Recombinant Human CXCL5/ENA-70

Catalog Number: 649-EN

DESCRIPTION

Source	*E. coli*-derived Arg45-Asn114 Accession # P42830
N-terminal Sequence Analysis	Arg45
Predicted Molecular Mass	7.7 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 0.1-0.3 µg/mL. Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2. The ED_{50} for this effect is 0.15-0.75 ng/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 25 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
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 upregulates 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 upregulation 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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Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449