

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived	
	Human VEGF-A (Ala27-Arg191) Accession # NP_001303939	SAGQEEGASLRVSGTRSLTRKD
	N-terminus	C-terminus
N-terminal Sequence Analysis	Ala27	
Structure / Form	Disulfide-linked homodimer	
Predicted Molecular Mass	21 kDa	

SPECIFICATIONS

SDS-PAGE	22-29 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human VEGF R2/KDR/Fik-1 Fc Chimera (Catalog # 357-KD) is immobilized at 0.5 µg/mL (100 µL/well), the concentration of Recombinant Human VEGF-Ax that produces 50% of the optimal binding response is approximately 1-6 ng/mL. Recombinant Human VEGF-Ax weakly binds to Recombinant Human Neuropilin-1 (Catalog # 3870-N1).
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 250 µg/mL in 4 mM HCl.
Shipping	The product is shipped at ambient temperature. 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.

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 VEGF₁₂₁ contain basic heparin-binding regions and are not freely diffusible (3, 4). VEGF₁₆₅ appears to be the most abundant and potent of the angiogenic isoform set, followed by VEGF₁₂₁ and VEGF₁₈₉ (3, 5). The anti-angiogenic or "b" set of isoforms is differentially spliced to contain six alternative amino acids at the C-terminus, and are the more highly expressed isoforms in normal adult tissue (6). VEGF_{165b}, like VEGF₁₂₁ but unlike most angiogenic isoforms, does not bind heparins and is therefore diffusible (3). 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 VEGF₁₆₅, 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 (Fik-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). VEGF₁₆₅ binds the semaphorin receptor, Neuropilin-1 and promotes complex formation with VEGF R2 (7). An extended form of VEGF-A, known as VEGF-Ax, results from the C-terminal addition of 22 amino acids (8). VEGF-Ax is produced by vascular endothelial cells, retains the ability to bind VEGF R2 but not Neuropilin-1, and functions as an anti-angiogenic protein (8). 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 (9). Circulating VEGF levels correlate with disease activity in autoimmune diseases such as rheumatoid arthritis, multiple sclerosis, and systemic lupus erythematosus (10). VEGF is induced by hypoxia and cytokines such as IL-1, IL-6, IL-8, Oncostatin M, and TNF-α (5, 11).

References:

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