

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived Ala207-Arg318 Accession # NP_001020541
<b>N-terminal Sequence Analysis</b>	Ala207
<b>Structure / Form</b>	Disulfide-linked homodimer
<b>Predicted Molecular Mass</b>	14.1 kDa (monomer)

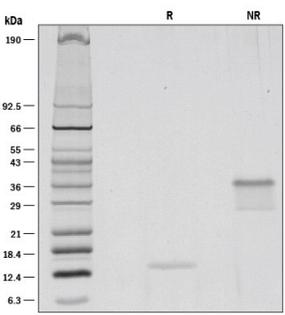
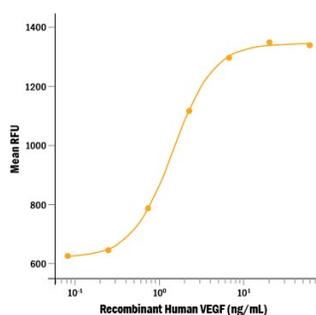
**SPECIFICATIONS**

<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 typically 0.75-3.75 ng/mL.
<b>Endotoxin Level</b>	<0.01 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	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**

<b>Reconstitution</b>	Reconstitute at 50 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
<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>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <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**

<p><b>SDS-PAGE</b></p>  <p>1 µg/lane of Recombinant Human VEGF (aa 207-318) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing bands at 13 kDa and 36 kDa, respectively.</p>	<p><b>Bioactivity</b></p>  <p>Recombinant Human VEGF (aa 207-318) (Catalog # 298-VS) stimulates cell proliferation in HUVEC human umbilical vein endothelial cells in a dose-dependent manner. The ED<sub>50</sub> is typically 0.75-3.75 ng/mL.</p>
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**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 cysteine 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). VEGF<sub>121</sub> is the only form that lacks a basic heparin-binding region and is freely diffusible (4). Mouse embryos expressing only the corresponding isoform (VEGF<sub>120</sub>) do not survive to term, and show defects in skeletogenesis (5). Human VEGF<sub>121</sub> shares 87% aa sequence identity with corresponding regions of mouse and rat, 93% with feline, equine and bovine, and 91%, 95% and 96% with ovine, canine and porcine 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; VEGF<sub>121</sub> binding has also been reported (6). 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 (7, 8). Circulating VEGF levels correlate with disease activity in autoimmune diseases such as rheumatoid arthritis, multiple sclerosis and systemic lupus erythematosus (9). VEGF is induced by hypoxia and cytokines such as IL-1, IL-6, IL-8, oncostatin M and TNF- $\alpha$  (3, 4, 10).

**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.
5. Zelzer, E. *et al.* (2002) *Development* **129**:1893.
6. Pan, Q. *et al.* (2007) *J. Biol. Chem.* **282**:24049.
7. Weis, S.M. and D.A. Cheresh (2005) *Nature* **437**:497.
8. Thurston, G. (2002) *J. Anat.* **200**:575.
9. Carvalho, J.F. *et al.* (2007) *J. Clin. Immunol.* **27**:246.
10. Angelo, L.S. and R. Kurzrock (2007) *Clin. Cancer Res.* **13**:2825.