

HIF1A Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP4776b**Specification**

HIF1A Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [Q16665](#)**HIF1A Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 3091**Other Names**

Hypoxia-inducible factor 1-alpha, HIF-1-alpha, HIF1-alpha, ARNT-interacting protein, Basic-helix-loop-helix-PAS protein MOP1, Class E basic helix-loop-helix protein 78, bHLHe78, Member of PAS protein 1, PAS domain-containing protein 8, HIF1A, BHLHE78, MOP1, PASD8

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.

HIF1A Antibody (C-term) Blocking Peptide - Protein Information**Name** HIF1A {ECO:0000303|PubMed:7539918, ECO:0000312|HGNC:HGNC:4910}**Function**

Functions as a master transcriptional regulator of the adaptive response to hypoxia (PubMed:11292861, PubMed:11566883, PubMed:15465032, PubMed:16973622, PubMed:17610843, PubMed:18658046, PubMed:20624928, PubMed:22009797, PubMed:9887100, PubMed:30125331). Under hypoxic conditions, activates the transcription of over 40 genes, including erythropoietin, glucose transporters, glycolytic enzymes, vascular endothelial growth factor, HILPDA, and other genes whose protein products increase oxygen delivery or facilitate metabolic adaptation to hypoxia (PubMed:11292861, PubMed:11566883).

PubMed:15465032,
PubMed:16973622,
PubMed:17610843,
PubMed:20624928,
PubMed:22009797,
PubMed:9887100,
PubMed:30125331).
Plays an essential role in embryonic vascularization, tumor angiogenesis and pathophysiology of
ischemic disease (PubMed:22009797). Heterodimerizes with ARNT; heterodimer binds to core DNA
sequence 5'-TACGTG-3' within the hypoxia response element (HRE) of target gene promoters (By
similarity). Activation requires recruitment of transcriptional coactivators such as CREBBP and
EP300 (PubMed:9887100),
PubMed:16543236). Activity is enhanced by interaction with NCOA1 and/or NCOA2
(PubMed:10594042).
Interaction with redox regulatory protein APEX1 seems to activate CTAD and potentiates activation
by NCOA1 and CREBBP (PubMed:10202154),
PubMed:10594042). Involved in the axonal distribution and transport of
mitochondria in neurons during hypoxia (PubMed:19528298).

Cellular Location

Cytoplasm. Nucleus. Nucleus speckle {ECO:0000250|UniProtKB:Q61221}. Note=Colocalizes with
HIF3A in the nucleus and speckles (By similarity). Cytoplasmic in normoxia, nuclear translocation
in response to hypoxia (PubMed:9822602) {ECO:0000250|UniProtKB:Q61221,
ECO:0000269|PubMed:9822602}

Tissue Location

Expressed in most tissues with highest levels in kidney and heart. Overexpressed in the majority
of common human cancers and their metastases, due to the presence of intratumoral hypoxia and
as a result of mutations in genes encoding oncoproteins and tumor suppressors. A higher level
expression seen in pituitary tumors as compared to the pituitary gland.

HIF1A Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

HIF1A Antibody (C-term) Blocking Peptide - Images

HIF1A Antibody (C-term) Blocking Peptide - Background

Hypoxia-inducible factor-1 (HIF1) is a transcription factor found in mammalian cells cultured under
reduced oxygen tension that plays an essential role in cellular and systemic homeostatic responses
to hypoxia. HIF1 is a heterodimer composed of an alpha subunit and a beta subunit. The beta
subunit has been identified as the aryl hydrocarbon receptor nuclear translocator (ARNT). This
protein encodes the alpha subunit of HIF-1. Overexpression of a natural antisense transcript (aHIF)
of this gene has been shown to be associated with nonpapillary renal carcinomas.

HIF1A Antibody (C-term) Blocking Peptide - References

Lee, M.N., et al. J. Natl. Cancer Inst. 102(6):426-442(2010)Mayer, A., et al. Adv. Exp. Med. Biol. 662,
399-405 (2010) Brouwer, E., et al. Clin. Exp. Rheumatol. 27(6):945-951(2009)Cho, H., et al. FEBS

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