

EGLN3 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14221B**Specification**

EGLN3 Antibody (C-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9H6Z9
Other Accession	O62630 , O91UZ4 , NP_071356.1
Reactivity	Human
Predicted	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	27261
Antigen Region	203-231

EGLN3 Antibody (C-term) - Additional Information**Gene ID** 112399**Other Names**

Egl nine homolog 3, HPH-1, Hypoxia-inducible factor prolyl hydroxylase 3, HIF-PH3, HIF-prolyl hydroxylase 3, HPH-3, Prolyl hydroxylase domain-containing protein 3, PHD3, EGLN3

Target/Specificity

This EGLN3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 203-231 amino acids from the C-terminal region of human EGLN3.

Dilution

WB~~1:1000
IHC-P~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

EGLN3 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

EGLN3 Antibody (C-term) - 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 and HIF1A (PubMed:[19584355](#), PubMed:[21620138](#), PubMed:[21483450](#), PubMed:[22797300](#), PubMed:[20978507](#), PubMed:[21575608](#)). 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 hypoxia-inducible genes (PubMed:[11595184](#), PubMed:[12181324](#)). ELGN3 is the most important isozyme in limiting physiological activation of HIFs (particularly HIF2A) in hypoxia. Also hydroxylates PKM in hypoxia, limiting glycolysis (PubMed:[21620138](#), PubMed:[21483450](#)). 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

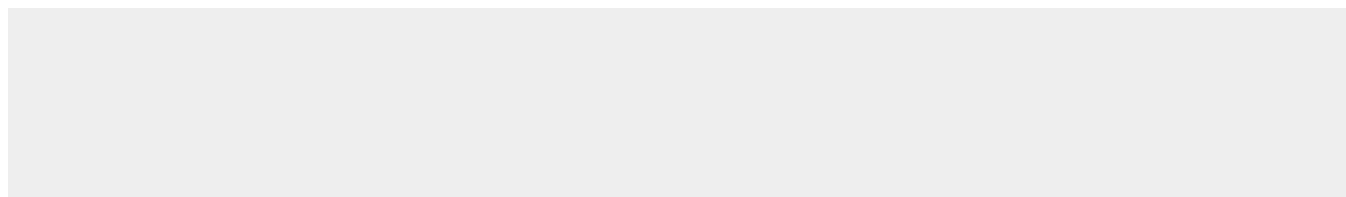
Widely expressed at low levels. Expressed at higher levels in adult heart (cardiac myocytes, aortic endothelial cells and coronary artery smooth muscle), lung and placenta, and in fetal spleen, heart and skeletal muscle. Also expressed in pancreas. Localized to pancreatic acini and islet cells.

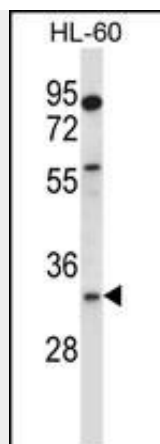
EGLN3 Antibody (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

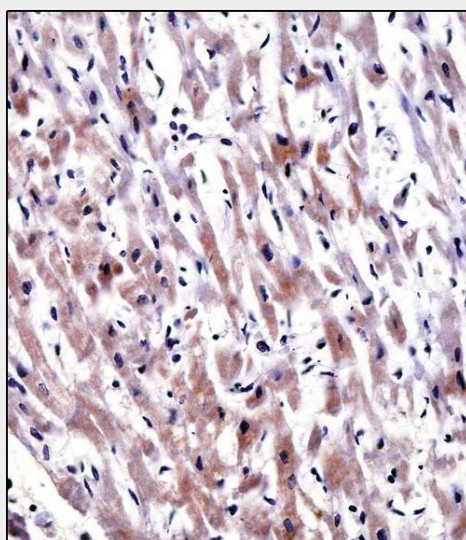
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

EGLN3 Antibody (C-term) - Images





EGLN3 Antibody (C-term) (Cat. #AP14221b) western blot analysis in HL-60 cell line lysates (35ug/lane). This demonstrates the EGLN3 antibody detected the EGLN3 protein (arrow).



EGLN3 Antibody (C-term) (AP14221b) immunohistochemistry analysis in formalin fixed and paraffin embedded human heart tissue followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of EGLN3 Antibody (C-term) for immunohistochemistry. Clinical relevance has not been evaluated.

EGLN3 Antibody (C-term) - Background

EGLN3 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. May play a role in cell growth regulation in muscle cells and in apoptosis in neuronal tissue. Promotes cell death through a caspase-dependent mechanism (By similarity).

EGLN3 Antibody (C-term) - References

- Sato, M., et al. Exp. Cell Res. 316(17):2871-2882(2010)
- Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
- Xue, J., et al. Gastroenterology 138(2):606-615(2010)
- Henze, A.T., et al. Cancer Res. 70(1):357-366(2010)
- Hatzimichael, E., et al. Eur. J. Haematol. 84(1):47-51(2010)