

**IL-10R $\alpha$  Polyclonal Antibody**  
**Catalog # AP73813****Specification**

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**IL-10R $\alpha$  Polyclonal Antibody - Product Information**

Application	WB
Primary Accession	<a href="#">Q13651</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal

**IL-10R $\alpha$  Polyclonal Antibody - Additional Information****Gene ID** 3587**Other Names**

IL10RA; IL10R; Interleukin-10 receptor subunit alpha; IL-10 receptor subunit alpha; IL-10R subunit alpha; IL-10RA; CDw210a; Interleukin-10 receptor subunit 1; IL-10R subunit 1; IL-10R1; CD210

**Dilution**

WB~~Western Blot: 1/500 - 1/2000. ELISA: 1/10000. Not yet tested in other applications.

**Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

**Storage Conditions**

-20°C

**IL-10R $\alpha$  Polyclonal Antibody - Protein Information****Name** IL10RA**Synonyms** IL10R**Function**

Cell surface receptor for the cytokine IL10 that participates in IL10-mediated anti-inflammatory functions, limiting excessive tissue disruption caused by inflammation. Upon binding to IL10, induces a conformational change in IL10RB, allowing IL10RB to bind IL10 as well (PubMed:<a href="http://www.uniprot.org/citations/16982608" target="\_blank">16982608</a>). In turn, the heterotetrameric assembly complex, composed of two subunits of IL10RA and IL10RB, activates the kinases JAK1 and TYK2 that are constitutively associated with IL10RA and IL10RB respectively (PubMed:<a href="http://www.uniprot.org/citations/12133952" target="\_blank">12133952</a>). These kinases then phosphorylate specific tyrosine residues in the intracellular domain in IL10RA leading to the recruitment and subsequent phosphorylation of STAT3. Once phosphorylated, STAT3 homodimerizes, translocates to the nucleus and activates the expression of anti-inflammatory genes. In addition, IL10RA-mediated activation of STAT3 inhibits starvation-induced autophagy (PubMed:<a href="http://www.uniprot.org/citations/26962683" target="\_blank">26962683</a>).

**Cellular Location**

Cell membrane; Single-pass type I membrane protein Cytoplasm

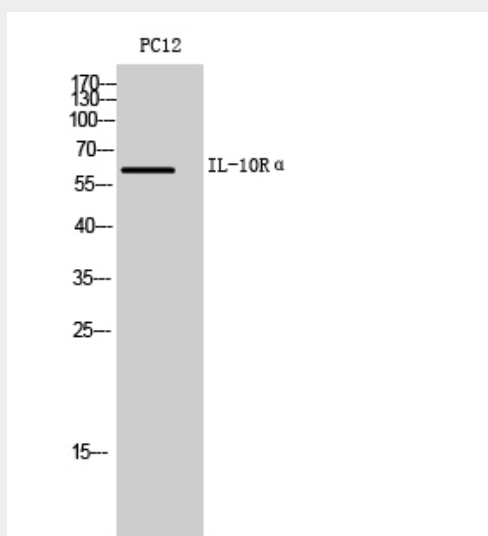
**Tissue Location**

Primarily expressed in hematopoietic cells including B-cells, T-cells, NK cells, monocytes and macrophages. Not expressed in non-hematopoietic cells such as fibroblasts or endothelial cells

**IL-10R $\alpha$  Polyclonal 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-10R $\alpha$  Polyclonal Antibody - Images****IL-10R $\alpha$  Polyclonal Antibody - Background**

Cell surface receptor for the cytokine IL10 that participates in IL10-mediated anti-inflammatory functions, limiting excessive tissue disruption caused by inflammation. Upon binding to IL10, induces a conformational change in IL10RB, allowing IL10RB to bind IL10 as well (PubMed:16982608). In turn, the heterotetrameric assembly complex, composed of two subunits of IL10RA and IL10RB, activates the kinases JAK1 and TYK2 that are constitutively associated with IL10RA and IL10RB respectively (PubMed:12133952). These kinases then phosphorylate specific tyrosine residues in the intracellular domain in IL10RA leading to the recruitment and subsequent phosphorylation of STAT3. Once phosphorylated, STAT3 homodimerizes, translocates to the nucleus and activates the expression of anti-inflammatory genes. In addition, IL10RA-mediated activation of STAT3 inhibits starvation- induced autophagy (PubMed:26962683).