Recombinant Human CXCL1/GROα/KC/CINC-1 aa 39-107

Catalog Number: 9196-GR/CF

DESCRIPTION

Source	*E. coli*-derived Thr39-Asn107 Accession # P09341
N-terminal Sequence Analysis	Thr39
Predicted Molecular Mass	8 kDa

SPECIFICATIONS

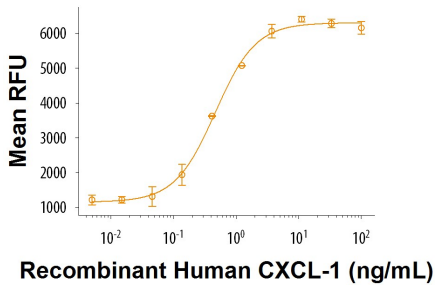
SDS-PAGE	6 kDa, reducing conditions
Activity	Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2. The ED_{50} for this effect is typically 0.15-0.9 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in 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.

DATA

Bioactivity



Recombinant Human CXCL1/GRO alpha (Catalog # 9196-GR/CF) attracts BaF3 mouse pro B cells transfected with human CXCR2. The ED_{50} for this effect is typically 0.15-0.9 ng/mL.

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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 human CXCL1 shares 64% and 67% amino acid sequence identity with mouse and rat CXCL1, respectively (2). 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 (3-5). It induces neutrophil migration, extravasation, respiratory burst, and degranulation and also induces T cells to produce proinflammatory IL-17 (4, 6, 7). CXCL1 additionally binds to Syndecan-1 on epithelial cells which acts as a sink for CXCL1 activity until Syndecan-1 cleavage by MMP-7 (8). CXCL1 is upregulated in spinal cord astrocytes by inflammatory stimuli or tumor cell injection, and it exacerbates pain sensation by potentiating excitatory NMDA neurotransmission (9, 10). In the circulatory system, CXCL1 interacts with CXCR2 on endothelial cells to promote lymphatic tube formation and angiogenesis (11, 12). It promotes the hypertrophic differentiation of chondrocytes resulting in cartilage matrix deposition, calcification, and remodeling (13). It interacts with both CXCR1 and CXCR2 on adipose stromal cells and promotes their recruitment to prostate tumors in obese patients (14). It also binds CXCR2 on ovarian cancer cells, leading to cleavage of cell surface HB-EGF, transactivation of EGF R, and cell proliferation (15). Truncated forms of CXCL1 with 3-5 amino acids removed from the N-terminus are secreted by peripheral blood monocytes and are 30-fold more active than the intact form (16).

References:

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