

**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 ED<sub>50</sub> for this effect is typically 1-6 ng/mL.  
The specific activity of recombinant human VEGF<sub>165</sub> is approximately 1.7 x 10<sup>3</sup> U/μg, which is calibrated against recombinant human VEGF<sub>165</sub> 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 trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution** Reconstitute at 100 μg/mL in sterile PBS. Alternatively, reconstitute at 500 μg/mL in sterile 4 mM HCl.

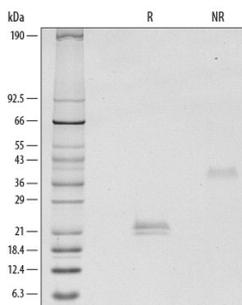
**Shipping** The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

**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.

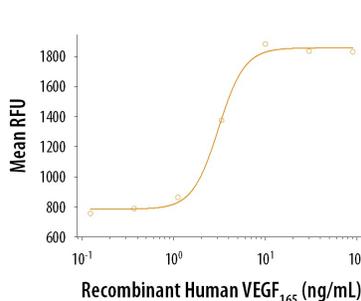
**DATA**

**SDS-PAGE**



1 μg/lane of Recombinant Human VEGF<sub>165</sub> 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 VEGF<sub>165</sub> (Catalog # 293-VE/CF) stimulates proliferation in HUVEC human umbilical vein endothelial cells. The ED<sub>50</sub> 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). VEGF<sub>165</sub> appears to be the most abundant and potent isoform, followed by VEGF<sub>121</sub> and VEGF<sub>189</sub> (3, 4). Isoforms other than VEGF<sub>121</sub> contain basic heparin-binding regions and are not freely diffusible (4). Human VEGF<sub>165</sub> 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). VEGF<sub>165</sub> 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- $\alpha$  (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.