Recombinant Mouse CXCL2/MIP-2 Protein

Catalog Number: PKSM040996

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

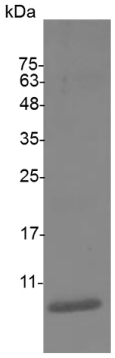
Description

Species	Mouse
Source	E.coli-derived Mouse CXCL2/MIP-2 protein Ala28-Asn100 , with an N-terminal His
Calculated MW	8.7 kDa
Observed MW	11 kDa
Accession	P10889
Bio-activity	Measure by its ability to chemoattract BaF3 cells transfected with human CXCR2.The ED_{50} for this effect is <0.5 ng/mL.

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	< 0.1 EU per μg 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 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



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Background

C-X-C motif chemokine 2 (CXCL2,MIP-2) belongs to the intercrine alpha (chemokine CxC) family. It was originally identified as a heparin-binding protein secreted from a murine macrophage cell line in response to endotoxin stimulation. The expression of mouse MIP-2 is stimulated by endotoxin. The mouse MIP-2 shares approximately 63% aa sequence identity with murine KC, another mouse alpha chemokine, which is induced by PDGF. It has been suggested that mouse KC and MIP-2 are the homologs of the human GROs and rat CINCs. Chemotactic for human polymorphonuclear leukocytes but does not induce chemokinesis or an oxidative burst. The expression of MIP-2 was found to be associated with neutrophil influx in pulmonary inflammation and glomerulonephritis, suggesting that MIP-2 may contribute to the pathogenesis of inflammatory diseases.