

**DESCRIPTION**

<b>Source</b>	Human embryonic kidney cell, HEK293-derived human VEGF protein Ala27-Arg191 Accession # NP_001165097.1
<b>N-terminal Sequence Analysis</b>	Ala27
<b>Structure / Form</b>	Disulfide linked homodimer
<b>Predicted Molecular Mass</b>	19 kDa

**SPECIFICATIONS**

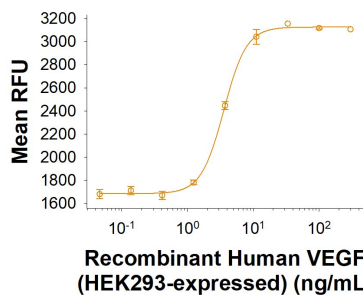
<b>SDS-PAGE</b>	19-25 kDa, under reducing conditions.
<b>Activity</b>	Measured in a cell proliferation assay using HUVEC human umbilical vein endothelial cells. Conn, G. <i>et al.</i> (1990) Proc. Natl. Acad. Sci. USA 87:1323. The ED <sub>50</sub> for this effect is 1.50-12.0 ng/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute the 10 µg size at 100 µg/mL in 4mM HCl. Reconstitute all other sizes at 500 µg/mL in 4mM HCl.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

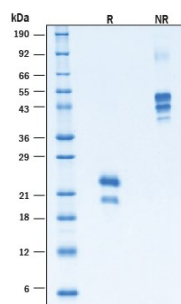
**DATA**

**Bioactivity**



**Recombinant Human VEGF (HEK293-expressed) Protein Bioactivity.** Recombinant Human VEGF (HEK293-expressed) (Catalog # 11458-VE) stimulates proliferation in HUVEC human umbilical vein endothelial cells. The ED<sub>50</sub> for this effect is 1.50-12.0 ng/mL.

**SDS-PAGE**



**Recombinant Human VEGF (HEK293-expressed) Protein SDS-PAGE.** 2 µg/lane of Recombinant Human VEGF (HEK293-expressed) Protein (Catalog # 11458-VE) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 19-25 kDa, under reducing conditions.

**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). Due to its role in angiogenesis of blood vessels, tumor and stroma cells use VEGF to stimulate formation of blood vessels and the proliferation and survival of endothelial cells. Specific immunotherapies targeting the VEGF signaling pathway include the recombinant antibody against VEGF (Bevacizumab), antibodies targeting the main VEGF receptor (VEGFR2), and small molecule inhibitors against VEGF receptor tyrosine kinases (10). Immune checkpoint inhibitors are an important tool in cancer therapies as tumor cells can hijack immune checkpoint signals to evade detection by immune cells. In addition to stimulating the formation of tumor blood vessels, VEGF has immunosuppressive effects by acting on dendritic cells to block their antigen-presenting and T cell stimulatory functions. Targeting VEGF in combination with other immune checkpoint ligands or receptors may prove more effective in immunotherapy approaches to certain cancer types (11). Because of its role in the formation of blood vessels, VEGF is also an important factor in skeletal development where blood supply and vascularization are crucial. This has made VEGF an important molecule in regenerative studies for bone repair as sustained release of VEGF has been shown to improve the efficiency of bone regeneration (12). In differentiation protocols for stem cells, VEGF is a commonly added growth factor for the transformation of induced pluripotent stem cells into hematopoietic progenitor cells used to make Natural Killer cells (13, 14). VEGF has also been used to transform intermediate mesoderm into kidney glomerular podocytes or stem cell-derived liver spheres (15, 16). VEGF may also be used in assistance of stem cell transplantations by supporting angiogenesis at sites of stem cell transplants or as a honing tool for adipose-derived mesenchymal stem cells or bone marrow stem cells to migrate to (17, 18).

**References:**

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