

DESCRIPTION

Source	<i>E. coli</i> -derived human VEGF protein Ala27-Arg191 Accession # NP_001165097
N-terminal Sequence Analysis	Met-Ala27 & Ala27
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	19 kDa

SPECIFICATIONS

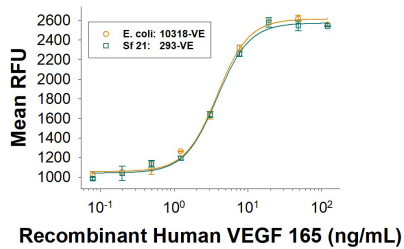
SDS-PAGE	19-22 kDa
Activity	Measured in a cell proliferation assay using HUVEC human umbilical vein endothelial cells. Conn, G. <i>et al.</i> (1990) Proc. Natl. Acad. Sci. USA 87:1323. The ED ₅₀ for this effect is 1-8 ng/mL. The specific activity of Recombinant Human VEGF 165 (Catalog # 10318-VE) is approximately 1.1 x 10 ³ U/μg, which is calibrated against recombinant human VEGF 165 WHO Standard (NIBSC code: 02/286).
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 HCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 μg/mL in sterile PBS. Alternatively, reconstitute at 100-500 μg/mL in sterile 4 mM HCl.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 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.

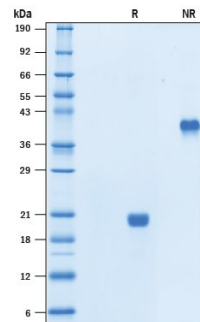
DATA

Bioactivity



E. coli-expressed Recombinant Human VEGF 165 (Catalog # 10318-VE) stimulated proliferation in HUVEC human umbilical endothelial cells. The ED₅₀ for this effect is 1-8 ng/mL. *E. coli*-derived has similar activity to Sf 21 (baculovirus)-derived Recombinant Human VEGF 165 (Catalog # 293-VE).

SDS-PAGE



2 μg/lane of Recombinant Human VEGF 165 (Catalog # 10318-VE) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 19-22 kDa and 38-44 kDa, respectively.

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.
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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.