Recombinant Mouse LIX

Catalog Number: 433-MC/CF

DESCRIPTION

Source	*E. coli*-derived mouse LIX protein Val45-Ala118 Accession # P50228
N-terminal Sequence Analysis	Val45
Predicted Molecular Mass	8 kDa

SPECIFICATIONS

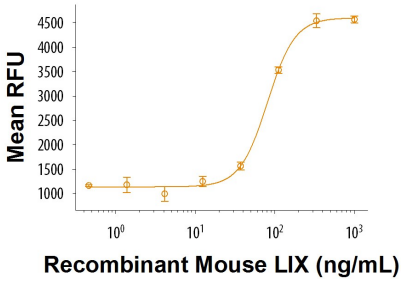
Activity	Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CXCR2. The ED_{50} for this effect is 0.03-0.1 µg/mL. Measured by its ability to induce myeloperoxidase release from cytochalasin B-treated human neutrophils. Schröder, J.M. *et al.* (1987) J. Immunol. **139**:3474. The ED_{50} for this effect is 1-3 µg/mL.
Endotoxin Level	<0.01 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.

DATA

Bioactivity



Recombinant Mouse LIX Protein Bioactivity
Recombinant Mouse LIX (Catalog # 433-MC/CF) chemoattracts BaF3 mouse pro B cells transfected with human CXCR2. The ED_{50} for this effect is 0.03-0.1 µg/mL.

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Recombinant Mouse LIX

Catalog Number: 433-MC/CF

BACKGROUND

Mouse LIX [lipopolysaccharide (LPS)-induced CXC chemokine], also previously called GCP-2, is a member of the ELR+ group of 7-9 kDa neutrophil and monocyte chemotactic proteins in the CXCL cytokine family (1). This group contains the ELR/GluLeuArg amino acid (aa) motif immediately N-terminal to the CXC motif. Other ELR+ chemokines also include mouse CXCL1 thru 3 and 15 (or KC, MIP-2, DCIP-1 and lungkine, respectively), plus human CXCL1 thru 3 and 5 thru 8 (or GROα, GROβ, GROγ, ENA-78, GCP-2, NAP-2 and IL-8, respectively) (1-4). Mouse LIX cDNA encodes 132 aa's, including a 40 aa signal peptide and up to a 92 aa mature protein that has a longer C-terminus than other ELR+ cytokines (5). The first 78 aa of mature LIX shares 63% and 55% aa identity with human CXCL6/GCP-2 and CXCL5/ENA-78, respectively. Its activity is similar but not identical to these human chemokines (1, 5). In the mouse, CXCL1/KC and CXCL2/MIP-2 expression and function may overlap with that of LIX (1, 6-10).

LIX can be produced by a variety of cells including fibroblasts, epithelial cells (including alveolar type II epithelia), endothelial cells, platelets, cardiac myocytes, preosteoblasts, oligodendrocytes, and adipose-resident macrophages (1, 4-8, 11-16). It is mainly produced when induced by LPS, IL-17 and/or TNF-α (1, 5-7, 11, 15). LIX can also be stored within specialized granules, such as platelet α-granules and endothelial cytoplasmic granules, but not Weibel-Palade bodies (6, 13). Endotoxemia increases LIX expression, especially in the heart, but also in the lung, spleen, and liver (8). LIX is downregulated by glucocorticoids and is considered a glucocorticoid-attenuated response gene or GARG (5, 8). It can also be downregulated by IL-10 and viral proteins (12, 17). Natural mouse LIX includes short forms that may be N-terminally cleaved by MMP1, 2, 8, 9, 12 or 13, and/or C-terminally cleaved by MMP1, 8, 9 or 12 (1-3, 18, 19).

Unlike other ELR+ chemokines, it is not cleaved within the ELR motif, and short forms show enhanced activity as compared to the full-length form (1-3, 18, 19). LIX activities are mainly mediated by its receptor CXCR2, which is expressed on neutrophils, mast cells and macrophages (9, 16, 17). Unlike other ELR+ chemokines, mouse LIX and human IL-8 can also signal through CXCR1 (2, 17). LIX is the most potent mouse neutrophil chemoattractant and activator (1-4, 7-9, 11, 13, 14). CXCR2-expressing cells treated with LIX show activation of NFκB signaling pathways, resulting in increased production of inflammatory cytokines such as IL-1β and TNF-α (9, 17). TNF-α produced as a result of endotoxemia or ischemia-reperfusion can, in turn, induce cardiomyocyte production of LIX, influx of neutrophils, and impedance of cardiac contractile activity (7, 14). LIX participates in the induction of LPS-induced acute lung injury and in lung ischemia-induced angiogenesis (10, 11). LIX protects neurons from apoptosis, and its downregulation by viral proteins is thought to allow neuronal apoptotic death (12). It is thought to play a protective role in inflammatory bone loss (15). High expression of LIX in obese white adipose tissue is thought to contribute to insulin resistance (16). LIX also binds the erythrocyte receptor, DARC (Duffy Antigen Receptor for Chemokines) and is reported to impair chemokine scavenging by DARC (13).

References:

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