ATAGENIX LABORATORIES

Catalog Number:ATEP00059HU

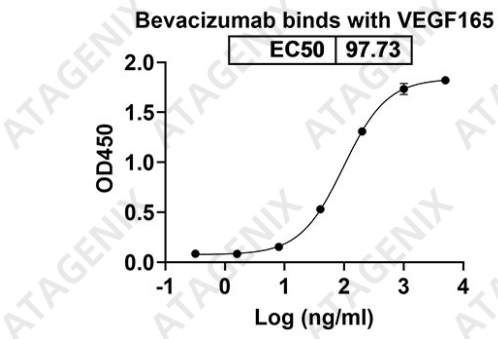
Recombinant Human VEGFA/VEGF protein ,N- MAT Tag

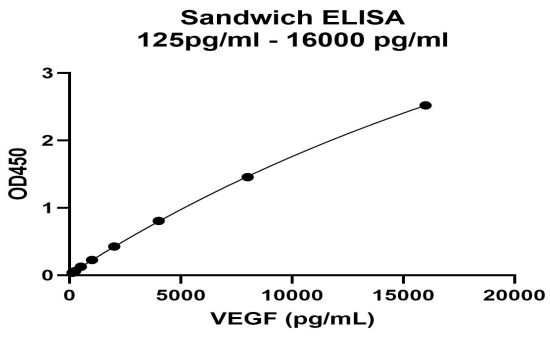
Product Details

Summary

English name	Recombinant Human VEGFA/VEGF protein ,N- MAT Tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	Please contact with the lab for this information.
Construction	A DNA sequence encoding the human VEGFA(Ala27~Arg136) was fused with the N-terminal MAT Tag
Accession #	P15692
Host	E.coli
Species	Homo sapiens (Human)
Predicted Molecular Mass	14.65kDa
Formulation	Supplied as solution form in PBS, pH 7.5 8M urea or lyophilized from PBS, pH 7.5 8M urea.
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution.A copy of datasheet will be provided with the products, please refer to it for details.

Bioactivity





Background

Background	VEGF165 is the most abundant splice variant of VEGF-A. VEGF165 is produced

by a number of cells including endothelial cells, macrophages and T cells. VEGF165 is involved in angiogenesis, vascular endothelial cell survival, growth, migration and vascular permeability. VEGF gene expression is induced by hypoxia, inflammatory cytokines and oncogenes. VEGF165 binds to heparan sulfate and is retained on the cell surface and in the extracellular matrix. VEGF165 binds to the receptor tyrosine kinases, VEGFR1 and VEGFR2. VEGF165 is the only splice variant that binds to co-receptors NRP-1 and NRP-2 that function to enhance VEGFR2 signaling. Binding of VEGF165 to VEGFR1 and VEGFR2 leads to activation of the PI3K/AKT, p38 MAPK, FAK and paxillin. VEGF plays a key role in tumor angiogenesis in many cancers.

Alternative Names RP1-261G23.1,MGC70609,MVCD1,VEGFA,VPF

References Vanderstraeten, Baselet, Buset, Ben Said, de Ville de Goyet, Many, Gérard, Derradji (2020) Modulation of VEGF Expression and Oxidative Stress Response by Iodine Deficiency in Irradiated Cancerous and Non-Cancerous Breast Cells International journal of molecular sciences 21(11)

Frontier progress

Breast cancer remains a major concern and its physiopathology is influenced by iodine deficiency (ID) and radiation exposure. Since radiation and ID can separately induce oxidative stress (OS) and microvascular responses in breast, their combination could additively increase these responses. Therefore, ID was induced in MCF7 and MCF12A breast cell lines by medium change. Cells were then X-irradiated with doses of 0.05, 0.1, or 3 Gy. In MCF12A cells, both ID and radiation (0.1 and 3 Gy) increased OS and vascular endothelial growth factor (VEGF) expression, with an additive effect when the highest dose was combined with ID. However, in MCF7 cells no additive effect was observed. VEGF mRNA up-regulation was reactive oxygen species (ROS)-dependent, involving radiation-induced mitochondrial ROS. Results on total VEGF mRNA hold true for the pro-angiogenic isoform VEGF165 mRNA, but the treatments did not modulate the anti-angiogenic isoform VEGF165b. Radiation-induced antioxidant response was differentially regulated upon ID in both cell lines. Thus, radiation response is modulated according to iodine status and cell type and can lead to additive effects on ROS and VEGF. As these are often involved in cancer initiation and progression, we believe that iodine status should be taken into account in radiation prevention policies.