

DESCRIPTION

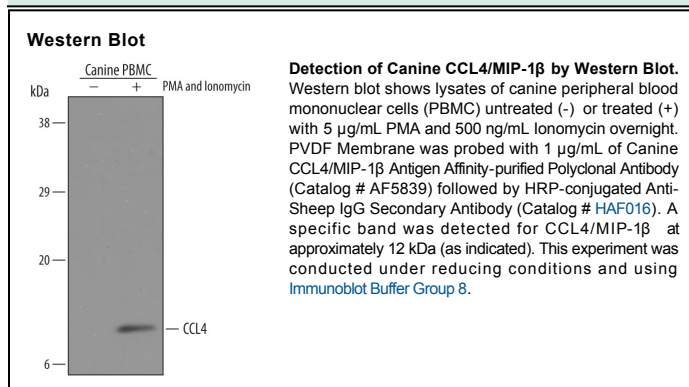
| | |
|---------------------------|--|
| Species Reactivity | Canine |
| Specificity | Detects canine CCL4/MIP-1 β in direct ELISAs and Western blots. In direct ELISAs, approximately 10% cross-reactivity with recombinant human CCL4/MIP-1 β and recombinant mouse CCL4/MIP-1 β is observed and less than 1% cross-reactivity with recombinant viral CCL4/MIP-1 β is observed. |
| Source | Polyclonal Sheep IgG |
| Purification | Antigen Affinity-purified |
| Immunogen | <i>E. coli</i> -derived recombinant canine CCL4/MIP-1 β Ala24-Asn92 Accession # NP_001005250 |
| 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 | 1 μ g/mL | See Below |
| Immunocytochemistry | 5-15 μ g/mL | Immersion fixed canine peripheral blood mononuclear cells |

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

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 inflammations 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 downregulation of CCR5 (10). Mature canine CCL4 shares 80-84% amino acid sequence identity with human, mouse, and rat CCL4.

References:

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