

Recombinant Mouse CXCL2/MIP-2 Protein(Sumo Tag)

Catalog Number: PDEM100172

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

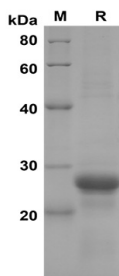
Description

Species	Mouse
Source	E.coli-derived Mouse CXCL2/MIP-2 protein Ala28-Asn100, with an N-terminal Sumo
Calculated MW	20.9 kDa
Observed MW	27 kDa
Accession	P10889
Bio-activity	Not validated for activity

Properties

Purity	> 90% 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 Mouse CXCL2/MIP-2 proteins, 2 µg/lane of Recombinant Mouse CXCL2/MIP-2 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 27 KD

Background

Chemokine (C-X-C motif) ligand 2 (CXCL2), also called macrophage inflammatory protein 2 (MIP-2), Growth-regulated protein beta (Gro-beta) and Gro oncogene-2 (Gro-2), is a small cytokine belonging to the CXC chemokine family. CXCL2/MIP-2 is selectively up-regulated in tolerance-conferring APCs and serves to recruit NKT cells to the splenic marginal zone, where they form clusters with APCs and T cells. In the absence of the high-affinity receptor for CXCL2/MIP-2 or in the presence of a blocking Ab to CXCL2/MIP-2, peripheral tolerance is prevented, and Ag-specific T regulatory cells are not generated. CXCL2/MIP-2 is selectively up-regulated in tolerance-conferring APCs and serves to recruit NKT cells to the splenic marginal zone, where they form clusters with APCs and T cells. In the absence of the high-affinity receptor for MIP-2 (as in CXCR2-deficient mice) or in the presence of a blocking Ab to MIP-2, peripheral tolerance is prevented, and Ag-specific T regulatory cells are not generated. Understanding the regulation of lymphocyte traffic during tolerance induction may lead to novel therapies for autoimmunity, graft acceptance, and tumor rejection. Several studies have implicated the CXCL2 chemokine as a mediator in the development of sepsis. CXCL2/MIP-2 also plays a major role in mediating the neutrophilic inflammatory response of the rodent lung to particles such as quartz, crocidolite asbestos, as well as high doses of other relative innocuous dusts such as titanium dioxide.