

Product datasheet for DA3518XC

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VEGF-A (Isoform 164) Mouse Protein

Product data:

Product Type: Recombinant Proteins

Description: VEGF-A (Isoform 164) mouse recombinant protein, 20 μg

Species: Mouse
Expression Host: Insect
Predicted MW: 48 kDa

Purity: >95% > 95%, by SDS-PAGE and visualised by silver stain.

Buffer: Presentation State: Purified

State: Lyophilized without stabilizer.

Bioactivity: Biological: The ED50 for stimulation of 3H-thymidine incorporation and cell proliferation by

human umbilical vein endothelial cells for VEGF164 has been determined to be in the range

of

1-2 ng/ml.

Specific: 1 x 10e6 units/mg

Endotoxin: < 0.1 ng/µg of VEGF

Reconstitution Method: Restore in PBS or medium containing at least 0.1% human or bovine serum albumin to a

concentration not lower than

50 μg/ml.

Preparation: Lyophilized without stabilizer.

Protein Description: Recombinant Murine VEGF164 is produced as a homodimer consisting of 164 amino acid

residues.

Storage: Store VEGF164 Lyophilized at -20°C to -70°C.

Reconstituted VEGF164 should be stored in working aliquots at -20°C.

Avoid repeated freeze-thaw cycles!

Stability: Shelf life: One year from despatch.

RefSeg: NP 001020421

Locus ID: 22339

UniProt ID: Q00731, A0A1L1SVG2

Cytogenetics: 17 22.79 cM





VEGF-A (Isoform 164) Mouse Protein - DA3518XC

Synonyms:

V; Veg; Vegf; VEGF12; VEGF16; VEGF18; Vpf

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 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]