Human IL-1 beta ELISA Kit ab100562

Overview

**Product name**
Human IL-1 beta ELISA Kit

**Detection method**
Colorimetric

**Sample type**
Cell culture supernatant, Plasma

**Assay type**
Sandwich (quantitative)

**Sensitivity**
< 0.3 pg/ml

**Range**
0.48 pg/ml - 100 pg/ml

**Recovery**
99%

<table>
<thead>
<tr>
<th>Sample type</th>
<th>Average %</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell culture supernatant</td>
<td>100.43</td>
<td>89% - 110%</td>
</tr>
<tr>
<td>Plasma</td>
<td>99.66</td>
<td>90% - 107%</td>
</tr>
</tbody>
</table>

**Assay duration**
Multiple steps standard assay

**Species reactivity**
Reacts with: Human

**Product overview**

Human IL-1 beta ELISA (Enzyme-Linked Immunosorbent Assay) kit is an in vitro enzyme-linked immunosorbent assay for the quantitative measurement of human IL-1 beta in plasma and cell culture supernatants. (Human IL-1 beta concentration is quite low in normal plasma, it may not be detected in this assay).

This assay employs an antibody specific for human IL-1 beta coated on a 96-well plate. Standards and samples are pipetted into the wells and IL-1 beta present in a sample is bound to the wells by the immobilized antibody. The wells are washed and biotinylated anti-Human IL-1 beta antibody is added. After washing away unbound biotinylated antibody, HRP-conjugated streptavidin is pipetted to the wells. The wells are again washed, a TMB substrate solution is added to the wells and color develops in proportion to the amount of IL-1 beta bound. The Stop Solution changes the color from blue to yellow, and the intensity of the color is measured at 450 nm.

We have not been able to detect the endogenous human IL-1 beta in normal serum with ab100562, only in serum spiked with human IL-1 beta.
Optimization may be required with urine samples.

Platform

Microplate

Properties

Storage instructions

Store at -20°C. Please refer to protocols.

<table>
<thead>
<tr>
<th>Components</th>
<th>1 x 96 tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>20X Wash Buffer Concentrate</td>
<td>1 x 25ml</td>
</tr>
<tr>
<td>300X HRP-Streptavidin Concentrate</td>
<td>1 x 200µl</td>
</tr>
<tr>
<td>5X Assay Diluent B</td>
<td>1 x 15ml</td>
</tr>
<tr>
<td>Assay Diluent A</td>
<td>1 x 30ml</td>
</tr>
<tr>
<td>Biotinylated anti-Human IL-1 beta</td>
<td>2 vials</td>
</tr>
<tr>
<td>IL-1 beta Microplate (12 x 8 wells)</td>
<td>1 unit</td>
</tr>
<tr>
<td>Recombinant Human IL-1 beta Standards (lyophilized)</td>
<td>2 vials</td>
</tr>
<tr>
<td>Stop Solution</td>
<td>1 x 8ml</td>
</tr>
<tr>
<td>TMB One-Step Substrate Reagent</td>
<td>1 x 12ml</td>
</tr>
</tbody>
</table>

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.

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

Hu IL-1 beta measured in biological fluids showing quantity (pg) per mL of tested sample (U937: stimulated 24h with 10ng/mL PMA and 6h with LPS 1microgram/mL – THP-1: stimulated 24h with 10ng/mL PMA)

Representative standard curve using ab100562

Typical standard curve

Representative standard curve using ab100562

Typical standard curve
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