

Goat Anti-IL-1 beta Antibody (internal region)
Purified Goat Polyclonal Antibody
Catalog # AF4170a

Specification

Goat Anti-IL-1 beta Antibody (internal region) - Product Information

Application	WB
Primary Accession	P01584
Other Accession	NP_000567.1
Reactivity	Human
Predicted	Human
Host	Goat
Clonality	Polyclonal
Concentration	0.5
Calculated MW	30748

Goat Anti-IL-1 beta Antibody (internal region) - Additional Information

Gene ID 3553

Other Names

IL1B; interleukin 1, beta; IL-1; IL1-BETA; IL1F2; IL-1 beta; catabolin; interleukin-1 beta; preinterleukin 1 beta; pro-interleukin-1-beta

Format

Supplied at 0.5 mg/ml in Tris saline, 0.02% sodium azide, pH7.3 with 0.5% bovine serum albumin. Aliquot and store at -20°C. Minimize freezing and thawing.

Immunogen

Peptide with sequence C-QLESVDPKNYPKK, from the internal region of the protein sequence according to NP_000567.1.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Goat Anti-IL-1 beta Antibody (internal region) is for research use only and not for use in diagnostic or therapeutic procedures.

Goat Anti-IL-1 beta Antibody (internal region) - Protein Information

Name IL1B ([HGNC:5992](#))

Synonyms IL1F2

Function

Potent pro-inflammatory cytokine (PubMed:10653850, PubMed:12794819, PubMed:28331908, PubMed:3920526). 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 (PubMed:3920526). 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). Involved in transduction of inflammation downstream of pyroptosis: its mature form is specifically released in the extracellular milieu by passing through the gasdermin-D (GSDMD) pore (PubMed:33377178, PubMed:33883744). Acts as a sensor of *S.pyogenes* infection in skin: cleaved and activated by pyogenes SpeB protease, leading to an inflammatory response that prevents bacterial growth during invasive skin infection (PubMed:28331908).

Cellular Location

Cytoplasm, cytosol. Secreted. Lysosome Secreted, extracellular exosome {ECO:0000250|UniProtKB:P10749} Note=The precursor is cytosolic (PubMed:15192144). In response to inflammasome-activating signals, such as ATP for NLRP3 inflammasome or bacterial flagellin for NLRC4 inflammasome, cleaved and secreted (PubMed:24201029, PubMed:33377178, PubMed:33883744). Mature form is secreted and released in the extracellular milieu by passing through the gasdermin-D (GSDMD) pore (PubMed:33883744). In contrast, the precursor form is not released, due to the presence of an acidic region that is proteolytically removed by CASP1 during maturation (PubMed:33883744). The secretion is dependent on protein unfolding and facilitated by the cargo receptor TMED10 (PubMed:32272059)

Tissue Location

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

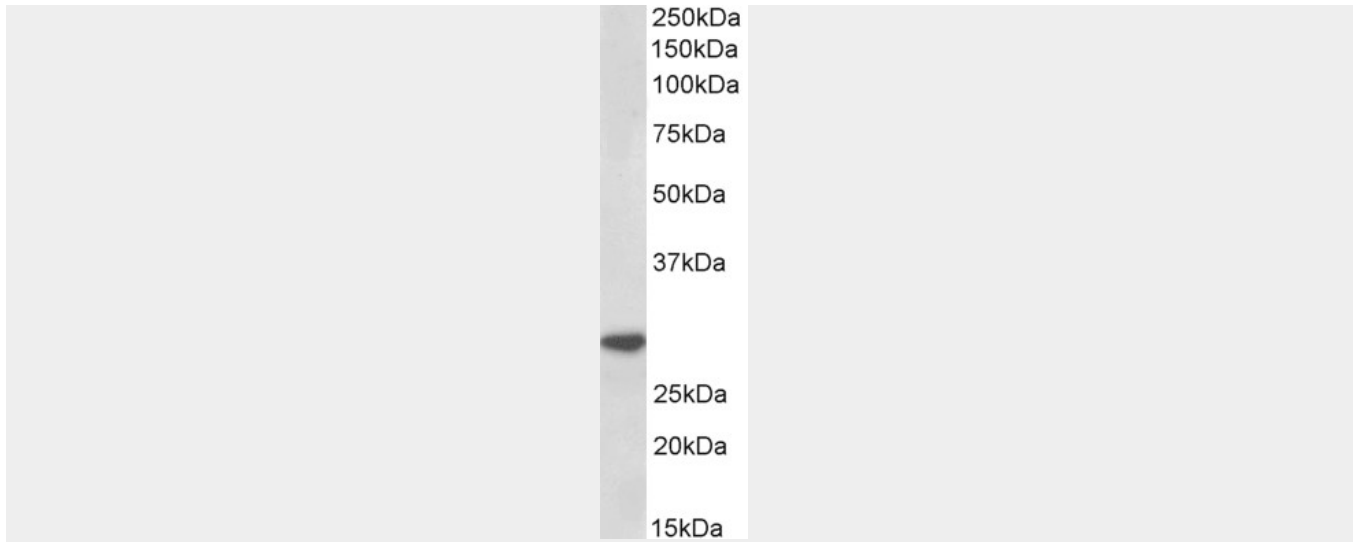
Goat Anti-IL-1 beta Antibody (internal region) - Protocols

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](#)

Goat Anti-IL-1 beta Antibody (internal region) - Images





AF4170a (1 $\mu\text{g/ml}$) staining of Human Peripheral Blood Lymphocytes lysate (35 μg protein in RIPA buffer). Primary incubation was 1 hour. Detected by chemiluminescence.

Goat Anti-IL-1 beta Antibody (internal region) - References

NLRC4 inflammasome-mediated production of IL-1 β modulates mucosal immunity in the lung against gram-negative bacterial infection. Cai S, Batra S, Wakamatsu N, Pacher P, Jeyaseelan S. *Journal of immunology* (Baltimore, Md. : 1950) 2012 Jun 188 (11): 5623-35.