27 reveals to be a potent inhibitor of TH17 cell development and of IL-17 production. Indeed IL-27 subunit p28 alone is also able to inhibit the production of IL17 by CD4 and CD8 T-cells. While IL-27 suppressed the development of proinflammatory Th17 cells via STAT1, it inhibits the development of anti-inflammatory inducible regulatory T-cells, iTreg, independently of STAT1. IL-27 has also an effect on cytokine production, it suppresses proinflammatory cytokine production such as IL2, IL4, IL5 and IL6 and activates suppressors of cytokine signaling such as SOCS1 and SOCS3. Apart from suppression of cytokine production, IL-27 also antagonizes the effects of some cytokines such as IL6 through direct effects on T cells. Another important role of IL-27 is its antitumor activity as well as its antiangiogenic activity with activation of production of antiangiogenic chemokines such as IP-10/CXCL10 and MIG/CXCL9.

### **Mouse IL27 Antibody (Center) Blocking Peptide - References**

Li, J.J., et al. J. Immunol. 185(7):4401-4409(2010)Zhang, J., et al. J. Biol. Chem. 285(28):21269-21281(2010)Baker, B.J., et al. Glia 58(9):1082-1093(2010)Murugaiyan, G., et al. Proc. Natl. Acad. Sci. U.S.A. 107(25):11495-11500(2010)Zhu, S., et al. J. Immunol. 184(5):2348-2354(2010)