IL-8/CXCL8 Protein, Human (HEK293, His)

Cat. No.:	HY-P70569
Synonyms:	Interleukin-8; IL-8; C-X-C Motif Chemokine 8; CXCL8; Emoctakin; Granulocyte Chemotactic Protein 1; GCP-1; Monocyte-Derived Neutrophil Chemotactic Factor; MDNCF; Monocyte-Derived Neutrophil-Activating Peptide; MONAP; Neutrophil-Activating Protein 1; NAP-1
Species:	Human
Source:	HEK293
Accession:	P10145 (E21-S99)
Gene ID:	3576
Molecular Weight:	Approximately 14.0 kDa

PROPERTIES

AA Sequence	E G A V L P R S A K E L R C Q C I K T Y S K P F H P K F I K E L R V I E S G P H C A N T E I I V K L S D G R E L C L D P K E N W V Q R V V E K F L K R A E N S
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM PB, 150 mM NaCl, pH 7.4.
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	IL-8 (CXCL8) belongs to the ELR^+ CXC chemokines family. IL-8 is initially produced as a protein of 99 amino acids that undergoes cleavage to form active IL-8 isoforms, a 77 amino acid peptide in non-immune cells or a 72 amino acid peptide in monocytes and macrophages. The gene encoding IL-8 is located on chromosome 4q13-q21. Dimerisation of IL-8 forms the structural basis for receptor binding. IL-8 is expressed by various cells including monocytes, macrophages, leukocytes, endothelial cells, and epithelial cells[1][2][3]. Mature human IL-8/CXCL8 shares 75% amino acid sequence identity with canine IL-8/CXCL8. While, human IL-8 shares 94.95% aa sequence identity with Rhesus Macaque IL-8 protein. IL-8 is responsible for the recruitment and activation of neutrophils and granulocytes to the site of inflammation. IL-8 is almost undetectable in physiological states, but is rapidly induced by pro-inflammatory cytokines such as TNFα and IL-1β. The function of IL-8 mainly relies on its interaction with specific cell surface GPCR, CXCR1 and CXCR2. In addition, IL-8 is

reported to promote integrin β3 upregulation and the invasion of hepatocellular carcinoma cells through activation of the PI3K/Akt pathway. In odontogenic lesions, IL-8 has been proven to be highly expressed in ameloblastoma epithelial cells and irreversible pulpitis. In rheumatoid arthritis and other inflammatory joint diseases IL-8 could bring about the accumulation of neutrophils, which are considered a major source of cartilage-degrading enzymes. IL-8 stimulates the MAPK and tyrosine phosphorylation of cellular proteins. Tumour cells and fibroblasts communicate with each other, including autocrine and paracrine factors, including IL-8, resulting in the upregulation of MMP2 and MMP9 degradable extracellular matrix (ECM) components that trigger tumour invasion[1][2][3][4].

IL-8 is typically known to promote angiogenesis, but it also activates matrix metalloproteinase (MMP) that is involved in metastasisrelated tissue remodelling. IL-8 is induced in lipopolysaccharide (LPS)-stimulated monocytes and shown to induce neutrophil migration. IL-8 exerts multiple effects on biological activities of tumour cells including proliferation, invasion and migration. IL-8 also increases the expression of Akt in androgen-independent prostate cancer (AIPC) cell lines. IL-8 activates MAPK signalling via PI3K in neutrophils, and via transactivation of EGFR resulting in Ras-GTPase activation in ovarian and lung cancer cell lines. There is substantial amount of experimental data suggesting that IL-8 and receptors contribute to elimination of pathogens, but may also contribute significantly to disease-associated processes, including tissue injury, fibrosis, angiogenesis and tumorigenesis[3][5].

REFERENCES

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