

DESCRIPTION

Source *Spodoptera frugiperda*, Sf 21 (stably transfected)-derived human VEGF protein
Ala27-Asp191
Accession # AAL27435

N-terminal Sequence Analysis Ala27

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 19.1 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 19-24 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA. Serially diluted Recombinant Human (rh) VEGF_{165b} is added to a Recombinant Human VEGF R2/KDR/FIk-1 Fc Chimera (Catalog # 357-KD) coated plate (2 µg/mL, 100 µL/well). Bound rhVEGF_{165b} is detected by a human VEGF_{165b} specific antibody (Catalog # MAB3045). The concentration of rhVEGF_{165b} that produces half-maximum response is approximately 0.12-0.6 nM.

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

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in PBS.

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 (VEGF or VEGF-A), also known as vascular permeability factor (VPF), is a potent mediator of both angiogenesis and vasculogenesis in the fetus and adult (1-3). It is a member of the PDGF family that is characterized by the presence of eight conserved cysteine residues and a cystine knot structure (4). Humans express two sets of alternately spliced isoforms of 121, 145, 165, 183, 189, and 206 amino acids (aa) in length (4). VEGF₁₆₅ appears to be the most abundant and potent of the angiogenic isoform set, followed by VEGF₁₂₁ and VEGF₁₈₉ (3, 4). The anti-angiogenic or "b" set of isoforms is differentially spliced to contain six alternate amino acids at the C-terminus, and are the more highly expressed isoforms in normal adult tissue (5). VEGF_{165b}, like VEGF₁₂₁ but unlike most angiogenic isoforms, does not bind heparins and is therefore diffusible (4, 6). Human VEGF_{165b} shares 88% aa sequence identity with corresponding regions of mouse and rat, 96% with porcine, 95% with canine, and 93% with feline, equine and bovine VEGF_{165b}, respectively. VEGFs bind the type I transmembrane receptor tyrosine kinases VEGF R1 (also called Flt-1) and VEGF R2 (Flk-1/KDR) on endothelial cells (4). Although VEGF affinity is highest for binding to VEGF R1, VEGF R2 appears to be the primary mediator of VEGF angiogenic activity (3, 4). The affinity of VEGF_{165b} for VEGF R2 is similar to that of VEGF₁₆₅, but VEGF_{165b} only partially activates VEGF R2 such that the kinase regulatory site Y1054 is not phosphorylated (6). VEGF_{165b} also does not bind neuropilin-1, suggesting that the functional difference between VEGF₁₆₅ and VEGF_{165b} maybe due to either the lack of neuropilin-1 co-signaling or unique downstream signaling activated by VEGF_{165b} (8). Since VEGF_{165b} may compete with angiogenic VEGFs for VEGF R2 sites, its ectopic expression in tumors has been shown to inhibit their growth (7).

References:

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6. Kawamura, H. *et al.* (2008) *Cancer Res.* **68**:4683.
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