Mouse CXCL2 / MIP-2 Protein (His Tag)

Catalog Number: 50070-M08Y



General Information

Gene Name Synonym:

CINC-2a; Gro2; GROb; Mgsa-b; MIP-2; MIP-2a; Mip2; Scyb; Scyb2

Protein Construction:

A DNA sequence encoding the mouse Cxcl2 (NP_033166.1) (Ala28-Asn100) was expressed with a polyhistidine tag at the C-terminus.

Mouse Source: Yeast

QC Testing

Expression Host:

> 95 % as determined by SDS-PAGE. **Purity:**

Endotoxin:

Please contact us for more information.

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Ala 28

Molecular Mass:

The recombinant mouse Cxcl2 consists of 83 amino acids and predicts a molecular mass of 9.2 kDa.

Formulation:

Lyophilized from sterile 0.1% TFA, 30% ACN.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

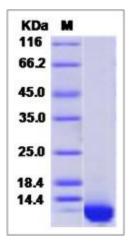
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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 toleranceconferring 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.

References

1.Driscoll KE. (2000) TNFalpha and MIP-2: role in particle-induced inflammation and regulation by oxidative stress. Toxicol Lett. 112-113: 177-83. 2. Walpen S, et al. (2001) Nitric oxide induces MIP-2 transcription in rat renal mesangial cells and in a rat model of glomerulonephritis. FASEB J. 15(3): 571-3. 3. Fahey TJ, et al. (1990) Cytokine production in a model of wound healing: the appearance of MIP-1, MIP-2, cachectin/TNF and IL-1. Cytokine. 2(2): 92-9.

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