CXCR3 polyclonal antibody

Catalog # PAB8944 Size 100 ug

Specification

Product Description	Rabbit polyclonal antibody raised against synthetic peptide of CXCR3.
Immunogen	A synthetic peptide corresponding to amino acids 8-33 of human CXCR3.
Sequence	VETFGTTSYYDDVGLLCEKADTRALM
Host	Rabbit
Reactivity	Human
Form	Liquid
Quality Control Testing	Antibody Reactive Against Synthetic Peptide.
Recommend Usage	Western Blot (1:500) Immunoprecipitation (1:250) The optimal working dilution should be determined by the end user.
Storage Buffer	In buffer containing 0.02% sodium azide
Storage Instruction	Store at -20°C. Aliquot to avoid repeated freezing and thawing.
Note	This product contains sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which shoul d be handled by trained staff only.

Applications

- Immunoprecipitation
- Western Blot

Gene Info — CXCR3

Copyright © 2023 Abnova Corporation. All Rights Reserved.

Entrez GeneID	2833
Gene Name	CXCR3
Gene Alias	CD182, CD183, CKR-L2, CMKAR3, GPR9, IP10-R, Mig-R, MigR
Gene Description	chemokine (C-X-C motif) receptor 3
Omim ID	300574
Gene Ontology	Hyperlink
Gene Summary	This gene encodes a G protein-coupled receptor with selectivity for three chemokines, termed IP10 (interferon-g-inducible 10 kDa protein), Mig (monokine induced by interferon-g) and I-TAC (interferon-inducible T cell a-chemoattractant). IP10, Mig and I-TAC belong to the structural subfamily of CXC chemokines, in which a single amino acid residue separates the first two of four highly conserved Cys residues. Binding of chemokines to this protein induces cellular responses that are involved in leukocyte traffic, most notably integrin activation, cytoskeletal changes and chemotactic migration. Inhibition by Bordetella pertussis toxin suggests that heterotrimeric G protein of the Gi-subclass couple to this protein. Signal transduction has not been further analyzed but may include the same enzymes that were identified in the signaling cascade induced by other chemokine receptors. As a consequence of chemokine-induced cellular desensitization (phosphorylation-dependent receptor internalization), cellular responses are typically rapid and short in duration. Cellular responsiveness is restored after dephosphorylation of intracellular receptors and subsequent recycling to the cell surface. This gene is prominently expressed in in vitro cultured effector/memory T cells, and in T cells present in many types of inflamed tissues. In addition, IP10, Mig and I-TAC are commonly produced by local cells in inflammatory lesion, suggesting that this gene and its chemokines participate in the recruitment of inflammatory cells. Therefore, this protein is a target for the development of small molecular weight antagonists, which may be used in the treatment of diverse inflammatory diseases. Multiple transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq
Other Designations	G protein-coupled receptor 9|IP10 receptor|Mig receptor|OTTHUMP00000070257|chemokine (C-X-C) receptor 3

Publication Reference

- Antibody-mediated blockade of the CXCR3 chemokine receptor results in diminished recruitment of T helper 1 cells into sites of inflammation.

  Xie JH, Nomura N, Lu M, Chen SL, Koch GE, Weng Y, Rosa R, Di Salvo J, Mudgett J, Peterson LB, Wicker LS, DeMartino JA.

  Journal of Leukocyte Biology 2003 Jun; 73(6):771.

Copyright © 2023 Abnova Corporation. All Rights Reserved.

- Chemokine receptors: signposts to brain development and disease.

  Tran PB, Miller RJ.

  Nature Reviews. Neuroscience 2003 Jun; 4(6):444.

- The role of chemokines and chemokine receptors in progressive renal diseases.

  Segerer S.

  American Journal of Kidney Diseases: the Official Journal of the National Kidney Foundation 2003 Mar; 41(3 Suppl 1):S15.

Pathway

- Chemokine signaling pathway
- Cytokine-cytokine receptor interaction

Disease

- Asthma
- Bronchiolitis
- Coronary Artery Disease
- Genetic Predisposition to Disease
- Infant
- Respiratory Syncytial Virus Infections

Copyright © 2023 Abnova Corporation. All Rights Reserved.