

DESCRIPTION

Source *Spodoptera frugiperda*, Sf 21 (stably transfected)-derived
Gln20-Ala207 & Gln20-Arg148
Accession # P49766

N-terminal Sequence Analysis Gln20

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 15.2 kDa

SPECIFICATIONS

SDS-PAGE 28 kDa and 17 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
Immobilized rmFlt-1/Fc Chimera at 1 µg/mL (100 µL/well) can bind rmVEGF-B₁₈₆ with a linear range of 0.1-10 ng/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >90%, by SDS-PAGE under reducing conditions and visualized by silver stain.

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

PREPARATION AND STORAGE

Reconstitution Reconstitute at 10 µg/mL in sterile PBS 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 P/IGF(-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:

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