

Product datasheet

Recombinant Human IL1 beta protein ab53666

1 Image

Overview

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<b>Product name</b>	Recombinant Human IL1 beta protein
<b>Protein length</b>	Full length protein

Description

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<b>Nature</b>	Recombinant
<b>Source</b>	Escherichia coli

Amino Acid Sequence

<b>Species</b>	Human
<b>Amino acids</b>	117 to 269

Specifications

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Our [Abpromise guarantee](#) covers the use of **ab53666** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

<b>Applications</b>	SDS-PAGE
<b>Purity</b>	> 95 % SDS-PAGE.
<b>Form</b>	Liquid

Preparation and Storage

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<b>Stability and Storage</b>	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles. Preservative: None Constituents: 50% Glycerol, 20mM Tris, pH 8
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General Info

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<b>Function</b>	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.
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Promotes Th17 differentiation of T-cells.

**Tissue specificity**

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

**Sequence similarities**

Belongs to the IL-1 family.

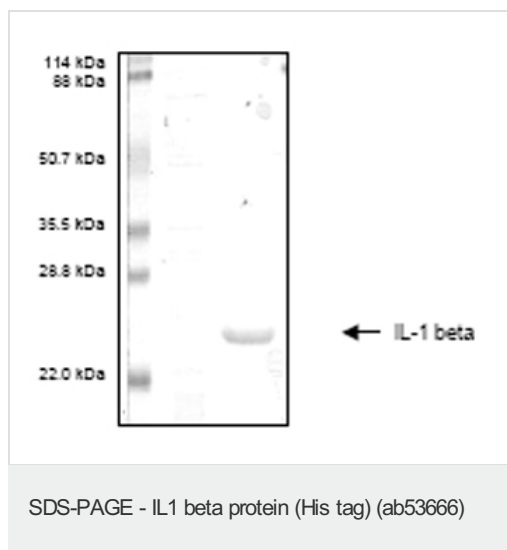
**Post-translational modifications**

Activation of the IL1B precursor involves a CASP1-catalyzed proteolytic cleavage. Processing and secretion are temporarily associated.

**Cellular localization**

Cytoplasm, cytosol. Lysosome. Secreted, exosome. Cytoplasmic vesicle, autophagosome. Secreted. 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). These mechanisms may not be mutually exclusive.

**Images**



ab53666 on SDS-PAGE

**Please note:** All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE"

## **Our Abpromise to you: Quality guaranteed and expert technical support**

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- Response to your inquiry within 24 hours
  
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