

DESCRIPTION

Source *E. coli*-derived
Pro22-Lys188
Accession # AAC52553

N-terminal Sequence Analysis Pro22

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 19 kDa (monomer)

SPECIFICATIONS

Activity Measured by its binding ability in a functional ELISA. Immobilized rrNeuropilin-1/Fc Chimera at 4 µg/mL (100 µL/well) can bind rmVEGF-B₁₆₇ with a linear range of 0.8-50 ng/mL.

Endotoxin Level <0.01 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile 4 mM HCl containing at least 0.1% human or bovine serum albumin.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.
- 3 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 PlGF(-2) (1, 5). VEGF family members are disulfide-linked homo- and heterodimeric proteins that are important regulators of vasculogenesis and lymphangiogenesis. Mouse VEGF-B has two isoforms, a 32 kDa single chain and a 21 kDa single chain form (6, 7). The long form (VEGF-B₁₈₆) is 207 amino acids (aa) in length, with a 21 aa signal sequence and a 186 aa mature region. The short form (VEGF-B₁₆₇) is 188 aa in length, with a 21 aa signal sequence and a 167 aa mature segment. Each mature isoform shows the same N-terminal 94 aa that contains a cysteine knot VEGF homology domain (6 - 8). VEGF-B₁₈₆ is O-glycosylated; VEGF-B₁₆₇ is not. VEGF-B₁₆₇ binds heparin; VEGF-B₁₈₆ does not. Thus, VEGF-B₁₈₆ is secreted and freely diffusible in tissues (7). However, the VEGF-B₁₆₇ isoform is the predominant form in tissue (9). Mouse VEGF-B₁₈₆ is 93% and 87% aa identical to bovine and human VEGF-B₁₈₆, respectively; mouse VEGF-B₁₆₇ is 90% and 88% aa identical to bovine and human VEGF-B₁₆₇, respectively. The mouse VEGF-B₁₆₇ homodimer is 42 kDa in size, while the VEGF-B₁₈₆ homodimer is 62 kDa in size. Unlike VEGF₁₆₇, VEGF-B₁₈₆ undergoes proteolytic processing that creates a partially processed 48 kDa homodimer and a fully processed 32 kDa homodimer. Processing appears to occur at Arg127 of the mature form (10). Both forms of VEGF-B can heterodimerize with VEGF (7). Both VEGF-B isoforms 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, 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.