

DESCRIPTION

Source *E. coli*-derived mouse CCL4/MIP-1 beta protein
Ala24-Asn92
Accession # Q5QNV9

N-terminal Sequence Analysis Ala24

Predicted Molecular Mass 7.8 kDa

SPECIFICATIONS

Activity Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CCR5. The ED₅₀ for this effect is 0.75-5 ng/mL.

Endotoxin Level <0.01 EU per 1 μ g 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 μ m filtered solution in Acetonitrile and TFA with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 μ g/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

CCL4, also known as macrophage inflammatory protein 1 beta (MIP-1 β), is a 12 kDa β chemokine that is secreted at sites of inflammation by activated leukocytes, lymphocytes, vascular endothelial cells, and pulmonary smooth muscle cells (1, 2). CCL4 attracts a variety of immune cells to sites of microbial infection as well as to other pathologic inflammation such as allergic asthma and ischemic myocardium (3 - 8). A CCL4 deficiency in mice promotes the development of autoantibodies, possibly as a result of compromised regulatory T cell recruitment (6). CCL4 is secreted from activated monocytes as a heterodimer with CCL3/MIP-1 α (9). The first two N-terminal amino acids can be cleaved from human CCL4 by CD26/DPPIV (10, 11). Both the full length and truncated forms exert biological activity through CCR5, and the truncated form additionally interacts with CCR1 and CCR2 (10). In humans, the ability of CCL4 to bind CCR5 inhibits the cellular entry of M-tropic HIV-1 which utilizes CCR5 as a coreceptor (2). Both forms of CCL4 block HIV-1 infection of T cells by inducing the downregulation of CCR5 (10). Mature mouse CCL4 shares 77% and 86% aa sequence identity with human and rat CCL4, respectively.

References:

1. Rot, A. and U.H. von Andrian (2004) *Annu. Rev. Immunol.* **22**:891.
2. Menten, P. *et al.* (2002) *Cytokine Growth Factor Rev.* **13**:455.
3. Sun, X. *et al.* (2006) *Infect. Immun.* **74**:5943.
4. Bisset, L.R. and Schmid-Grendelmeier, P. (2005) *Curr. Opin. Pulm. Med.* **11**:35.
5. Frangogiannis, N.G. (2004) *Inflamm. Res.* **53**:585.
6. Bystry, R.S. *et al.* (2001) *Nat. Immunol.* **2**:1126.
7. Oliveira, S.H.P. *et al.* (2002) *J. Leukoc. Biol.* **71**:1019.
8. Schall, T.J. *et al.* (1993) *J. Exp. Med.* **177**:1821.
9. Guan, E. *et al.* (2001) *J. Biol. Chem.* **276**:12404.
10. Guan, E. *et al.* (2002) *J. Biol. Chem.* **277**:32348.
11. Guan, E. *et al.* (2004) *J. Cell. Biochem.* **92**:53.