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IL1B Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP8531c

Specification

IL1B Antibody (Center) Blocking Peptide - Product Information

Primary Accession P01584

IL1B Antibody (Center) Blocking Peptide - Additional Information

Gene ID 3553

Other Names
Interleukin-1 beta, IL-1 beta, Catabolin, IL1B, IL1F2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8531c was selected from the Center region of human IL1B. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

IL1B Antibody (Center) Blocking Peptide - Protein Information

Name IL1B (HGNC:5992)

Synonyms IL1F2

IL1B Antibody (Center) Blocking Peptide - Background

IL1B is a member of the interleukin 1 cytokine family. This cytokine is produced by activated macrophages as a proprotein, which is proteolytically processed to its active form by caspase 1 (CASP1/ICE). This cytokine is an important mediator of the inflammatory response, and is involved in a variety of cellular activities, including cell proliferation, differentiation, and apoptosis. The induction of cyclooxygenase-2 (PTGS2/COX2) by this cytokine in the central nervous system (CNS) is found to contribute to inflammatory pain hypersensitivity.

IL1B Antibody (Center) Blocking Peptide - References

Yu,J., et.al., Am. J. Gastroenterol. (2009)Ito,A., et.al., J. Biol. Chem. 271 (25), 14657-14660 (1996)

Function

Potent proinflammatory cytokine. Initially discovered as the major endogenous pyrogen, induces prostaglandin synthesis, neutrophil influx and activation, T-cell activation and cytokine production, B- cell activation and antibody production, and fibroblast proliferation and collagen production. Promotes Th17 differentiation of T-cells. Synergizes with IL12/interleukin-12 to induce IFNG synthesis from T- helper 1 (Th1) cells (PubMed:10653850). Plays a role in angiogenesis by inducing VEGF production synergistically with TNF and IL6 (PubMed:12794819).

Cellular Location

Cytoplasm, cytosol. Lysosome. Secreted, extracellular exosome {ECO:0000250|UniProtKB:P10749}. Secreted. Note=The precursor is cytosolic. In response to inflammasome-activating signals, such as ATP for NLRP3 inflammasome or bacterial flagellin for NLRC4 inflammasome, cleaved and secreted. IL1B lacks any known signal sequence and the pathway(s) of its secretion is(are) not yet fully understood (PubMed:24201029). On the basis of experimental results, several unconventional secretion mechanisms have been proposed. 1. Secretion via secretory lysosomes: a fraction of CASP1 and IL1B precursor may be incorporated, by a yet undefined mechanism, into secretory lysosomes that undergo Ca(2+)- dependent exocytosis with release of mature IL1B (PubMed:15192144). 2 Secretory autophagy: IL1B-containing autophagosomes may fuse with endosomes or multivesicular bodies (MVBs) and then merge with the plasma membrane releasing soluble IL1B or IL1B-containing exosomes (PubMed:24201029). However, autophagy impacts IL1B production at several levels and its role in secretion is still controversial. 3 Secretion via exosomes: ATP-activation of P2RX7 leads to the formation of MVBs containing exosomes with entrapped IL1B, CASP1 and other inflammasome components. These MVBs undergo exocytosis with the release of exosomes. The release of soluble IL1B occurs after the

lysis of exosome membranes (By similarity). 4. Secretion by microvesicle shedding: activation of the ATP receptor P2RX7 may induce an immediate shedding of membrane-derived microvesicles containing IL1B and possibly inflammasome components. The cytokine is then released in the extracellular compartment after microvesicle lysis (PubMed:11728343) 5. Release by translocation through permeabilized plasma membrane. This may occur in cells undergoing pyroptosis due to sustained activation of the inflammasome (By similarity). 6. The secretion is dependent on protein unfolding and facilitated by the cargo receptor TMED10; it results in the protein translocation from the cytoplasm into the ERGIC (endoplasmic reticulum-Golgi intermediate compartment) followed by vesicle entry and secretion, and enhanced by chaperones HSP90AB1 and HSP90B1/GRP9 (PubMed:32272059). These mechanisms may not be mutually exclusive. {ECO:0000250|UniProtKB:P10749, ECO:0000269|PubMed:11728343, ECO:0000269|PubMed:15192144, ECO:0000269|PubMed:32272059, ECO:0000305|PubMed:24201029}

Tissue Location
Expressed in activated monocytes/macrophages (at protein level).

IL1B Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides