

EGLN1 Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP18620b**Specification**

EGLN1 Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [Q9GZT9](#)**EGLN1 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 54583**Other Names**

Egl nine homolog 1, Hypoxia-inducible factor prolyl hydroxylase 2, HIF-PH2, HIF-prolyl hydroxylase 2, HPH-2, Prolyl hydroxylase domain-containing protein 2, PHD2, SM-20, EGLN1 (http://www.genenames.org/cgi-bin/gene_symbol_report?hgnc_id=1232)>HGNC:1232), C1orf12

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

EGLN1 Antibody (C-term) Blocking Peptide - Protein Information**Name** EGLN1 ([HGNC:1232](#))**Synonyms** C1orf12**Function**

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 HIF1B. 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 hypoxia-inducible genes. EGLN1 is the most important isozyme under normoxia and, through regulating the stability of HIF1, involved in various hypoxia-influenced processes such as angiogenesis in retinal and cardiac functionality. Target proteins are preferentially recognized via a LXXLAP motif.

Cellular Location

Cytoplasm. Nucleus. Note=Mainly cytoplasmic. Shuttles between the nucleus and cytoplasm (PubMed:19631610). Nuclear export requires functional XPO1.

Tissue Location

According to PubMed:11056053, widely expressed with highest levels in skeletal muscle and heart, moderate levels in pancreas, brain (dopaminergic neurons of adult and fetal substantia nigra) and kidney, and lower levels in lung and liver. According to PubMed:12351678 widely expressed with highest levels in brain, kidney and adrenal gland. Expressed in cardiac myocytes, aortic endothelial cells and coronary artery smooth muscle. According to PubMed:12788921; expressed in adult and fetal heart, brain, liver, lung, skeletal muscle and kidney. Also expressed in placenta. Highest levels in adult heart, brain, lung and liver and fetal brain, heart spleen and skeletal muscle.

EGLN1 Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

EGLN1 Antibody (C-term) Blocking Peptide - Images

EGLN1 Antibody (C-term) Blocking Peptide - Background

The protein encoded by this gene catalyzes the post-translational formation of 4-hydroxyproline in hypoxia-inducible factor (HIF) α proteins. HIF is a transcriptional complex that plays a central role in mammalian oxygen homeostasis. This protein functions as a cellular oxygen sensor, and under normal oxygen concentration, modification by prolyl hydroxylation is a key regulatory event that targets HIF subunits for proteasomal destruction via the von Hippel-Lindau ubiquitylation complex. Mutations in this gene are associated with erythrocytosis familial type 3 (ECYT3).

EGLN1 Antibody (C-term) Blocking Peptide - References

Aggarwal, S., et al. Proc. Natl. Acad. Sci. U.S.A. 107(44):18961-18966(2010) Vogel, S., et al. J. Biol. Chem. 285(44):33756-33763(2010) Simonson, T.S., et al. Science 329(5987):72-75(2010) Thoms, B.L., et al. J. Biol. Chem. 285(27):20472-20480(2010) Spinella, F., et al. PLoS ONE 5 (6), E11241 (2010) :