

**Product Name:** PHD3 (11Y3) Rabbit Monoclonal Antibody  
**Catalog #:** AMRe16061

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## Summary

<b>Production Name</b>	PHD3 (11Y3) Rabbit Monoclonal Antibody
<b>Description</b>	Rabbit Monoclonal Antibody
<b>Host</b>	Rabbit
<b>Application</b>	WB,ELISA
<b>Reactivity</b>	Human,Mouse,Rat

## Performance

<b>Conjugation</b>	Unconjugated
<b>Modification</b>	Unmodified
<b>Isotype</b>	IgG
<b>Clonality</b>	Monoclonal
<b>Form</b>	Liquid
<b>Storage</b>	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
<b>Buffer</b>	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% New type preservative N and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.
<b>Purification</b>	Affinity purification

## Immunogen

<b>Gene Name</b>	EGLN3 {ECO:0000303 PubMed:16098468, ECO:0000312 HGNC:HGNC:14661}
<b>Alternative Names</b>	Egl nine homolog 3; EGLN3; Factor responsive smooth muscle protein; HIF Prolyl Hydroxylase 3; HIFP4H3; HIFPH3; P4H3; PHD3; SM20;
<b>Gene ID</b>	112399.0
<b>SwissProt ID</b>	Q9H6Z9.

## Application

<b>Dilution Ratio</b>	WB 1:500-1:2000
<b>Molecular Weight</b>	27kDa

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## Background

Catalyzes the post-translational formation of 4-hydroxyproline in hypoxia-inducible factor (HIF) alpha proteins. Hydroxylates HIF-1 alpha at 'Pro-564', and HIF-2 alpha. Functions as a cellular oxygen sensor and, under normoxic conditions, targets HIF through the hydroxylation for proteasomal degradation via the von Hippel-Lindau ubiquitination complex. Prolyl hydroxylase that mediates hydroxylation of proline residues in target proteins, such as PKM, TELO2, ATF4 and HIF1A (PubMed:<a href="http://www.uniprot.org/citations/19584355" target="\_blank">19584355</a>, PubMed:<a href="http://www.uniprot.org/citations/21620138" target="\_blank">21620138</a>, PubMed:<a href="http://www.uniprot.org/citations/21483450" target="\_blank">21483450</a>, PubMed:<a href="http://www.uniprot.org/citations/22797300" target="\_blank">22797300</a>, PubMed:<a href="http://www.uniprot.org/citations/20978507" target="\_blank">20978507</a>, PubMed:<a href="http://www.uniprot.org/citations/21575608" target="\_blank">21575608</a>). 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:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). Hydroxylates a specific proline found in each of the oxygen-dependent degradation (ODD) domains (N-terminal, NODD, and C-terminal, CODD) of HIF1A (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). Also hydroxylates HIF2A (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). Has a preference for the CODD site for both HIF1A and HIF2A (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). Hydroxylation on the NODD site by EGLN3 appears to require prior hydroxylation on the CODD site (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). Hydroxylated HIFs are then targeted for proteasomal degradation via the von Hippel-Lindau ubiquitination complex (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). 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 hypoxo- inducible genes (PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>, PubMed:<a href="http://www.uniprot.org/citations/12181324" target="\_blank">12181324</a>). EGLN3 is the most important isozyme in limiting physiological activation of HIFs (particularly HIF2A) in hypoxia. Also hydroxylates PKM in hypoxia, limiting glycolysis (PubMed:<a href="http://www.uniprot.org/citations/21620138" target="\_blank">21620138</a>, PubMed:<a href="http://www.uniprot.org/citations/11595184" target="\_blank">11595184</a>).

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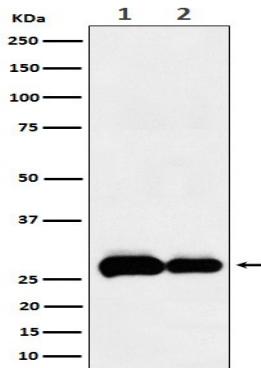
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Under normoxia, hydroxylates and regulates the stability of ADRB2 (PubMed: [21483450](http://www.uniprot.org/citations/21483450)). Regulator of cardiomyocyte and neuronal apoptosis. In cardiomyocytes, inhibits the anti-apoptotic effect of BCL2 by disrupting the BAX-BCL2 complex (PubMed: [19584355](http://www.uniprot.org/citations/19584355)). Regulator of cardiomyocyte and neuronal apoptosis. In neurons, has a NGF-induced proapoptotic effect, probably through regulating CASP3 activity (PubMed: [20849813](http://www.uniprot.org/citations/20849813)). Also essential for hypoxic regulation of neutrophilic inflammation (PubMed: [16098468](http://www.uniprot.org/citations/16098468)). 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: [21317538](http://www.uniprot.org/citations/21317538)). Also mediates hydroxylation of ATF4, leading to decreased protein stability of ATF4 (Probable).

## Research Area

### Image Data



Western blot analysis of PHD3 expression in (1) A549 cell lysate; (2) NIH/3T3 cell lysate.

### Note

For research use only.