Recombinant Human CXCL5/ENA-74

Catalog Number: 651-NA/CF

DESCRIPTION

Source	*E. coli*-derived Ala41-Asn114 Accession # P42830
N-terminal Sequence Analysis	Ala41
Predicted Molecular Mass	8.0 kDa

SPECIFICATIONS

Activity	Measured by its ability to induce myeloperoxidase release from cytochalasin B-treated human neutrophils. Schröder, J.M. *et al.* (1987) J. Immunol. **139**:3474. The ED_{50} for this effect is 0.5-1.5 µg/mL. Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2. The ED_{50} for this effect is 0.3-1.5 ng/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

CXCL5, also known as epithelial cell-derived neutrophil-activating peptide (ENA-78), is an 8 kDa proinflammatory member of the CXC subfamily of chemokines. Its Glu-Leu-Arg (ELR) motif confers angiogenic properties and distinguishes it from ELR-CXC chemokines which are angiostatic (1-3). Human CXCL5 shares 57% amino acid (aa) sequence identity with mouse and rat CXCL5. Among other human ELR^+ chemokines, it shares 77% aa sequence identity with CXCL6/GCP-2 and 35%-51% with CXCL1/GRO alpha, CXCL2/GRO beta, CXCL3/GRO gamma, CXCL7/NAP-2, and CXCL8/IL-8. Inflammatory stimulation up-regulates CXCL5 production in multiple hematopoietic cell types, fibroblasts, endothelial cells, and vascular smooth muscle cells. *In vivo*, CXCL5 is elevated at sites of inflammation and pulmonary fibrosis where it promotes neutrophil infiltration and activation as well as angiogenesis (3-6). Its up-regulation contributes to increased vascularization, tumor growth, and metastasis in many cancers (6-9). Full length CXCL5 (78 aa) is trimmed at the N-terminal end by cathepsin G and chymotrypsin to ENA-74 (74 aa) and ENA-70 (70 aa), with the shortened forms showing increased potency relative to full length CXCL5 (10, 11). CXCL5 exerts its effects primarily through interactions with CXCR2 (6, 12). It also binds DARC, a decoy chemokine receptor which can limit CXCR2-mediated responses (13, 14).

References:

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