

MI γ /CXCL9 Polyclonal Antibody

catalog number: D-AB-10440L

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

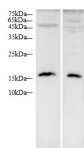
Reactivity	Mouse;Rat
Immunogen	Recombinant Mouse MI γ /CXCL9 protein expressed by E.coli
Host	Rabbit
Isotype	IgG
Purification	Antigen Affinity Purification
Conjugation	Unconjugated
buffer	PBS with 0.05% proclin 300, 1% protective protein and 50% glycerol,pH7.4

Applications

Recommended Dilution

WB	1:500-1:1000
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Data



Western blot with MI γ /CXCL9 Polyclonal antibody at dilution of 1:1000.lane 1:Mouse brain,lane 2:Rat brain

Observed-MV:15 kDa

Calculated-MV:14 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

Background

Chemokine (C-X-C motif) ligand 9 (CXCL9),also known as Monokine induced by gamma interferon (MIG),is a small cytokine belonging to the CXC chemokine family. The function of this chemokine has not been specifically defined; however,it is thought to be involved in T cell trafficking. CXCL9/MIG functions as one of the three ligands of chemokine receptor CXCR3 which is a G protein-coupled receptor found predominantly on T cells. CXCL9/MIG,together with CXCL10 and CXCL11,may activate CXCR3 by binding to it. CXCL9 serves as a cytokine that affects the growth, movement,or activation state of cells that participate in immune and inflammatory response. It has been observed that tumour endothelial cells secrete high levels of CXCL9 in all,and CXCL10 in most melanoma metastases. Experiment data represent novel mechanisms by which tumour cells in melanoma metastases might use the chemokine-expressing endothelium to leave the tumour and eventually to form additional metastases at distinct sites. Experiment results also improved that CXCL9/MIG plays an important role in CD4⁺ T lymphocyte recruitment and development of CAV,MOM A-2⁺ macrophages are the predominant recipient-derived source of CXCL9/MIG,and recipient CD4 lymphocytes are necessary for sustained CXCL9/MIG production and CAV development in this model. Neutralization of the chemokine CXCL9/MIG may have therapeutic potential for the treatment of chronic rejection after heart transplantation.

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