

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived Ala27-Arg171, with an N-terminal Met Accession # NP_001191313
<b>N-terminal Sequence Analysis</b>	Met-Ala27 & Pro28
<b>Structure / Form</b>	Disulfide-linked homodimer
<b>Predicted Molecular Mass</b>	17 kDa (monomer)

**SPECIFICATIONS**

<b>SDS-PAGE</b>	20 kDa, 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 <b>87</b> :1323. The ED <sub>50</sub> for this effect is 12-60 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 under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in HCl. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 µg/mL in 4 mM 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>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <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>

**BACKGROUND**

Vascular endothelial growth factor (VEGF or VEGFA), 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 of proteins that is characterized by the presence of eight conserved cysteine residues that form a cystine knot structure (4). Humans express multiple alternatively spliced isoforms of VEGF that are 111, 121, 145, 165, 183, 189, and 206 amino acids (aa) in length (4, 5, 6). Isoforms other than VEGF<sub>111</sub> and 121 contain basic heparin binding regions and are not freely diffusible (4, 5). VEGF<sub>165</sub> appears to be the most abundant and potent isoform, followed by VEGF<sub>121</sub> and VEGF<sub>189</sub> (3-5). VEGF<sub>145</sub> expression is relatively low, and is found mainly in female and male reproductive tissues, carcinomas of the female reproductive system, and other solid tumors (5, 7, 8). Expression of mouse VEGF<sub>144</sub>, the rodent ortholog to human VEGF<sub>145</sub>, is particularly enhanced by metabolic stress, such as that encountered during sustained exercise or starvation (8, 9). Human VEGF<sub>145</sub> shares 89% aa sequence identity with mouse and rat VEGF<sub>144</sub>. VEGF<sub>145</sub> binds to the type I transmembrane receptor tyrosine kinase VEGF R2/KDR/Flk1 on endothelial cells. This receptor appears to be the primary mediator of VEGF angiogenic activity (3-5, 7). Unlike VEGF<sub>165</sub>, VEGF<sub>145</sub> does not bind VEGF R1/Flt1 or VEGF R3/Flt-4 (4, 5, 7). VEGF<sub>145</sub> also binds the semaphorin receptor Neuropilin2 and promotes complex formation with VEGF R2, but unlike VEGF<sub>165</sub> and VEGF<sub>121</sub>, it does not bind Neuropilin1 (10, 11). VEGF is required during embryogenesis to regulate the proliferation, migration, and survival of endothelial cells (3, 4, 7). In adults, VEGF acts mainly during wound healing and the female reproductive cycle (3). Pathologically, isoforms that include VEGF<sub>145</sub> are involved in tumor angiogenesis and vascular leakage (5, 12, 13).

**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. Woolard, J. *et al.* (2009) Microcirculation **16**:572.
6. Mineur, P. *et al.* (2007) J. Cell Biol. **179**:1261.
7. Poltorak, Z. *et al.* (1997) J. Biol. Chem. **272**:7151.
8. Zhang, L. *et al.* (2002) Biochem. Biophys. Res. Commun. **292**:860.
9. Ding, Y.H. *et al.* (2004) Curr. Neurovasc. Res. **1**:411.
10. Pan, Q. *et al.* (2007) J. Biol. Chem. **282**:24049.
11. Gluzman-Poltorak, Z. *et al.* (2000) J. Biol. Chem. **275**:18040.
12. Weis, S.M. and D.A. Cheresh (2005) Nature **437**:497.
13. Thurston, G. (2002) J. Anat. **200**:575.