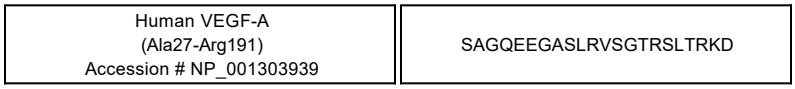
Recombinant Human VEGF-Ax

Catalog Number: 9018-VE

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived



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/Flk-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 with BSA as a carrier protein. 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.** ● 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 $VEGF_{121}$ contain basic heparin-binding regions and are not freely diffusible (3, 4). $VEGF_{165}$ appears to be the most abundant and potent of the angiogenic isoform set, followed by $VEGF_{121}$ and $VEGF_{189}$ (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 $VEGF_{121}$ but unlike most angiogenic isoforms, does not bind heparins and is therefore diffusible (3). Human $VEGF_{165}$ 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 $VEGF_{165}$, 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). $VEGF_{165}$ 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:

1. Leung, D.W. *et al.* (1989) Science **246**:1306.
2. Keck, P.J. *et al.* (1989) Science **246**:1309.
3. Robinson, C.J. and S.E. Stringer (2001) J. Cell. Sci. **114**:853.
4. Vempati, P. *et al.* (2014) Cytokine Growth Factor Rev. **25**:1.
5. Byrne, A.M. *et al.* (2005) J. Cell. Mol. Med. **9**:777.
6. Nowak, D.G. *et al.* (2008) J. Cell Sci. **121**:3487.
7. Pan, Q. *et al.* (2007) J. Biol. Chem. **282**:24049.
8. Eswarappa, S.M. *et al.* (2014) Cell **157**:1605.
9. Goel, H.L. and A.M. Mercurio (2013) Nat. Rev. Cancer **13**:871.
10. Carvalho, J.F. *et al.* (2007) J. Clin. Immunol. **27**:246.
11. Angelo, L.S. and R. Kurzrock (2007) Clin. Cancer Res. **13**:2825.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449