

DESCRIPTION

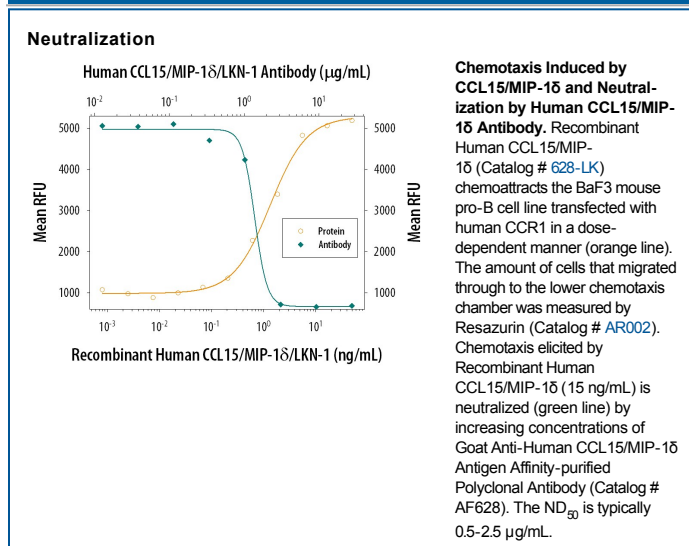
Species Reactivity	Human
Specificity	Detects human CCL15/MIP-1 δ in direct ELISAs and Western blots. In direct ELISAs and Western blots, a preference for the 68 aa residue isoform over the 92 aa residue isoform of human CCL15/LKN-1 is shown. Additionally, in direct ELISAs, less than 1% cross-reactivity with rh6Ckine, rhBLC/BCA1, rhENA78, rhEotaxin, rhEotaxin2, rhFractalkine, rhGCP2, rhGRO α , rhGRO β , rhGRO γ , rhHCC1, rhHCC4, rhI309, rhIL8, rhMCP1, rhMCP2, rhMCP3, rhMCP4, rhMDC, rhMIG, rhMIP1 α , rhMIP1 β , rhMIP3 α , rhMIP3 β , rhMPIF1, rhNAP2, rhPARC, rhRANTES, rhSDF1 α , rhSDF1 β , rhTarc, rhTECK, and rhVIC is observed. Neutralizes the biological activity of the 68 aa isoform of recombinant human (rh) CCL15/LKN-1. It will also neutralize the biological activity of the 92 aa residue isoform of rhCCL15/LKN-1 using a 2-fold higher Ig concentration.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human CCL15/MIP-1 δ Ser46-Ile113 Accession # Q16663
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μ m filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 μ g/mL	Recombinant Human CCL15/MIP-1 δ 68 aa (Catalog # 628-LK)
Neutralization		Measured by its ability to neutralize CCL15/MIP-1 δ -induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CCR1. The Neutralization Dose (ND ₅₀) is typically 0.5-2.5 μ g/mL in the presence of 15 ng/mL Recombinant Human CCL15/MIP-1 δ 68 aa.

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

CCL15, also named Leukotactin-1 (LKN-1), MIP-5, HCC-2, and NCC-3, is a novel human CC chemokine whose gene was mapped to human chromosome 17 adjacent to the HCC-1 gene. CCL15/LKN-1, together with mouse C10, mouse MIP-1 γ and human MPIF-1, constitute a subgroup of CC chemokines which contain six instead of four conserved cysteine residues. The two additional cysteine residues in CCL15/LKN-1 have been shown to form a third disulfide bond CCL15/LKN-1 cDNA encodes a 113 amino acid (aa) residue precursor protein with a putative signal peptide of 21 aa residues that is cleaved to generate a 92 aa residue mature protein. In recombinant CCL15/LKN-1 preparations produced in insect cells and in yeast, amino-terminal truncations were found to have occurred. The major forms of CCL15/LKN-1 secreted by insect cells and yeast were reported to be proteins of 68 and 66 aa residues, respectively. The full length and the amino-terminal truncated forms of human CCL15/LKN-1 have been shown to be potent chemoattractants for monocytes and T-lymphocytes. These proteins can also chemoattract eosinophils and have been shown to induce calcium flux in human CCR1 transfected cells. Additionally, CCL15/LKN-1 can suppress colony formation by human granulocyte-macrophage, erythroid, and multipotential progenitor cells stimulated by combinations of growth factors.

References:

1. Youn, B.-S. *et al.* (1997) *J. Immunol.* **159**:5201.
2. Pardigol, A. *et al.* (1998) *Proc. Natl. Acad. Sci. USA* **95**:6308.
3. Wang, W. *et al.* (1998) *J. Clinical Immunol.* **18**:214.
4. Coulin, F. *et al.* (1997) *Eur. J. Biochem.* **248**:507.