

DESCRIPTION

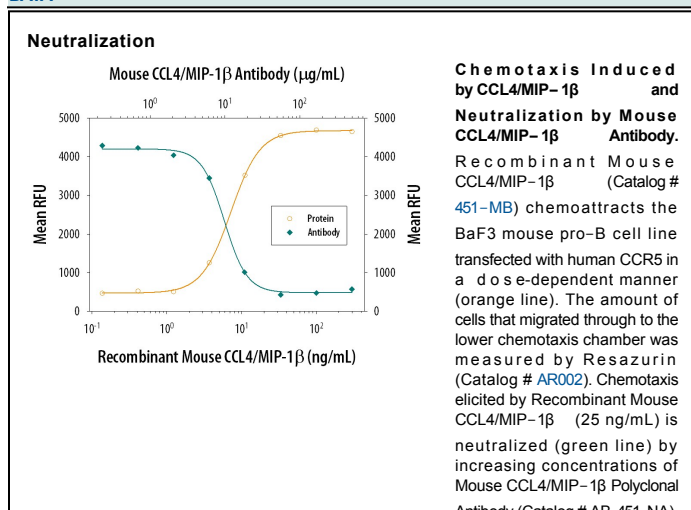
Species Reactivity	Mouse
Specificity	Detects CCL4/MIP-1 β in direct ELISAs and Western blots. In direct ELISAs and Western blots, less than 2% cross-reactivity with recombinant mouse (rm) MIP-1 α , recombinant human (rh) MIP-1 α and rhMIP-1 β is observed. This antibody will not neutralize the biological activity of rmMIP-1 α , rhMIP-1 α or rhMIP-1 β .
Source	Polyclonal Goat IgG
Purification	Protein A or G purified
Immunogen	<i>E. coli</i> -derived recombinant mouse CCL4/MIP-1 β Ala24-Asn92 Accession # Q5QNV9
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.

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	1 μ g/mL	Recombinant Mouse CCL4/MIP-1 β (Catalog # 451-MB)
Neutralization		Measured by its ability to neutralize CCL4/MIP-1 β -induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CCR5. The Neutralization Dose (ND ₅₀) is typically 4-12 μ g/mL in the presence of 25 ng/mL Recombinant Mouse CCL4/MIP-1 β .

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 1 mg/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. <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

CCL4, also known as macrophage inflammatory protein 1 beta (MIP-1 β), is a 12 kDa β chemokine that is secreted at sites of inflammation by activated leukocytes, lymphocytes, vascular endothelial cells, and pulmonary smooth muscle cells (1, 2). CCL4 attracts a variety of immune cells to sites of microbial infection as well as to other pathologic inflammation such as allergic asthma and ischemic myocardium (3–8). A CCL4 deficiency in mice promotes the development of autoantibodies, possibly as a result of compromised regulatory T cell recruitment (6). CCL4 is secreted from activated monocytes as a heterodimer with CCL3/MIP-1 α (9). The first two N-terminal amino acids can be cleaved from human CCL4 by CD26/DPPIV (10, 11). Both the full length and truncated forms exert biological activity through CCR5, and the truncated form additionally interacts with CCR1 and CCR2 (10). In humans, the ability of CCL4 to bind CCR5 inhibits the cellular entry of M-tropic HIV-1 which utilizes CCR5 as a coreceptor (2). Both forms of CCL4 block HIV-1 infection of T cells by inducing the down-regulation of CCR5 (10). Mature mouse CCL4 shares 77% and 86% aa sequence identity with human and rat CCL4, respectively.

References:

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