Recombinant Human Interleukin-1 beta (IL-1β), Animal Component-Free

Cat. No. :	H004E
Alternative Names:	IL1b; IL-1β; IL1β; IL-1B; IL1B; IL1F2; IL-1 beta; IL-1b; Interleukin-1 beta; Interleukin 1 beta; Catabolin
Species:	Human
Accession No.:	P01584
Expression System:	E. coli
Protein Sequence:	Ala117-Ser269
Theoretical MW:	17.38 kDa
Theoretical pI:	5.91
Tag:	Tag-Free.
Formulation buffer:	PBS, 5% Trehalose and 0.01% Tween 80, pH7.4.
Appearance:	Lyophilized Powder.
Purity:	≥95% as determined by SDS-PAGE.
Bioactivity:	The ED_{50} was ≤10 pg/mL, as measured by its ability to induce NF-κB reporter gene activity in MCF-7 cells.
Endotoxin Level:	≤0.1 EU/μg, as determined by the LAL assay.
Application:	Cell Culture; Activity Assays.

Preparation & Storage

Reconstitution: Reconstitute with sterile double-distilled water (ddH_2O).

⚠ Centrifuge the vial briefly before opening to ensure full recovery of the solution. Avoid vortexing and minimize vigorous pipetting to maintain protein stability.

❄ Immediately aliquot the reconstituted protein solution and store under recommended conditions. Avoid repeated freeze-thaw cycles.

Shipping: Shipped on dry ice. Short-term transit on cold packs (2-8℃) is acceptable.

Storage: **Use a manual defrost freezer and avoid repeated freeze-thaw cycles.**

- 12 months from date of receipt, -20 to -80℃ as supplied.
- 2-7 days at 2 to 8℃ under sterile conditions after reconstitution.
- 3-6 months at -20 to -80℃ under sterile conditions after reconstitution.

Protein Description

Background: Interleukin-1 beta (IL-1β) is a prototypical pro-inflammatory cytokine encoded by the IL1B gene in humans and is a central mediator of innate immunity, inflammation, and host defense. Unlike most secreted proteins, IL-1β lacks a conventional signal peptide and is synthesized as an inactive 31-kDa precursor (pro-IL-1 β). Its biological activity requires proteolytic cleavage by caspase-1 – activated within the NLRP3 inflammasome in response to pathogen-associated or damage-associated molecular patterns (PAMPs/DAMPs)–to generate the mature, secreted 17-kDa form.

IL-1β exerts its effects by binding to the IL-1 receptor type I (IL-1RI), which recruits the IL-1 receptor accessory protein (IL-1RAcP) to form a signaling-competent complex. This triggers downstream activation of NF-κB, MAPK (e.g., p38, JNK), and AP-1 pathways, leading to the expression of inflammatory mediators such as IL-6, IL-8, TNF-α, COX-2, and adhesion molecules. Due to its potent bioactivity, IL-1β signaling is tightly regulated at multiple levels–including transcriptional control, inflammasome activation, and extracellular inhibition by the natural antagonist IL-1Ra (interleukin-1 receptor antagonist).

IL-1β plays critical roles in both physiological and pathological contexts:

- It is essential for host defense against infections but also drives chronic inflammation in diseases such as rheumatoid arthritis, gout, atherosclerosis, and type 2 diabetes.
- In Alzheimer's disease, IL-1β is upregulated in activated microglia surrounding amyloid-β plaques and contributes to neuroinflammation and neuronal dysfunction.
- In cancer, IL-1β is overexpressed in multiple solid tumors (e.g., melanoma, lung, breast, colorectal, and head and neck cancers), where it promotes tumor growth, angiogenesis, metastasis, and immunosuppression. Elevated IL-1β correlates with poor prognosis and cancer-related cachexia.
- It also contributes to intervertebral disc degeneration and pulmonary fibrosis in experimental models.

Given its central role in inflammation and disease, IL-1β is a well-validated therapeutic target. Clinically approved agents such as anakinra (recombinant IL-1Ra), canakinumab (anti-IL-1β monoclonal antibody), and rilonacept (IL-1 trap) are used to treat autoinflammatory syndromes and are being investigated in cardiovascular disease and oncology.

References:

1. Dinarello, C. A. (2011). Immunological and inflammatory functions of the interleukin-1 family. Annual Review of Immunology, 29, 707-742.
2. Latz, E., Xiao, T. S., & Stutz, A. (2013). Activation and regulation of the inflammasomes. Nature Reviews Immunology, 13(6), 397-411.
3. Rider, P., et al. (2011). IL-1α and IL-1β are endogenous ligands for the NLRP3 inflammasome. European Journal of Immunology, 41(12), 3471-3479.
4. Heneka, M. T., et al. (2018). Neuroinflammation in Alzheimer's disease. The Lancet Neurology, 14(4), 388-405. https://doi.org/10.1016/S1474-4422(15)70016-5
5. Voronov, E., et al. (2014). IL-1 in chronic inflammation: The link between obesity and cancer? Cytokine & Growth Factor Reviews, 25(6), 675-687.
6. Marchetti, C., et al. (2018). The IL-1β pathway in inflammation-induced skeletal muscle wasting. Journal of Cachexia, Sarcopenia and Muscle, 9(4), 680-693.
7. Zhang, Y., et al. (2020). IL-1 β promotes intervertebral disc degeneration by inducing inflammatory responses in nucleus pulposus cells. Frontiers in Immunology, 11, 589654.

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