Mouse CCL5/RANTES Alexa Fluor® 750-conjugated Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF478S
100 µg

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse CCL5/RANTES in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant mouse CCL5/RANTES
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

Neutralization	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

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).

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