Mouse CCL4/MIP-1β Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF-451-NA

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects CCL4/MIP-1β in direct ELISAs and Western blots. In direct ELISAs, less than 5% cross-reactivity with recombinant human (rh) MIP-1β and recombinant mouse (rm) MIP-1γ is observed and approximately 10% cross-reactivity with rmMIP-1α is observed. Neutralizes mouse MIP-1β. This antibody will not neutralize the biological activity of rmMIP-1α. In the chemotaxis assay this antibody will partially neutralize rhMIP-1α and rhMIP-1β at a 10-30 fold higher IgG concentration.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant mouse CCL4/MIP-1β Ala24-Asn92 Accession # Q5QNV9
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

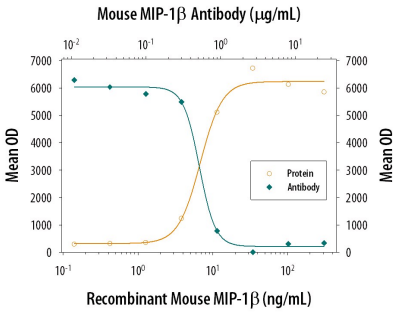
APPLICATIONS

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	0.1 µg/mL	Recombinant Mouse CCL4/MIP-1β (Catalog # 451-MB)
Immunohistochemistry	5-15 µg/mL	Perfusion fixed frozen sections of mouse thymus
Neutralization	Measured by its ability to neutralize CCL4/MIP-1β-induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CCR5. The Neutralization Dose (ND_{50}) is typically 0.2-1.0 µg/mL in the presence of 25 ng/mL Recombinant Mouse CCL4/MIP-1β.	

DATA

Neutralization



Chemotaxis Induced by CCL4/MIP-1β and Neutralization by Mouse CCL4/MIP-1β Antibody. Recombinant Mouse CCL4/MIP-1β (Catalog # 451-MB) chemoattracts the BaF3 mouse pro-B cell line transfected with human CCR5 in a dose-dependent manner (orange line). The amount of cells that migrated through to the lower chemotaxis chamber was measured by Resazurin (Catalog # AR002). Chemotaxis elicited by Recombinant Mouse CCL4/MIP-1β (25 ng/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Mouse CCL4/MIP-1β Antigen Affinity-purified Polyclonal Antibody (Catalog # AF-451-NA). The ND_{50} is typically 0.2-1.0 µg/mL.

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.
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.

Mouse CCL4/MIP-1β Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF-451-NA

BACKGROUND

CCL4, also known as macrophage inflammatory protein 1 beta (MIP-1β), 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-1α (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 downregulation of CCR5 (10). Mature mouse CCL4 shares 77% and 86% aa sequence identity with human and rat CCL4, respectively.

References:

1. Rot, A. and U.H. von Andrian (2004) Annu. Rev. Immunol. **22**:891.
2. Menten, P. *et al.* (2002) Cytokine Growth Factor Rev. **13**:455.
3. Sun, X. *et al.* (2006) Infec. Immun. **74**:5943.
4. Bisset, L.R. and Schmid-Grendelmeier, P. (2005) Curr. Opin. Pulm. Med. **11**:35.
5. Frangogiannis, N.G. (2004) Inflamm. Res. **53**:585.
6. Bystry, R.S. *et al.* (2001) Nat. Immunol. **2**:1126.
7. Oliveira, S.H.P. *et al.* (2002) J. Leukoc. Biol. **71**:1019.
8. Schall, T.J. *et al.* (1993) J. Exp. Med. **177**:1821.
9. Guan, E. *et al.* (2001) J. Biol. Chem. **276**:12404.
10. Guan, E. *et al.* (2002) J. Biol. Chem. **277**:32348.
11. Guan, E. *et al.* (2004) J. Cell. Biochem. **92**:53.