

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse VEGF-B ₁₈₆ in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human (rh) VEGF ₁₆₅ , rhVEGF-B ₁₈₆ , recombinant mouse (rm) VEGF-B ₁₆₇ , rhVEGF-C, rmVEGF-D, rhCTGF, rhP/GF, or rhLDGF is observed.
Source	Monoclonal Rat IgG _{2B} Clone # 124112
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant mouse VEGF-B ₁₈₆ Pro22-Ala207 Accession # P49766.2
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-B ₁₈₆ (Catalog # 767-VE)

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. <ul style="list-style-type: none"> ● 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 endothelial growth factor B (VEGF-B; also known as VFR) is a member of the VEGF-PDGF supergene family of growth factor molecules (1-4). Five mouse members have been identified, including VEGF-A, -B, -C, -D, and PIGF(-2) (1, 5). VEGF family members are disulfide-linked homo- and heterodimeric proteins that are important regulators of vasculogenesis and lymphangiogenesis. Two isoforms of mouse VEGF-B are produced by alternative splicing (6, 7). The long form (VEGF₁₈₆) is 207 amino acids (aa) in length, with a putative 21 aa signal sequence and a 186 aa (32 kDa) mature region. The short form (VEGF₁₆₇) is 188 aa in length, with a 21 aa signal sequence and a 167 aa (21 kDa) mature segment. The two isoforms share the same N-terminal 94 aa residue containing the cysteine knot VEGF homology domain (6-8). VEGF₁₈₆ is O-glycosylated; VEGF₁₆₇ is not. VEGF₁₆₇ binds heparin; VEGF₁₈₆ does not. Thus, VEGF₁₈₆ is secreted and freely diffusible in tissues (7). However, the VEGF-B₁₆₇ isoform is the predominant form in tissue (9). Mouse VEGF-B₁₈₆ shares 93% and 87% aa identity with bovine and human VEGF-B₁₈₆, respectively. Mouse VEGF-B₁₆₇ also shares 90% and 88% aa identity with bovine and human VEGF-B₁₆₇, respectively. Unlike VEGF₁₆₇, VEGF-B₁₈₆ can undergo proteolytic processing to generate a partially processed 48 kDa heterodimer (16 kDa and 32 kDa) and a fully processed 32 kDa homodimer (two 16 kDa). Processing appears to occur at Arg 127 of the mature protein (10). VEGF-B can heterodimerize with VEGF (7). Both VEGF-B isoforms can bind to VEGF receptor 1 (VEGF R1), but not VEGF R2 or VEGF R3 (11). VEGF-B₁₆₇ also binds neuropilin-1, but only the 127 aa processed form of VEGF-B₁₈₆ binds neuropilin-1 (10). As a dimer, the full length VEGF-B₁₈₆ does not interact with neuropilin-1, while any dimer that contains the processed VEGF-B₁₂₇ subunit will interact with neuropilin-1 (10). The importance of differential neuropilin binding is unclear. VEGF-B deficient mice display an atrial conduction deficit (12). On endothelial cells, ligation of VEGF R1 by VEGF-B has been shown to regulate the expression and activity of urokinase type plasminogen activator and plasminogen activator inhibitor 1 (11).

References:

1. Li, X. and U. Eriksson (2001) *Int. J. Biochem Cell Biol.* **33**:421.
2. Olofsson, B. *et al.* (1999) *Curr. Opin. Biotechnol.* **10**:528.
3. Clauss, M. (2000) *Semin. Thromb. Hemost.* **26**:561.
4. Matsumoto, T. and L. Claesson-Welsh (2001) *Sci STKE Dec.* **11**(112):RE21.
5. DiPalma, T. *et al.* (1996) *Mamm. Genome* **7**:6.
6. Olofsson, B. *et al.* (1996) *Proc. Natl. Acad. Sci. USA* **93**:2576.
7. Olofsson, B. *et al.* (1996) *J. Biol. Chem.* **271**:19310.
8. Twonson, S. *et al.* (1996) *Biochem. Biophys. Res. Commun.* **220**:922.
9. Li, X. *et al.* (2001) *Growth Factors* **19**:49.
10. Makinen, T. *et al.* (1999) *J. Biol. Chem.* **274**:21217.
11. Olofsson, B. *et al.* (1998) *Proc. Nat. Acad. Sci. USA* **95**:11709.
12. Aase, K. *et al.* (2001) *Circulation* **104**:358.