

DESCRIPTION

Source *Spodoptera frugiperda*, Sf 21 (baculovirus)-derived
Ala27-Arg191
Accession # NP_001165097

N-terminal Sequence Analysis Ala27

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 19.2 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 20-22 kDa, reducing conditions
39-42 kDa, non-reducing conditions

Activity Measured in a cell proliferation assay using HUVEC human umbilical vein endothelial cells. Conn, G. *et al.* (1990) Proc. Natl. Acad. Sci. USA 87:1323.
The ED₅₀ for this effect is typically 1-6 ng/mL.
The specific activity of recombinant human VEGF₁₆₅ is approximately 1.7 x 10³ U/μg, which is calibrated against recombinant human VEGF₁₆₅ WHO Standard (NIBSC code: 02/286).

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

Purity >97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

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 PBS containing at least 0.1% human or bovine serum albumin. Alternatively, reconstitute at 500 μg/mL in sterile 4 mM HCl containing 0.1% human or bovine serum albumin.

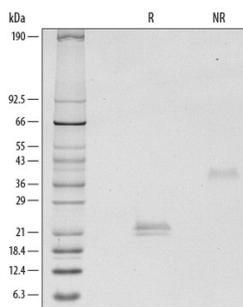
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

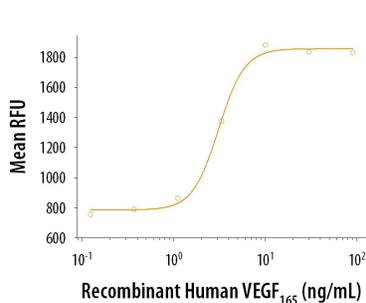
DATA

SDS-PAGE



1 μg/lane of Recombinant Human VEGF₁₆₅ was resolved by SDS-PAGE with silver staining, under reducing (R) and non-reducing (NR) conditions, showing major bands at 20-22 kDa and 39-42 kDa, respectively. Multiple bands in gel are due to glycosylation.

Bioactivity



Recombinant Human VEGF₁₆₅ (Catalog # 293-VE) stimulates proliferation in HUVEC human umbilical vein endothelial cells. The ED₅₀ is typically 1-6 ng/mL.

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 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 isoform, followed by VEGF₁₂₁ and VEGF₁₈₉ (3, 4). Isoforms other than VEGF₁₂₁ contain basic heparin-binding regions and are not freely diffusible (4). Human VEGF₁₆₅ 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, respectively. VEGF binds 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). VEGF₁₆₅ binds the semaphorin receptor, Neuropilin-1 and promotes complex formation with VEGF R2 (5). VEGF is required during embryogenesis to regulate the proliferation, migration, and survival of endothelial cells (3, 4). In adults, VEGF functions mainly in wound healing and the female reproductive cycle (3). Pathologically, it is involved in tumor angiogenesis and vascular leakage (6, 7). Circulating VEGF levels correlate with disease activity in autoimmune diseases such as rheumatoid arthritis, multiple sclerosis and systemic lupus erythematosus (8). VEGF is induced by hypoxia and cytokines such as IL-1, IL-6, IL-8, oncostatin M and TNF- α (3, 4, 9).

References:

1. Leung, D.W. *et al.* (1989) *Science* **246**:1306.
2. Keck, P.J. *et al.* (1989) *Science* **246**:1309.
3. Byrne, A.M. *et al.* (2005) *J. Cell. Mol. Med.* **9**:777.
4. Robinson, C.J. and S.E. Stringer (2001) *J. Cell. Sci.* **114**:853.
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6. Weis, S.M. and D.A. Cheresh (2005) *Nature* **437**:497.
7. Thurston, G. (2002) *J. Anat.* **200**:575.
8. Carvalho, J.F. *et al.* (2007) *J. Clin. Immunol.* **27**:246.
9. Angelo, L.S. and R. Kurzrock (2007) *Clin. Cancer Res.* **13**:2825.