Recombinant Human CXCL2/MIP-2 Protein

Catalog Number: PKSH033705

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

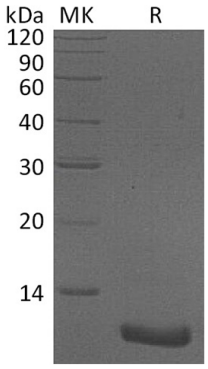
Description

Species	Human
Source	E.coli-derived Human CXCL2;MIP-2 protein Val 35-Asn107, with an N-terminal His
Calculated MW	8.7 kDa
Observed MW	12 kDa
Accession	P19875
Bio-activity	Measure by its ability to chemoattract human PBMCs using a concentration range of 10.0 - 100.0 ng/mL. Note: Results may vary from different PBMC donors.

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

Chemokine Ligand 2 (CXCL2) is a small secreted cytokine which belongs to the CXC chemokine family. It is secreted by monocytes and macrophages and chemotactic for polymorphonuclear leukocytes and hematopoietic stem cells. CXCL2 mobilizes cells by interacting with a cell surface chemokine receptor called CXCR2. It has been known to regulate immune functions mainly by chemo-attracting neutrophils. It is produced by activated monocytes and neutrophils and expressed at sites of inflammation. It is a hematoregulatory chemokine, which suppresses hematopoietic progenitor cell proliferation. It can be induced by receptor activator of NF-kappaB ligand, the osteoclast (OC) differentiation factor, through JNK and NF-kappaB signaling pathways in OC precursor cells. CXCL2 in turn enhanced the proliferation of OC precursor cells of bone marrow-derived macrophages (BMMs) through the activation of ERK. Knockdown of CXCL2 inhibited both the proliferation of and the ERK activation in BMMs. During osteoclastogenesis CXCL2 stimulated the adhesion and the migration of BMMs. CXCL2 is a novel therapeutic target for inflammatory bone destructive diseases.