MIP-1 alpha/CCL3 Protein, Human

Cat. No.:	HY-P7256
Synonyms:	rHuMIP-1α/CCL3; C-C motif chemokine 3; MIP1A; SCYA3
Species:	Human
Source:	E. coli
Accession:	P10147 (A23-A92)
Gene ID:	6348
Molecular Weight:	Approximately 7.8 kDa

PROPERTIES

AA Sequence	A S L A A D T P T A C C F S Y T S R Q I P Q N F I A D Y F E T S S Q C S K P G V I F L T K R S R Q V C A D P S E E W V Q K Y V S D L E L S A
Appearance	Lyophilized powder
Formulation	Lyophilized after extensive dialysis against 20 mM PB, pH 7.4, 150 mM NaCl.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	CCL3 also known as macrophage inflammatory protein 1-a, is a member of the CC subfamily. It's known that CCL3 is produced by monocytes/macrophages, lymphocytes, neutrophils as well as immune cells such as basophils, mast cells, fibroblasts, and dendritic cells. Meanwhile, CCL3 exerts various biological effects by binding to its three cell surface receptors, including CCR1, CCR3, and CCR5. MIP-1a induces a variety of pro-inflammatory activities such as leukocyte chemotaxis, and promotes the entry of T cells into the inflammatory tissue region from blood circulation. Chemotactic CD4+ cells, CD8+ cells, natural killer cells, and dendritic cells bind to the corresponding receptors and coordinate the occurrence of immune reactions in the immune response site by migrating through vascular endothelial cells. In addition, MIP-1a is considered as a key inflammatory mediator in granuloma, asthma, T1D as well as other autoimmune diseases[1].

REFERENCES

[1]. Zhang G, et al. CCL3 participates in the development of rheumatoid arthritis by activating AKT. Eur Rev Med Pharmacol Sci. 2018 Oct;22(20):6625-6632.

[2]. Fabrizio Montecucco, et al. Tumor necrosis factor-alpha (TNF-alpha) induces integrin CD11b/CD18 (Mac-1) up-regulation and migration to the CC chemokine CCL3 (MIP-1alpha) on human neutrophils through defined signalling pathways. Cell Signal. 2008 Mar;20(3):557-68.

[3]. Zhang G, et al. CCL3 participates in the development of rheumatoid arthritis by activating AKT. Eur Rev Med Pharmacol Sci. 2018 Oct;22(20):6625-6632.

Caution: Product has not been fully validated for medical applications. For research use only.

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