Recombinant Human CXCL5/ENA-78
Catalog Number: 254-XB

**DESCRIPTION**

**Source**  
E. coli-derived  
Ala37-Asn114  
Accession # P42830

**N-terminal Sequence Analysis**  
Ala37

**Predicted Molecular Mass**  
8.4 kDa

**SPECIFICATIONS**

**SDS-PAGE**  
7 kDa, reducing conditions

**Activity**  
The ED$_{50}$ is 3-7 µg/mL.

**Endotoxin Level**  
<0.01 EU per 1 µg of the protein by the LAL method.

**Purity**  
>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

**Formulation**  
Lyophilized from a 0.2 µm filtered solution in PBS containing BSA as carrier protein. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution**  
Reconstitute at 100 µg/mL in 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 CXCL8/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-terminus 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 Duffy antigen receptor for chemokines (DARC), which can limit CXCR2-mediated responses (13, 14).

**References:**