

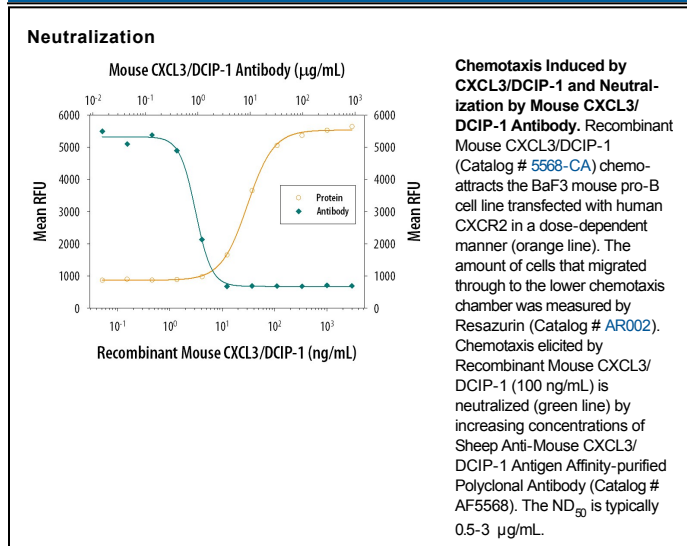
DESCRIPTION	
Species Reactivity	Mouse
Specificity	Detects mouse CXCL3/GRO γ /CINC-2/DCIP-1 in direct ELISAs and Western blots. In direct ELISAs, approximately 50% cross-reactivity with recombinant rat CINC-2 is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant mouse CXCL3/GRO γ /CINC-2/DCIP-1 Ala28-Ser100 Accession # AAl17017
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 Mouse CXCL3/DCIP-1 (Catalog # 5568-CA)
Neutralization		Measured by its ability to neutralize CXCL3/DCIP-1-induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CXCR2. The Neutralization Dose (ND ₅₀) is typically 0.5-3 μ g/mL in the presence of 100 ng/mL Recombinant Mouse CXCL3/DCIP-1.

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

CXCL3 is also known as MIP-2 β (macrophage inflammatory protein 2 beta), or DCIP-1 (dendritic cell inflammatory protein-1) in mouse, CINC2 (cytokine-induced neutrophil attractant 2) in rat, and GRO- γ (growth-regulated oncogene gamma) in humans (1, 2). It is an 8 kDa proinflammatory member of the CXC subfamily of heparin-binding chemokines, also called alpha chemokines (1-4). The Glu-Leu-Arg (ELR) motif near the CXCL3 N-terminus confers angiogenic properties and distinguishes it from interferon-inducible ELR⁻ CXC chemokines, which are angiostatic (4). ELR⁺ and ELR⁻ chemokines use CXCR2 and CXCR3 receptors, respectively (3, 4). Mature mouse CXCL3 shares 88% and 57% amino acid (aa) sequence identity with rat and human CXCL3, respectively. Among mouse ELR⁺ chemokines, it shares 82% aa sequence identity with CXCL2/GRO- β /MIP-2 and 34% - 58% with CXCL1/GRO- α /KC, CXCL5/ENA-78 and CXCL7/NAP-2. Due to their similar sequence and activity, CXCL2 and CXCL3 are sometimes referred to collectively as CXCL2/3, but are separate gene products (4-6). Mouse CXCL3 expression is induced in macrophages and early in maturation of DC by bacterial products such as lipopolysaccharides, and other inflammatory mediators (1, 7). It is chemotactic for CXCR2-expressing neutrophils, helping to recruit them to areas of inflammation (1, 7). ELR⁺ chemokines also elicit endothelial cell chemotaxis, stimulating angiogenesis and playing a role in tumor development (3, 4). ELR⁺ chemokines upregulated by ischemia play a role in ischemia-reperfusion injury (5, 6). A decoy receptor, DARC (Duffy antigen receptor for chemokines) competes with CXCR2 for ELR⁺ chemokine binding, thus downregulating their effect (8). Neutrophil influx may also be downregulated by MMP-12, which has been found to inactivate CXCL3 and other ELR⁺ chemokines by cleaving them at the ELR site (9). Over aa 28-100, mouse CXCL3 shares 87.8% aa identity with rat CINC2.

References:

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