

## IL10 Antibody(Ascites)

Mouse Monoclonal Antibody (Mab) Catalog # AM2094a

#### Specification

# IL10 Antibody(Ascites) - Product Information

Application Primary Accession Other Accession Reactivity Predicted Host Clonality Isotype Calculated MW Antigen Region WB,E <u>P22301</u> <u>P79338</u>, <u>P03180</u>, <u>NP\_000563.1</u> Human Epstein Barr Virus, Monkey Mouse Monoclonal IgM 20517 27-53

## IL10 Antibody(Ascites) - Additional Information

Gene ID 3586

**Other Names** Interleukin-10, IL-10, Cytokine synthesis inhibitory factor, CSIF, IL10

**Target/Specificity** 

This IL10 antibody is generated from mice immunized with a KLH conjugated synthetic peptide between 27-53 amino acids from human IL10.

**Dilution** WB~~1:500~1000 E~~Use at an assay dependent concentration.

**Format** Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions** IL10 Antibody(Ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

# IL10 Antibody(Ascites) - Protein Information

Name IL10

Function Major immune regulatory cytokine that acts on many cells of the immune system where



it has profound anti-inflammatory functions, limiting excessive tissue disruption caused by inflammation. Mechanistically, IL10 binds to its heterotetrameric receptor comprising IL10RA and IL10RB leading to JAK1 and STAT2-mediated phosphorylation of STAT3 (PubMed:<u>16982608</u>). In turn, STAT3 translocates to the nucleus where it drives expression of anti-inflammatory mediators (PubMed:<u>18025162</u>). Targets antigen-presenting cells (APCs) such as macrophages and monocytes and inhibits their release of pro- inflammatory cytokines including granulocyte-macrophage colony- stimulating factor /GM-CSF, granulocyte colony-stimulating factor/G- CSF, IL-1 alpha, IL-1 beta, IL-6, IL-8 and TNF-alpha (PubMed:<u>11564774</u>, PubMed:<u>1940799</u>, PubMed:<u>7512027</u>). Also interferes with antigen presentation by reducing the expression of MHC-class II and co- stimulatory molecules, thereby inhibiting their ability to induce T cell activation (PubMed:<u>8144879</u>). In addition, controls the inflammatory response of macrophages by reprogramming essential metabolic pathways including mTOR signaling (By similarity).

Cellular Location Secreted.

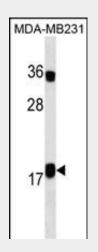
**Tissue Location** Produced by a variety of cell lines, including T- cells, macrophages, mast cells and other cell types

# IL10 Antibody(Ascites) - Protocols

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

- Western Blot
- <u>Blocking Peptides</u>
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- <u>Cell Culture</u>

#### IL10 Antibody(Ascites) - Images



IL10 Antibody (Cat. #AM2094a) western blot analysis in MDA-MB231 cell line lysates (35µg/lane). This demonstrates the IL10 antibody detected the IL10 protein (arrow).

#### IL10 Antibody(Ascites) - Background

The protein encoded by this gene is a cytokine produced



primarily by monocytes and to a lesser extent by lymphocytes. This cytokine has pleiotropic effects in immunoregulation and inflammation. It down-regulates the expression of Th1 cytokines, MHC class II Ags, and costimulatory molecules on macrophages. It also enhances B cell survival, proliferation, and antibody production. This cytokine can block NF-kappa B activity, and is involved in the regulation of the JAK-STAT signaling pathway. Knockout studies in mice suggested the function of this cytokine as an essential immunoregulator in the intestinal tract. [provided by RefSeq].

## IL10 Antibody(Ascites) - References

Huebinger, R.M., et al. J. Surg. Res. 164 (1), E141-E145 (2010) : Glocker, E.O., et al. Lancet 376 (9748), 1272 (2010) : Mosaad, Y.M., et al. Scand. J. Immunol. 72(4):358-364(2010) Kung, W.J., et al. Diabetes Technol. Ther. 12(10):809-813(2010) Santhosh, S., et al. Trop Gastroenterol 31(1):30-33(2010)