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Product datasheet for AR31053PU-S

VEGF-A (VEGF120) Rat Protein

Product data:

Product Type:	Recombinant Proteins
Description:	VEGF-A (VEGF120) rat recombinant protein, 2 µg
Species:	Rat
Expression Host:	E. coli
Expression cDNA Clone or AA Sequence:	APTTEGEQKA HEVVKFMDVY QRSYCRPIET LVDIFQEYPD EIEYIFKPSC VPLMRCAGCC NDEALECVPT SESNVTMQIM RIKPHQSQHI GEMSFLQHSR CECRPKKDRT KPEKCDKPRR Result by N-terminal sequencing: APTTEGEQKA H
Predicted MW:	14.02 kDa
Purity:	>95%
Buffer:	Presentation State: Purified State: Lyophilized freeze dried protein Buffer System: PBS without stabilizer
Bioactivity:	Biological: Determined by the dose-dependent stimulation of the proliferation of human umbilical vein endothelial cells (HUVEC) using a concentration range of 2-10 ng/ml.
Endotoxin:	< 0.1 ng/µg of Rat VEGF120
Reconstitution Method:	The lyophilized VEGF120 should be restored in ddH2O to a concentration not lower than 50 µg/ml.
Preparation:	Lyophilized freeze dried protein
Protein Description:	Recombinant Rat Vascular Endothelial Growth Factor 120
Storage:	Lyophilized samples are stable for greater than six months at –20°C to –70°C. Reconstituted VEGF120 should be stored in working aliquots at -20°C. Avoid repeated freeze-thaw cycles!
RefSeq:	NP_001103803
Locus ID:	83785
UniProt ID:	B5DEK7
Cytogenetics:	9q12
Synonyms:	Vegf; VEGF-A; VEGF111; VEGF164; VPF

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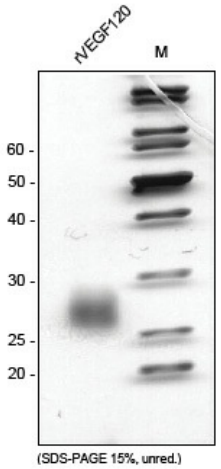
Summary:

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 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]

Product images:



(SDS-PAGE 15%, unred.)