

DESCRIPTION

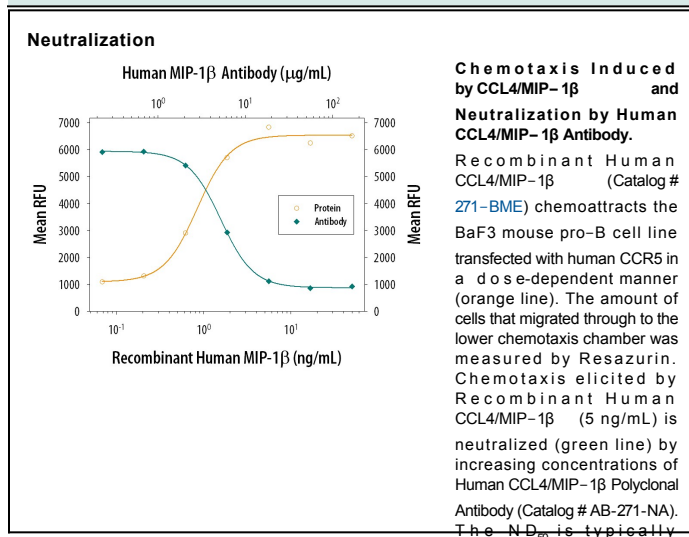
Species Reactivity	Human
Specificity	Detects human CCL4/MIP-1 β in direct ELISAs and Western blots. In direct ELISAs, less than 1% cross-reactivity with recombinant human (rh) MIP-1 α , recombinant mouse (rm) CCL4/MIP-1 β , rhMIP- γ and rmMIP-1 γ is observed.
Source	Polyclonal Goat IgG
Purification	Protein A or G purified
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant human CCL4/MIP-1 β Ala24-Asn92 Accession # P13236
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 Human CCL4/MIP-1 β (Catalog # 271-BME)
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 1.5-9 μ g/mL in the presence of 5 ng/mL Recombinant Human 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. 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 7.8 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 CCR2b (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 downregulation of CCR5 (10). Mature human CCL4 shares 77% and 80% aa sequence identity with mouse and rat CCL4, respectively.

References:

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