Recombinant Protein Technical Manual

Recombinant Human CXCL2/MIP-2 Protein

RPES3251

Product Data:

Product SKU: RPES3251

Size: 10µg

Species: Human

Expression host: E. coli

Uniprot: P19875

Protein Information:

Molecular Mass: 7.67 kDa

AP Molecular Mass: 10 kDa

Tag:

Bio-activity:

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

Endotoxin: < 1.0 EU per µg as determined by the LAL method.

Storage: Lyophilized protein should be stored at < -20°C, though stable at room temperature for 3 weeks. Reconstituted protein solution can be stored at 4-7°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 of 20mM TrisHCl, 400mM NaCl, pH 8.5.

Reconstitution: Please refer to the printed manual for detailed information.

Application:

Synonyms: C-X-C Motif Chemokine 2; Growth-Regulated Protein Beta; Gro-Beta; Macrophage Inflammatory Protein 2-Alpha; MIP2-Alpha; CXCL2; GRO2; GROB; MIP2A; SCYB2; GRO beta; MIP2

Immunogen Information:

Sequence: Thr39-Asn107

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.