

**DESCRIPTION**

**Source** *E. coli*-derived  
Ala23-Ser99 & Ser28-Ser99  
Accession # P10145

**N-terminal Sequence Analysis** Ala23 & Ser28

**Predicted Molecular Mass** 8.9 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<sub>50</sub> for this effect is 0.2-0.4 µg/mL.

Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2.  
The ED<sub>50</sub> for this effect is 1-5 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 PBS. 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**

Interleukin-8 (IL-8), also known as CXCL8, GCP-1, and NAP-1, is a widely expressed proinflammatory member of the CXC family of chemokines. Near its N-terminus, this 8-9 kDa chemokine contains an ELR motif which is important for its angiogenic properties (1). CXCL8 can associate into a homodimer or a heterodimer with CXCL4/PF4 (2), and it can also interact with matrix and cell surface glycosaminoglycans (3). Mature human CXCL8 shares 65%-69% amino acid (aa) sequence identity with canine, feline, and porcine CXCL8 (4). There is no CXCL8 gene counterpart in rodent. N-terminal truncation by multiple proteases generates a range of shorter forms, and an alternative splice form of human CXCL8 carries an eleven aa substitution at the C-terminus (5). The bioactivity of CXCL8 is regulated by these truncations, by CXCL8 citrullination at Arg5 (N-terminal to the ELR motif) (6), and by the decoy receptor DARC (7). CXCL8 effects are mediated through CXCR1/IL-8 RA, which is also used by CXCL6, and through CXCR2/IL-8 RB, which is used by multiple CXC chemokines (1). CXCR1 and CXCR2 associate into functional homodimers and heterodimers with each other (8). Through both CXCR1 and CXCR2, CXCL8 promotes neutrophil adhesion to the vascular endothelium and migration to sites of inflammation (9). It triggers the antimicrobial activation of neutrophils through CXCR1 (10). CXCL8 also binds to Serpin A1/alpha-1 Antitrypsin, and this prevents CXCL8 interaction with CXCR1 (11). CXCL8 is upregulated in atherosclerotic lesions and other cardiac pathologies where it exacerbates inflammatory tissue damage (12). In addition, it induces VEGF expression, vascular endothelial cell proliferation, angiogenesis, and tumor cell invasiveness (13-16).

**References:**

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