Datasheet

Cat. No. PM2038

Recombinant Mouse CCL5, Tag Free

Information

Accession #	P13500
Alternate Names	CCL5; chemokine (C-C motif) ligand 5; D17S136Enormally T-expressed, and presumably secreted; EoCP; Eosinophil chemotactic cytokine; RANTES; SISd; SIS-delta; small inducible cytokine A5 (RANTES); small inducible cytokine su bfamily A (Cys-Cys), member 5; Small-inducible cytokine A5.
Source	Human embryonic kidney cell, HEK293-derived human IL1-beta protein
Protein sequence	Ser24-Ser91.
M.Wt	7.9 kDa
Appearance	Solution protein
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. 3 years from date of receipt, -20 to -70°C as supplied.
Concentration	0. 2 mg/mL
Formulation	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.

Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	<0.010 EU per 1 ug of the protein by the LAL method.

Description

CCL5, also known as RANTES (Regulated upon Activation, Normal T cell Expressed and presumably Secreted), is an 8 kDa beta -chemokine that plays a primary role in the inflammatory immune response by means of its ability to attract and activate leukocytes [1 - 3]. Human and mouse RANTES exhibit cross-species activity on human and mouse cells [4]. Mature mouse CCL5 shares 100% aa sequence identity with rat CCL5 and 75% - 88% with canine, cotton rat, feline, and human CCL5 [5]. CCL5 is secreted by many cell types at inflammatory sites, and it exerts a wide range of activities through the receptors CCR1, CCR3, CCR4, and CCR5 [6, 7]. Inflammatory responses can be impaired by the sequestration of CCL5 by the cytomegalovirus

protein US28 [8]. In humans, CCR5 binding to CCL5 inhibits the infectivity of R5 (M-tropic) but not X4 (T-tropic) strains of HIV-1 [9]. The two N-terminal residues of CCL5 can be removed by CD26/DPPIV, generating a protein that functions as a chemotaxis inhibitor and more effectively blocks M-tropic HIV-1 infection of monocytes [10]. Oligomerization of CCL5 on glycosaminoglycans is required for CCR1-mediated leukocyte adhesion and activation as well as CCL5′ s interaction with the chemokine CXCL4/PF4 [11 - 13]. The deposition of CCL5 on activated vascular endothelial cells is crucial for monocyte adhesion to damaged vasculature, but CCL5 oligomerization is not required for the extravasation of adherent leukocytes [14 - 16]. CCL5 is upregulated in breast cancer and promotes tumor progression through the attraction of proinflammatory macrophages in addition to its actions on tumor cells, stromal cells, and the vasculature [17].

Reference

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