MIG/CXCL9 Protein, Human

Cat. No.:	HY-P7253
Synonyms:	rHuMIG/CXCL9; C-X-C motif chemokine 9; SCYB9
Species:	Human
Source:	E. coli
Accession:	Q07325 (T23-T125)
Gene ID:	4283
Molecular Weight:	Approximately 11.7 kDa

PROPERTIES

AA Sequence	T P V V R K G R C S C I S T N Q G T I H L Q S L K D L K Q F A P S P S C E K I E I I A T L K N G V Q T C L N P D S A D V K E L I K K W E K Q V S Q K K K Q K N G K K H Q K K K V L K V R K S Q R S R Q K K T T
Biological Activity	Full biological activity determined by a chemotaxis bioassay using human peripheral blood T-lymphocytes is in a concentration range of 10-100 ng/mL.
Appearance	Lyophilized powder.
Formulation	Lyophilized after extensive dialysis against 20 mM PB, pH 7.4, 50 mM NaCl.
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	CXCL9 is a member of the CXC family and has an important role in the chemotaxis of immune cells. It is secreted by various cell types including immune cells (T lymphocytes, NK cells, dendritic cells, macrophages, eosinophils, etc.), and non-immune cells (hepatic stellate cells, preadipocytes, thyrocytes, endothelial cell, tumor cells, and fibroblasts, etc)[1]. The amino acid sequence of human CXCL9 protein has low homology between mouse and rat CXCL9 protein. CXCL9 is one of the ligands of chemokine receptor CXCR3 that mediates the infiltration of lymphocytes to focal sites and suppresses tumor growth. CXCL9 attracts CXCR3- (CXCR3-A and CXCR3-B) T lymphocytes, is involved in the pathogenesis of

a variety of physiologic diseases during their initiation and their maintenance. The transcriptional regulation of CXCL9 is a multistep process involving many transcription factors, of which STAT1 and NF-κB are two most well-characterized members. Both the gene mutation of STAT1 and the blocking of the JA/STAT1 pathway can reduce CXCL9 expression induced by IFN-γ. Moreover, CXCL9 expression can be suppressed by reducing the levels of components of the STAT1-IRF-1 transcriptional activation pathway by Porphyromonas gingivalis that leads to the immune function decline. Lipopolysaccharide (LPS) and D-galactosamine could induce the phosphorylation of STAT1 and enhance the transcription of CXCL9 leading to the enhancement of liver inflammation, and even liver apoptosis and injury[1][2][3].
CXCL9 could promote cancer metastasis via enhanced migration and invasion of tumor cells, and breaking of the endothelial cells monolayer. However, as a tumor suppressor, it mainly recruited tumor-infiltrating $CD8^+$ T cells and NK cells, and inhibited tumor angiogenesis. In Addition, IL-12 and Th1-derived IFN-γ exerted antitumor effects through the inhibitory effects of endogenous CXCL9 on tumor vasculature in human Burkitt's lymphoma. In cutaneous T-cell lymphoma, expression of CXCL9 was found at early stage but low at advanced stage. CXCL9 is also associated with human hepatic fibrosis and anti-fibrosis in mice. Furthermore, CXCL9 is highly expressed in atherosclerotic plaques of coronary arteries and specifically recruits CXCR3-bearing Th1 cells that increase the risk of plaque progression and the occurrences of myocardial infarction[1][2][3][4].

REFERENCES

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