Recombinant Mouse CX3CL1/Fractalkine N-truncated, aa 29-102

Catalog Number: 3215-MF

DESCRIPTION

Source	*E. coli*-derived Met29-Lys102 Accession # O35188
N-terminal Sequence Analysis	Met29
Predicted Molecular Mass	8.4 kDa

SPECIFICATIONS

Activity	Measured by its ability to antagonize Recombinant Mouse CX3CL1/Fractalkine aa 25-105 (Catalog # 571-MF) induced chemotaxis of BaF3 mouse pro-B cells transfected with mouse CX3CR1. Inoue, A. *et al.* (2005) Arthritis Rheum. **52**:1522. The ED_{50} for this effect is 0.25-1.5 µg/mL in the presence of 50 ng/mL of Recombinant Mouse CX3CL1/Fractalkine aa 25-105 (Catalog # 571-MF). **Optimal concentration depends on cell type as well as the application or research objective.**
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Fractalkine, designated CX3CL1 and also known as neurotactin, is the only member of the CX3C, or delta, chemokine subfamily (1-4). Unlike most other chemokines, CX3CL1 is a type I transmembrane (TM) adhesion protein (1). The mouse CX3CL1 cDNA encodes 395 amino acids (aa), including a signal sequence (aa 1-24), a chemokine domain (aa 25-100), a mucin stalk region (aa 101-336), a transmembrane segment (aa 337-357), and a cytoplasmic tail (aa 358-395). The chemokine domain contains binding and chemotactic determinants, while the mucin stalk appears to function only as a spacer (4, 5). Mouse CX3CL1 shares 85% and 78% aa sequence identity with rat and human CX3CL1, respectively, within the chemokine domain, but lower sequence identity within other domains. CX3CL1 is up-regulated by pro-inflammatory stimuli, especially IFN-γ and TNF-α, on cell types including macrophages, dendritic cells, endothelium, neurons, smooth muscle and epithelium lining the intestines and other tubules (1, 8, 9). The 40 kDa, 7-TM non-glycosylated G-protein coupled CX3CL1 receptor, CX3CR1, is expressed by cytotoxic effector cells and cytokine producers, including type I helper and cytotoxic T cells, γδ T cells, $CD16^+$ NK cells, monocytes and microglia (1, 2). The 95-100 kDa TM CX3CL1 can be inducibly cleaved near the TM segment by ADAM10 or ADAM17 to generate a 60-80 kDa soluble form (6, 7). TM CX3CL1 functions as an adhesion molecule, while both forms are chemoattractants for target cells expressing CX3CR1 (1, 2). During extravasation, membrane-bound CX3CL1 traps leukocytes, then is cleaved to allow diapedesis (6). In coronary artery disease, soluble CX3CL1 and $CD8^+$ T cell CX3CR1 are overexpressed and appear to contribute to pathogenesis (1, 10). In the brain, CX3CL1/CX3CR1 interaction protects against microglial neurotoxicity (11). CX3CL1 also contributes to wound healing by recruiting macrophages, and to bone resorption by recruiting and mediating adhesion of osteoclast precursors (12, 13).

References:

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