GRO-alpha/CXCL1 Protein, Rat

Cat. No.:	HY-P7189
Synonyms:	rRtGRO-α/CXCL1; Growth-regulated alpha protein; C-X-C motif chemokine 1; CINC-1
Species:	Rat
Source:	E. coli
Accession:	P14095 (A25-K96)
Gene ID:	81503
Molecular Weight:	7-11 kDa

PROPERTIES

AA Sequence	A P V A N E L R C Q C L Q T V A G I H F K N I Q S L K V M P P G P H C T Q T E V I A T L K N G R E A C L D P E A P M V Q K I V Q K M L K G V P K
Biological Activity	1. Full biological activity determined by a chemotaxis bioassay using rat neutrophils is in a concentration range of 10-100 ng/mL. 2. Measured in a cell proliferation assay using human umbilical vein endothelial cells. The ED_{50} this effect is 16.43 ng/mL, corresponding to a specific activity is 6.0864×10^4 units/mg.
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	CXCL1, also known as GRO-α, is a polypeptide that is initially isolated from human melanoma cells. CXCL1 acts as a key chemoattractant for neutrophils by binding specifically to its corresponding G-protein-coupled receptor CXCR2. CXCL1 modulates angiogenesis, tumorigenesis, and wound healing. In general, CXCL1 levels are extremely low under normal physiological conditions and greatly increased during inflammatory conditions[2][3]. The amino acid sequence of human CXCL1 protein has low homology between mouse and rat CXCL1 protein.

After translation, the synthesized CXCL1 precursor is 107aa long. A signal peptide is removed from its N-terminus, which shortens the precursor to 73aa. Two other amino acids can also be removed from the C-terminus. In addition, two disulfide bridges are formed from all four cysteine residues in CXCL1. The disulfide bridges give the appropriate structure to CXCL1, which determines the properties of this chemokine. After secretion, CXCL1 undergoes further proteolytic processing, which regulates the activity of this chemokine. From the N-terminus, three, four or five amino acids are removed, which produce CXCL1(4-73), CXCL1(5-73), and CXCL1(6-73), respectively. This increases CXCL1 activity 30 times, as measured by its ability to induce the chemotaxis of treated cells. To date, three CXCL1 receptors have been discovered-CXCR1, CXCR2 and atypical chemokine receptor 1 (ACKR1). Through NF-κB activation, CXCL1 expression is increased by cytokines such as IL-1β, TNF-α and IL-17. CXCL1 can associate into bioactive dimers and primarily signals through CXCR2/IL-8 RB[1].

After CXCL1 expression is induced by carcinogens, it participates in inflammatory responses by recruiting neutrophils. This leads to chronic inflammation. In addition to increasing proliferation, CXCL1 also induces cancer cell migration, particularly EMT. Produced by lymphatic endothelial cells (LECs), CXCL1 enables tumor cell migration into the lymphatic vessels during lymphangiogenesis, leading to lymph node metastasis. CXCL1 is a chemotactic factor for neutrophils. Additionally, it causes the mobilization of these cells from the bone marrow. CXCL1 can also induce recruitment of regulatory T cells (Treg) and MSCs into the tumor niche. Another no-less-important property of CXCL1 is its ability to induce angiogenesis[1].

REFERENCES

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