

# Product datasheet for RR200262L3V

## Vegfa (NM\_031836) Rat Tagged ORF Clone Lentiviral Particle

## **Product data:**

#### **Product Type: Lentiviral Particles Product Name:** Vegfa (NM\_031836) Rat Tagged ORF Clone Lentiviral Particle Symbol: Vegfa Vegf; VEGF-A; VEGF111; VEGF164; VPF Synonyms: **Mammalian Cell** Puromycin Selection: Vector: pLenti-C-Myc-DDK-P2A-Puro (PS100092) Myc-DDK Tag: NM 031836 ACCN: ORF Size: 1179 bp The ORF insert of this clone is exactly the same as(RR200262). **ORF** Nucleotide Sequence: **OTI Disclaimer:** The molecular sequence of this clone aligns with the gene accession number as a point of reference only. However, individual transcript sequences of the same gene can differ through naturally occurring variations (e.g. polymorphisms), each with its own valid existence. This clone is substantially in agreement with the reference, but a complete review of all prevailing variants is recommended prior to use. More info **OTI** Annotation: This clone was engineered to express the complete ORF with an expression tag. Expression varies depending on the nature of the gene. **RefSeq:** NM 031836.3, NP 114024.2 **RefSeq Size:** 3546 bp **RefSeq ORF:** 1182 bp Locus ID: 83785 **UniProt ID:** P16612 Cytogenetics: 9q12



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### **GRIGENE** Vegfa (NM\_031836) Rat Tagged ORF Clone Lentiviral Particle – RR200262L3V

Gene 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]

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