SDF-1 alpha/CXCL12 Protein, Mouse (Tag free)

Cat. No.:	HY-P70518
Synonyms:	Cxcl12; Stromal cell-derived factor 1; SDF-1; 12-O-tetradecanoylphorbol 13-acetate repressed protein 1; TPAR1; C-X-C motif chemokine 12; Pre-B cell growth-stimulating factor; PBSF; Thymic lymphoma cell-stimulating factor; TLSF; Sdf1
Species:	Mouse
Source:	E. coli
Accession:	P40224 (K22-K89)
Gene ID:	20315
Molecular Weight:	Approximately 10.0 kDa

PROPERTIES

AA Sequence	K P V S L S Y R C P C R F F E S H I A R A N V K H L K I L N T P N C A L Q I V A R L K N N N R Q V C I D P K L K W I Q E Y L E K A L N K
Biological Activity	Measured by its ability to chemoattract IL-2-activated human T cells. The ED_{50} for this effect is approximately 25.04 ng/mL, corresponding to a specific activity is 3.994×10^4 U/mg.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 25 mM Tris-HCl, 150 mM NaCl, pH 8.5.
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	CXCL12 (SDF-1), is a small protein that belongs to the chemokine family, whose members have a crucial role in directing cell migration. It is ubiquitously expressed in many tissues and cell types. CXCL12 acts through two receptors, CXCR4 and CXCR7. While the former is a classic G protein-coupled transmembrane chemokine receptor, the latter primarily function as a scavenger of CXCL12. CXCL12 is a chemoattractant for T-lymphocytes and monocytes, but not neutrophils[1][2][3]. CXCL12 gene in human is located on chromosome 10 (10q11.21) and recognizes seven isoforms deriving from alternative gene splicing (α, β, γ, δ, ε, θ) with α and β being the most studied and three (CXCL12α to γ) in mice. CXCL12 is described classically as a homing chemokine as it exhibits chemoattraction of tumoral cells toward the target tissues. CXCL12,

although being homeostatic in classification, also takes inflammatory activities. CXCL12 binds to glycosaminoglycans (GAGs) exposed on the surface of endothelial cells through a cluster of basic residues-the BBXB motif (B for basic amino acid and X any amino acid) generating its chemotactic gradients and promoting leukocyte/cancer cell migration. CXCL12/CXCR4 axis is involved in tumor progression, angiogenesis, metastasis, and survival. CXCR7 binds with high-affinity CXCL12 and with lower-affinity CXCL11. Although CXCR7 acts as a CXCL12 scavenger through ligand internalization and degradation, it transduces the signal mainly through β-arrestin with a pivotal role in endothelial and neural cells. CXCL12 is constitutively expressed in several organs including lung, liver, skeletal muscle, brain, kidney, heart, skin, and bone marrow. CXCL12 secretion is also associated with tissue damage such as heart infarct, limb ischemia, toxic liver damage, excessive bleeding, total body irradiation, and after tissue damage related to chemotherapy. CXCR4 is expressed by endothelial cells and pericytes of hypoxic, injured, or pathological tissues, including injured carotid arteries and atherosclerotic plaques[1][2]. CXCL12 has been considered as a standard pro-inflammatory molecule for a long time, as it attracts leukocytes to inflammatory sites and contributes to their activation. CXCL12 is very importantfor vascularization, hematopoiesis and neuronal development, but also for HIV-infection and cancer metastasis. CXCL12 plays a classic chemokine role in immune response by attracting activated, CXCR4+ T cells and monocytes to the sites of inflammation. However, CXCL12 plays an anti-inflammatory role in neuroinflammatory demyelinating disorders ofthe CNS, i.e. inmultiple sclerosis (MS) and its animal model experimental autoimmune encephalomyelitis (EAE). In addition, CXCL12 targeting affects tumor primary growth, mesenchymal transition, and migration but also shapes the tumor microenvironment (TME) toward immunoresponsive TME[1][2][3].

REFERENCES

[1]. Beverly A Teicher, et al. CXCL12 (SDF-1)/CXCR4 pathway in cancer. Clin Cancer Res. 2010 Jun 1;16(11):2927-31.

[2]. Luigi Portella, et al. CXCL12 Signaling in the Tumor Microenvironment. Adv Exp Med Biol. 2021;1302:51-70.

[3]. Gordana Timotijević, et al. CXCL12: role in neuroinflammation. Int J Biochem Cell Biol. 2012 Jun;44(6):838-41.

[4]. Kevin S Carbajal, et al. Migration of engrafted neural stem cells is mediated by CXCL12 signaling through CXCR4 in a viral model of multiple sclerosis. Proc Natl Acad Sci U S A. 2010 Jun 15;107(24):11068-73.

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