

DESCRIPTION

Source *E. coli*-derived
Ser24-Lys91
Accession # AAL16932

N-terminal Sequence Analysis Ser24

Predicted Molecular Mass 7.9 kDa

SPECIFICATIONS

Activity Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with mouse CCR5. The ED₅₀ for this effect is 20-100 ng/mL.

Endotoxin Level <0.10 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 with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 25 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

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

CCL5, also known as RANTES (Regulated upon Activation, Normal T cell Expressed and presumably Secreted), is an 8 kDa β-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 cotton rat CCL5 shares 88% aa sequence identity with mouse and rat CCL5 and 71%-77% with canine, feline, and human CCL5. 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 (5, 6). Inflammatory responses can be impaired by the sequestration of CCL5 by the cytomegalovirus protein US28 (7). In humans, CCR5 binding to CCL5 inhibits the infectivity of R5 (M-tropic) but not X4 (T-tropic) strains of HIV-1 (8). 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 (9). 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 (10-12). 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 (13-15). CCL5 is up-regulated 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 (16).

References:

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