

Recombinant Human CCL13 Protein(Trx Tag)

Catalog Number: PDEH100512

Note: Centrifuge before opening to ensure complete recovery of vial contents.

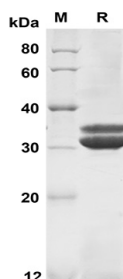
Description

Species	Human
Source	E.coli-derived Human CCL13 protein Gln24-Thr98, with an N-terminal Trx
Mol_Mass	28.3 kDa
Accession	Q99616
Bio-activity	Not validated for activity

Properties

Purity	> 80% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis

Data



SDS-PAGE analysis of Human CCL13 proteins, 2µg/lane of

Recombinant Human CCL13 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 31 kDa

Background

For Research Use Only

Tel:400-999-2100

Web:www.elabscience.cn

Email:techsupport@elabscience.cn

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Monocyte Chemoattractant Proteins 4 (MCP-4/CCL13) is a member of a distinct, structurally-related subclass of CC chemokines mainly involved in recruitment of eosinophils to inflammatory sites. CCL13/MCP-4, is a CC family chemokine that is chemoattractant for eosinophils, basophils, monocytes, macrophages, immature dendritic cells, and T cells, and is capable of inducing crucial immuno-modulatory responses through its effects on epithelial, muscular and endothelial cells. Similar to other CC chemokines, CCL13 binds to several chemokine receptors (CCR1, CCR2 and CCR3), allowing it to elicit different effects on its target cells. A number of studies have shown that CCL13 is involved in many chronic inflammatory diseases, in which it functions as a pivotal molecule involved in the selective recruitment of cell lineages to the inflamed tissues and their subsequent activation. MCP-4/CCL13 is secreted from chondrocytes and activates the proliferation of rheumatoid synovial cells, thereby leading to joint destruction in RA. The interferon-gamma in combination with interleukin-1beta/tumor necrosis factor-alpha activates the production of MCP-4/CCL13 from chondrocytes in RA joints, and that secreted MCP-4/CCL13 enhances fibroblast-like synoviocyte proliferation by activating the extracellular signal-regulated kinase mitogen-activated protein kinase cascade. CCL13 may have some role in the pathogenesis of systemic sclerosis (SSc).