Recombinant Mouse CXCL1/GROα/KC/CINC-1 aa 29-96

Catalog Number: 1395-KC/CF

DESCRIPTION

Source	*E. coli*-derived Asn29-Lys96 Accession # P12850
N-terminal Sequence Analysis	Asn29
Predicted Molecular Mass	7.5 kDa

SPECIFICATIONS

Activity	Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2. The ED_{50} for this effect is 0.8-4 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

CXCL1, also known as KC, GROα, and CINC-1, is an approximately 8 kDa proinflammatory chemokine that plays a key role in neutrophil migration and activation (1). Mature mouse CXCL1 shares 64% and 92% aa sequence identity with human and rat CXCL1, respectively [oquendo 4133, ryseck 266, cochran 939]. It is produced by many cell types in inflammatory sites and during chronic inflammatory diseases (1). CXCL1 can associate into bioactive dimers and primarily signals through CXCR2/IL-8 RB but can also bind with lower affinity to CXCR2/IL-8 RA (5-7). It induces neutrophil migration, extravasation, respiratory burst, and degranulation and also induces T cells to produce proinflammatory IL-17 (6, 8, 9). CXCL1 additionally binds to Syndecan-1 on epithelial cells which acts as a sink for CXCL1 activity until Syndecan-1 cleavage by MMP-7 (10). CXCL1 is up-regulated in spinal cord astrocytes by inflammatory stimuli or tumor cell injection, and it exacerbates pain sensation by potentiating excitatory NMDA neurotransmission (11, 12). In the circulatory system, CXCL1 interacts with CXCR2 on endothelial cells to promote lymphatic tube formation and angiogenesis (13, 14). It promotes the hypertrophic differentiation of chondrocytes resulting in cartilage matrix deposition, calcification, and remodeling (15). It interacts with both CXCR1 and CXCR2 on adipose stromal cells and promotes their recruitment to prostate tumors in obese patients (16). It also binds CXCR2 on ovarian cancer cells, leading to cleavage of cell surface HB-EGF, transactivation of EGF R, and cell proliferation (17).

References:

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