Recombinant Human Interleukin-1 alpha (IL-1α), Animal Component-Free

Cat. No. :	H003E
Alternative Names:	IL1a; IL1A; IL-1A; IL-1a； IL-1 alpha; IL-1α; Interleukin-1 alpha; Interleukin 1 alpha; IL1F1; Hematopoietin-1
Species:	Human
Accession No.:	P01583
Expression System:	E. coli
Protein Sequence:	Ser113-Ala271
Theoretical MW:	18.05 kDa
Theoretical pI:	5.30
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} for NF-κB reporter activation was determined to be ≤ 0.05 ng/mL using a reporter gene assay.
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 alpha (IL-1α) is a pleiotropic pro-inflammatory cytokine encoded by the IL1A gene and is a core member of the IL-1 cytokine family. Unlike conventional secreted cytokines, IL-1α functions as an alarmin: it can be passively released from damaged or stressed cells to trigger inflammation, and—uniquely among cytokines—it also exists in biologically active precursor form (pro-IL-1α) within the nucleus, cytoplasm, and plasma membrane of viable cells, where it modulates transcriptional regulation and cellular homeostasis.

IL-1α is primarily produced by activated monocytes, macrophages, endothelial cells, and epithelial cells. Upon binding to the IL-1 receptor type I (IL-1RI) and recruiting the co-receptor IL-1RAcP, it activates MyD88-dependent signaling pathways (e.g., NF-κB and MAPK), leading to the expression of downstream inflammatory mediators such as IL-6, TNF-α, and COX-2. This drives innate immune responses and bridges innate and adaptive immunity.

IL-1α plays critical roles in multiple pathophysiological contexts:

- In endometriosis, IL-1α derived from M2 macrophages has been identified as the key effector molecule at the 2q14.1 genetic risk locus, promoting lesion vascularization and pain.
- In breast cancer, host-derived IL-1α drives immunosuppression by reprogramming tumor-associated myeloid cells (e.g., $CX3CR1^+$ macrophages) via a PGE_2-dependent axis, thereby impairing $CD8^+$ T cell function.
- In inflammaging and the lung tumor microenvironment, elevated IL-1α contributes to chronic inflammation and tumor progression.
- IL-1α is also implicated in autoinflammatory disorders, atherosclerosis, and tissue repair.

Due to its dual localization (intracellular and extracellular) and multifaceted biological activities, IL-1α represents a promising therapeutic target in inflammatory and oncologic diseases. Clinical strategies targeting the IL-1 pathway—including the IL-1 receptor antagonist anakinra and the decoy receptor rilonacept—are already in use, while selective anti-IL-1α antibodies (e.g., bermekimab/MABp1) are under clinical investigation for cancer and chronic inflammatory conditions.

References:

1. Chiu, J. W., et al. (2021). The Multifaceted Roles of Interleukin-1 Alpha in Immunity and Disease. Cells, 10(10), 2796.
2. 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.
3. Advanced Science (2025). M2 Macrophages Are Major Mediators of Germline Risk of Endometriosis and Explain Pleiotropy With Comorbid Traits. Advanced Science, 14(1).
4. npj Breast Cancer (2026). Host-derived interleukin-1α drives tumor immunosuppression by reprogramming tumor-associated myeloid cells. npj Breast Cancer.
5. Dinarello, C. A. (2011). Immunological and Inflammatory Functions of the Interleukin-1 Family. Annual Review of Immunology, 29, 707–742.
6. Garlanda, C., et al. (2013). Non-redundant properties of IL-1α and IL-1β during innate and adaptive immunity. European Journal of Immunology, 43(6), 1441–1446.

Product Disclaimer

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