**Description**

Species Reactivity: Mouse

Specificity: Detects mouse CCL5/RANTES in direct ELISAs and Western blots. In direct ELISAs, less than 50% cross-reactivity with recombinant human CCL5/RANTES and recombinant cotton rat CCL5/RANTES is observed and less than 10% cross-reactivity with recombinant mouse CCL3/MIP-1α is observed.

Source: Polyclonal Goat IgG

Purification: Antigen Affinity-purified

Immunogen: E. coli-derived recombinant mouse CCL5/RANTES

Endotoxin Level: <0.30 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. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS.

**Applications**

**Recommended Concentration**

- **Sample**
  - Western Blot: 0.1 μg/mL Recombinant Mouse CCL5/RANTES (Catalog # 478-MR)
  - Immunocytochemistry: 5-15 μg/mL

**Neutralization**

Neutralization is measured by its ability to neutralize CCL5/RANTES-induced chemotaxis in the BaF3 mouse pro-B cell line transfected with human CCR5. The Neutralization Dose (ND50) is typically 0.07-0.3 μg/mL in the presence of 0.025 μg/mL Recombinant Mouse CCL5/RANTES.

**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.
- 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.

**Data**

**Neutralization**

Mouse CCL5/RANTES Antibody (µg/mL)

**Chemotaxis Induced by CCL5/RANTES and Neutralization by Mouse CCL5/RANTES Antibody.**

- Recombinant Mouse CCL5/RANTES (Catalog # 478-MR) chemotacts 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 CCL5/RANTES (0.025 µg/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Mouse CCL5/RANTES Antibody (Catalog # AF478). The ND50 is typically 0.07-0.3 µg/mL.

**Immunocytochemistry**

CCL5/RANTES in Mouse Splenocytes. CCL5/RANTES was detected in immersion fixed mouse splenocytes using Goat Anti-Mouse CCL5/RANTES Antibody Affinity-purified Polyclonal Antibody (Catalog # AF478) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue).

View our protocol for Fluorescent ICC Staining of Non-adherent Cells.
CCL5, also known as RANTES (Regulated upon Activation, Normal T cell Expressed and presumably Secreted), is an 8 kDa β-chemokine that plays a primary role in the inflammatory immune response by means of its ability to attract and activate leukocytes (1-3). Human and mouse RANTES exhibit cross-species activity on human and mouse cells (4). Mature mouse CCL5 shares 100% aa sequence identity with rat CCL5 and 75-88% with canine, cotton rat, feline, and human CCL5 (5).

CCL5 is secreted by many cell types at inflammatory sites, and it exerts a wide range of activities through the receptors CCR1, CCR3, CCR4, and CCR5 (6, 7). Inflammatory responses can be impaired by the sequestration of CCL5 by the cytomegalovirus protein US28 (8). In humans, CCR5 binding to CCL5 inhibits the infectivity of R5 (M-tropic) but not X4 (T-tropic) strains of HIV-1 (9). The two N-terminal residues of CCL5 can be removed by CD26/DPPIV, generating a protein that functions as a chemotaxis inhibitor and more effectively blocks M-tropic HIV-1 infection of monocytes (10). Oligomerization of CCL5 on glycosaminoglycans is required for CCR1-mediated leukocyte adhesion and activation as well as CCL5’s interaction with the chemokine CXCL4/PF4 (11-13). The deposition of CCL5 on activated vascular endothelial cells is crucial for monocyte adhesion to damaged vasculature, but CCL5 oligomerization is not required for the extravasation of adherent leukocytes (14-16). CCL5 is upregulated in breast cancer and promotes tumor progression through the attraction of pro-inflammatory macrophages in addition to its actions on tumor cells, stromal cells, and the vasculature (17).

References: