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PHD3 Rabbit pAb

Catalog # AP53547

Product Information

Primary Accession	Q9H6Z9
Host	Rabbit
Clonality	Polyclonal Antibody
Calculated MW	27261

Additional Information

Gene ID	112399
Other Names	PHD3; HIFPH3; HIFP4H3
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

Protein Information

Name	EGLN3 {ECO:0000303|PubMed:16098468, ECO:0000312|HGNC:HGNC:14661}
Function	Prolyl hydroxylase that mediates hydroxylation of proline residues in target proteins, such as PKM, TELO2, ATF4, GPX4 and HIF1A (PubMed:19584355, PubMed:20978507, PubMed:21483450, PubMed:21575608, PubMed:21620138, PubMed:22797300, PubMed:40281343). Target proteins are preferentially recognized via a LXXLAP motif. Cellular oxygen sensor that catalyzes, under normoxic conditions, the post-translational formation of 4-hydroxyproline in hypoxia-inducible factor (HIF) alpha proteins (PubMed:11595184, PubMed:12181324). Hydroxylates a specific proline found in each of the oxygen-dependent degradation (ODD) domains (N-terminal, NODD, and C-terminal, CODD) of HIF1A (PubMed:11595184, PubMed:12181324). Also hydroxylates HIF2A (PubMed:11595184, PubMed:12181324). Has a preference for the CODD site for both HIF1A and HIF2A (PubMed:11595184, PubMed:12181324). Hydroxylation on the NODD site by EGLN3 appears to require prior hydroxylation on the CODD site (PubMed:11595184, PubMed:12181324). Hydroxylated HIFs are then targeted for proteasomal degradation via the von Hippel-Lindau ubiquitination complex (PubMed:11595184, PubMed:12181324). 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 (PubMed:11595184, PubMed:12181324). Acts as an inhibitor of ferroptosis by mediating hydroxylation of GPX4, thereby preventing GPX4 degradation via chaperone-mediated autophagy (PubMed:40281343). GPX4 hydroxylation is promoted by PSAT1, which provides 2-oxoglutarate substrate to EGLN3

(PubMed:40281343). ELGN3 is the most important isozyme in limiting physiological activation of HIFs (particularly HIF2A) in hypoxia. Also hydroxylates PKM in hypoxia, limiting glycolysis (PubMed:21483450, PubMed:21620138). Under normoxia, hydroxylates and regulates the stability of ADRB2 (PubMed:19584355). Regulator of cardiomyocyte and neuronal apoptosis. In cardiomyocytes, inhibits the anti-apoptotic effect of BCL2 by disrupting the BAX-BCL2 complex (PubMed:20849813). In neurons, has a NGF-induced proapoptotic effect, probably through regulating CASP3 activity (PubMed:16098468). Also essential for hypoxic regulation of neutrophilic inflammation (PubMed:21317538). 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 (PubMed:22797300). Also mediates hydroxylation of ATF4, leading to decreased protein stability of ATF4 (Probable).

Cellular Location

Nucleus. Cytoplasm Note=Colocalizes with WDR83 in the cytoplasm {ECO:0000250|UniProtKB:Q62630}

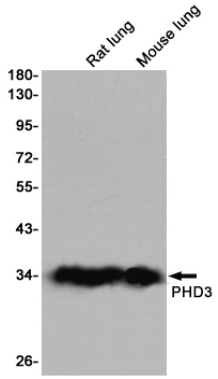
Tissue Location

Widely expressed at low levels. Expressed at higher levels in adult heart (cardiac myocytes, aortic endothelial cells and coronary artery smooth muscle), lung and placenta, and in fetal spleen, heart and skeletal muscle. Also expressed in pancreas. Localized to pancreatic acini and islet cells.

Background

Swiss-Prot Acc.Q9H6Z9.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.

Images



Western blot detection of PHD3 in Rat lung and Mouse lung lysates using PHD3 rabbit pAb (1:1000 diluted).Predicted band size:27kDa.Observed band size:27kDa.

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