Human Vascular Endothelial Growth Factor-165 (hVEGF\textsubscript{165})

**Source:** Recombinant human VEGF\textsubscript{165} (hVEGF\textsubscript{165}) Ala207-Arg371 (Accession #NP_001020539) was expressed in human 293 cells at Cell Signaling Technology.

**Molecular Characterization:** Recombinant hVEGF\textsubscript{165} contains no “tags” and has a calculated MW of 19,165. DTT-reduced protein migrates as a 24 kDa polypeptide and the non-reduced cystine-linked homodimer migrates as a 40 kDa protein. The expected amino-terminal APMAE of recombinant non-reduced cystine-linked homodimer migrates as a 24 kDa polypeptide and the contains no “tags” and has a calculated MW of 19,165. DTT-reduced human 293 cells at Cell Signaling Technology.

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**Purity:** >98% as determined by SDS-PAGE of 6 µg reduced (+) and non-reduced (-) recombinant hVEGF\textsubscript{165}. All lots are greater than 98% pure.

**Endotoxin:** Less than 0.01 ng endotoxin/1 µg hVEGF\textsubscript{165}.

**Bioactivity:** The bioactivity of recombinant hVEGF\textsubscript{165} was determined in a cell proliferation assay using HUVEC. The ED\textsubscript{50} of each lot is between 1-6 ng/ml.

**Formulation:** With carrier: Lyophilized from a 0.22 µm filtered solution of PBS, pH 7.2 containing 20 µg BSA per 1 µg hVEGF\textsubscript{165}.

**Reconstitution:** With carrier: Add sterile PBS or PBS containing 1% bovine or human serum albumin or 5-10% FBS to a final hVEGF\textsubscript{165} concentration of greater than 50 µg/ml. Solubilize for 30 minutes at room temperature with occasional gentle vortexing.

**Storage:** Stable in lyophilized state at 4°C for 1 year after receipt. Sterile stock solutions reconstituted with carrier protein are stable at 4°C for 2 months and at -20°C for 6 months. Avoid repeated freeze-thaw cycles.

**Applications:** Optimal concentration for the desired application should be determined by the user.

**Background References:**

**Background:** VEGF\textsubscript{165} is the most abundant splice variant of VEGF-A (1,2). VEGF\textsubscript{165} is produced by a number of cells including endothelial cells, macrophages and T cells. VEGF\textsubscript{165} is involved in angiogenesis, vascular endothelial cell survival, growth, migration and vascular permeability (1). VEGF gene expression is induced by hypoxia, inflammatory cytokines and oncogenes (1,2). VEGF\textsubscript{165} binds to heparan sulfate and is retained on the cell surface and in the extracellular matrix (2,3). VEGF\textsubscript{165} binds to the receptor tyrosine kinases, VEGFR1 and VEGFR2 (1). VEGF\textsubscript{165} is the most abundant splice variant of VEGF-A (1,2). VEGF\textsubscript{165} is produced by a number of cells including endothelial cells, macrophages and T cells. VEGF\textsubscript{165} is involved in angiogenesis, vascular endothelial cell survival, growth, migration and vascular permeability (1). VEGF gene expression is induced by hypoxia, inflammatory cytokines and oncogenes (1,2). VEGF\textsubscript{165} binds to heparan sulfate and is retained on the cell surface and in the extracellular matrix (2,3). VEGF\textsubscript{165} binds to the receptor tyrosine kinases, VEGFR1 and VEGFR2 (1). VEGF\textsubscript{165} is the only splice variant that binds to co-receptors NRP-1 and NRP-2 (1-3) that function to enhance VEGFR2 signaling (1). Binding of VEGF\textsubscript{165} to VEGFR1 and VEGFR2 leads to activation of the PI3K/AKT, p38 MAPK, FAK and paxillin (1). VEGF plays a key role in tumor angiogenesis in many cancers (2).

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