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Product datasheet for RC210319L1V

PHD3 (EGLN3) (NM_022073) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	PHD3 (EGLN3) (NM_022073) Human Tagged ORF Clone Lentiviral Particle
Symbol:	PHD3
Synonyms:	HIFP4H3; HIFPH3; PHD3
Mammalian Cell Selection:	None
Vector:	pLenti-C-Myc-DDK (PS100064)
Tag:	Myc-DDK
ACCN:	NM_022073
ORF Size:	717 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC210319).
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_022073.3
RefSeq Size:	2722 bp
RefSeq ORF:	720 bp
Locus ID:	112399
UniProt ID:	Q9H6Z9
Cytogenetics:	14q13.1
Domains:	2OG-FeII_Oxy, P4Hc
Protein Families:	Druggable Genome

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Protein Pathways: Pathways in cancer, Renal cell carcinoma

MW: 27.3 kDa

Gene Summary: Cellular oxygen sensor that catalyzes, under normoxic conditions, the post-translational formation of 4-hydroxyproline in hypoxia-inducible factor (HIF) alpha proteins. Hydroxylates a specific proline found in each of the oxygen-dependent degradation (ODD) domains (N-terminal, NODD, and C-terminal, CODD) of HIF1A. Also hydroxylates HIF2A. Has a preference for the CODD site for both HIF1A and HIF2A. Hydroxylation on the NODD site by EGLN3 appears to require prior hydroxylation on the CODD site. Hydroxylated HIFs are then targeted for proteasomal degradation via the von Hippel-Lindau ubiquitination complex. Under hypoxic conditions, the hydroxylation reaction is attenuated allowing HIFs to escape degradation resulting in their translocation to the nucleus, heterodimerization with HIF1B, and increased expression of hypoxy-inducible genes. EGLN3 is the most important isozyme in limiting physiological activation of HIFs (particularly HIF2A) in hypoxia. Also hydroxylates PKM in hypoxia, limiting glycolysis. Under normoxia, hydroxylates and regulates the stability of ADRB2. Regulator of cardiomyocyte and neuronal apoptosis. In cardiomyocytes, inhibits the anti-apoptotic effect of BCL2 by disrupting the BAX-BCL2 complex. In neurons, has a NGF-induced proapoptotic effect, probably through regulating CASP3 activity. Also essential for hypoxic regulation of neutrophilic inflammation. Plays a crucial role in DNA damage response (DDR) by hydroxylating TELO2, promoting its interaction with ATR which is required for activation of the ATR/CHK1/p53 pathway. Target proteins are preferentially recognized via a LXXLAP motif.[UniProtKB/Swiss-Prot Function]