

Product datasheet for TP723469

Vegfa (NM_001025250) Mouse Recombinant Protein

Product data:

Product Type: Recombinant Proteins Description: Purified recombinant protein of Mouse vascular endothelial growth factor A (Vegfa), transcript variant 1. Species: Mouse **Expression Host:** E. coli **Expression cDNA Clone** MAPTTEGEQK SHEVIKFMDV YQRSYCRPIE TLVDIFQEYP DEIEYIFKPS CVPLMRCAGC CNDEALECVP or AA Sequence: TSESNITMQI MRIKPHQSQH IGEMSFLQHS RCECRPKKDR TKPEKHCEPC SERRKHLFVQ DPQTCKCSCK NTDSRCKARQ LELNERTCRC DKPRR Tag: Tag Free Predicted MW: 39 kDa **Concentration:** lot specific **Purity:** >95% as determined by SDS-PAGE and Coomassie blue staining **Buffer:** Lyophilized from a 0.2 µM filtered solution of 20mM phosphate buffer,100mM NaCl, pH 7.2 Determined by the dose-dependent stimulation of the proliferation of human umbilical vein **Bioactivity:** endothelial cells (HUVEC) using a concentration range of 1.0-5.0 ng/ml. Endotoxin: Endotoxin level is < 0.1 ng/ μ g of protein (< 1 EU/ μ g) Storage: Store at -80°C. Stable for at least 6 months from date of receipt under proper storage and handling Stability: conditions. NP 001103736 RefSeq: Locus ID: 22339 Q00731, A0A1L1SVG2 UniProt ID: 3547 RefSeq Size: 17 22.79 cM Cytogenetics: **RefSeq ORF:** 1176 Synonyms: V; Veg; Vegf; VEGF12; VEGF16; VEGF18; Vpf



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Summary:	This gene is a member of the PDGF/VEGF growth factor family. It encodes a heparin-binding protein, which exists as a disulfide-linked homodimer. This growth factor induces proliferation and migration of vascular endothelial cells, and is essential for both physiological and pathological angiogenesis. Disruption of this gene in mice resulted in abnormal embryonic blood vessel formation. This gene is upregulated in many known tumors and its expression is correlated with tumor stage and progression. Alternatively spliced transcript variants encoding different isoforms have been found for this gene. There is also evidence for alternative translation initiation from upstream non-AUG (CUG) codons resulting

evidence for alternative translation initiation from upstream non-AUG (CUG) codons resulting in additional isoforms. A recent study showed that a C-terminally extended isoform is produced by use of an alternative in-frame translation termination codon via a stop codon readthrough mechanism, and that this isoform is antiangiogenic. Expression of some isoforms derived from the AUG start codon is regulated by a small upstream open reading frame, which is located within an internal ribosome entry site.[provided by RefSeq, Nov 2015]

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