

**DESCRIPTION**

<b>Source</b>	<i>Spodoptera frugiperda</i> , Sf 21 (baculovirus)-derived Ala27-Arg191 Accession # NP_001165097
<b>Structure / Form</b>	Disulfide-linked homodimer; Biotinylated protein via sugars
<b>Predicted Molecular Mass</b>	19 kDa

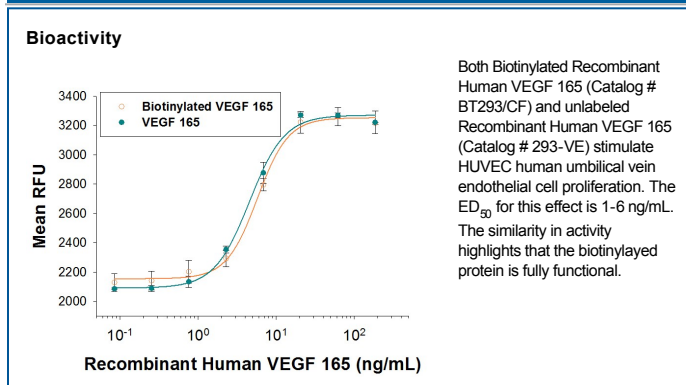
**SPECIFICATIONS**

<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 1-6 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 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>

**DATA**



**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, 2). It is a member of the PDGF family that is characterized by the presence of eight conserved cysteine residues and a cystine knot structure (3). Humans express two sets of alternatively spliced isoforms of 121, 145, 165, 183, 189, and 206 amino acids (aa) in length (3, 4). Isoforms other than VEGF<sub>121</sub> contain basic heparin-binding regions and are not freely diffusible (3, 4). VEGF<sub>165</sub> appears to be the most abundant and potent of the angiogenic isoform set, followed by VEGF<sub>121</sub> and VEGF<sub>189</sub> (3, 5). The anti-angiogenic or "b" set of isoforms is differentially spliced to contain five alternative amino acids at the C-terminus, and are the more highly expressed isoforms in normal adult tissue (6). VEGF<sub>165b</sub>, like VEGF<sub>121</sub> but unlike most angiogenic isoforms, does not bind heparins and is therefore diffusible (3). 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<sub>165</sub>, respectively. In addition to alternatively spliced VEGF isoforms, multiple fragments of VEGF can be generated by extracellular proteolysis (4). VEGFs bind the type I transmembrane receptor tyrosine kinases VEGF R1 (also called Flt-1) and VEGF R2 (Flk-1/KDR) on endothelial cells (3). Although VEGF affinity is highest for binding to VEGF R1, VEGF R2 appears to be the primary mediator of VEGF angiogenic activity (3, 5). VEGF<sub>165</sub> binds the semaphorin receptor, Neuropilin-1 and promotes complex formation with VEGF R2 (7). VEGF is required during embryogenesis to regulate the proliferation, migration, and survival of endothelial cells (3, 5). In adults, VEGF functions mainly in wound healing and the female reproductive cycle (5). Pathologically, it is involved in tumor development and tumor vascular leakage (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- $\alpha$  (5, 10).

**References:**

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