Mouse VEGF-D Antibody

Monoclonal Rat IgG_{2A} Clone # 90409

Catalog Number: MAB469

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse VEGF-D in direct ELISAs and Western blots. In Western blots, no cross-reactivity with recombinant mouse (rm) $VEGF_{165}$, rmVEGF-B_{186}, recombinant human (rh) VEGF-C, or rhVEGF-D is observed.
Source	Monoclonal Rat IgG_{2A} Clone # 90409
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	*E. coli*-derived recombinant mouse VEGF-D Phe98-Ser206 Accession # P97946
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 as a 0.2 μm 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 µg/mL	Recombinant Mouse VEGF-D (Catalog # 469-VD)
Mouse VEGF-D Sandwich Immunoassay		**Reagent**
ELISA Capture	2-8 µg/mL	Mouse VEGF-D Antibody (Catalog # MAB469)
ELISA Detection	0.1-0.4 µg/mL	Mouse VEGF-D Biotinylated Antibody (Catalog # BAF469)
Standard		Recombinant Mouse VEGF-D (Catalog # 469-VD)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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.

BACKGROUND

Vascular endothelia growth factor D (VEGF-D), also known as *c-fos*-induced growth factor (FIGF), is a secreted glycoprotein of the VEGF/PDGF family. VEGFs regulate angiogenesis and lymphangiogenesis during development and tumor growth, and are characterized by eight conserved cysteine residues that form a cysteine-knot structure (1-3). VEGF-C and VEGF-D, which share 23% amino acid (aa) sequence identity, are uniquely expressed as preproproteins that contain long N- and C-terminal propeptide extensions around the VEGF homology domain (VHD) (1, 2). Proteolytic processing of either 358 aa or 326 aa splice forms of mouse VEGF-D preproprotein creates a secreted proprotein. Further processing by extracellular serine proteases, such as plasmin or furin-like proprotein convertases, forms mature VEGF-D consisting of non-covalently linked 42 kDa homodimers of the 117 aa VHD (4-7). Mature mouse VEGF-D shares 94%, 99%, 93%, 91% and 89% aa identity with the VHD of human, rat, equine, canine and bovine VEGF-D, respectively. It is expressed in adult lung, heart, muscle, and small intestine, and is most abundantly expressed in fetal lungs and skin (1 - 4). Mouse and human VEGF-D are ligands for VEGF receptor 3 (VEGF-R3, also called Flt-4) that are active across species and show enhanced affinity when processed (8). Unlike human VEGF-D, which is also a ligand for VEGF-R2 (also called Flk-1 or KDR), mouse VEGF-D does not bind to VEGF-R2 (8). VEGF-R3 is strongly expressed in lymphatic endothelial cells and is essential for regulation of the growth and differentiation of lymphatic endothelium (1, 2). While VEGF-C is the critical ligand for VEGF-R3 during embryonic lymphatic development, VEGF-D is most active in neonatal lymphatic maturation and bone growth (9-11). Both promote tumor lymphangiogenesis (12). Consonant with their activity on VEGF receptors, binding of VEGF-C and VEGF-D to neuropilins contributes to VEGF-R3 signaling in lymphangiogenesis, while binding to integrin α9β1 mediates endothelial cell adhesion and migration (13, 14).

References:

1. Roy, H. *et al.* (2006) FEBS Lett. **580**:2879.
2. Otrock, Z.H. *et al.* (2007) Blood Cells Mol. Dis. **38**:258.
3. Orlandini, M. *et al.* (1996) Proc. Natl. Acad. Sci. USA **93**:11675.
4. Stacker, S.A. *et al.* (1999) J. Biol. Chem. **274**:32127.
5. McColl, B.K. *et al.* (2003) J. Exp. Med. **198**:863.
6. McColl, B.K. *et al.* (2007) FASEB J. **21**:1088.
7. Baldwin, M.E. *et al.* (2001) J. Biol. Chem. **276**:44307.
8. Baldwin, M.E. *et al.* (2001) J. Biol. Chem. **276**:19166.
9. Baldwin, M.E. *et al.* (2005) Mol. Cell. Biol. **25**:2441.
10. Karpanen, T. *et al.* (2006) Am. J. Pathol. **169**:708.
11. Orlandini, M. *et al.* (2006) J. Biol. Chem. **281**:17961.
12. Stacker, S.A. *et al.* (2001) Nature Med. **7**:186.
13. Karpanen, T. *et al.* (2006) FASEB J. **20**:1462.
14. Vlahakis, N.E. *et al.* (2005) J. Biol. Chem. **280**:4544.