Rabbit HIF1A Polyclonal Antibody | anti-HIF1A antibody

HIF1A Antibody

CATALOG #	HOST	REACTIVITY	APPLICATIONS
AAA321279	**Rabbit**	**Human, Mouse, Rat**	**ELISA, Immunocytochemistry, Immunofluorescence, Immunohistochemistry, Western Blot**

PRODUCT SYNONYMS

HIF1A; Polyclonal Antibody; HIF1A Antibody; ARNT interacting protein; ARNT-interacting protein; Basic helix loop helix PAS protein MOP1; Basic-helix-loop-helix-PAS protein MOP1; bHLHe78; Class E basic helix-loop-helix protein 78; HIF 1A; HIF 1alpha; HIF-1-alpha; HIF1 A; HIF1 Alpha; HIF1; HIF1-alpha; HIF1A_HUMAN; Hypoxia inducible factor 1 alpha; Hypoxia inducible factor 1 alpha isoform I.3; Hypoxia inducible factor 1 alpha subunit; Hypoxia inducible factor 1 alpha subunit basic helix loop helix transcription factor; Hypoxia inducible factor 1; alpha subunit (basic helix loop helix transcription factor); Hypoxia inducible factor1alpha; Hypoxia-inducible factor 1-alpha; Member of PAS protein 1; Member of PAS superfamily 1; Member of the PAS Superfamily 1; MOP 1; MOP1; PAS domain-containing protein 8; PASD 8; PASD8; anti-HIF1A antibody

PRODUCT SPECIFICATIONS

Host	Rabbit
Reactivity	Human, Mouse, Rat
Clonality	Polyclonal
Isotype	IgG
Specificity	HIF1a antibody detects endogenous levels of HIF1a.
Purity/Purification	The antiserum was purified by peptide affinity chromatography using SulfoLink Coupling Resin.
Form/Format	Liquid Phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Concentration	1mg/ml (varies by lot)
Sequence Length	850
Applicable Applications for anti-HIF1A antibody	ELISA, ICC (Immunocytochemistry), IF (Immunofluorescence), IHC (Immunohistochemistry), WB (Western Blot)
Immunogen	A synthesized peptide derived from human HIF1a.
Subcellular Location	Cytoplasm. Nucleus. Cytoplasmic in normoxia, nuclear translocation in response to hypoxia. Colocalizes with SUMO1 in the nucleus, under hypoxia.
Tissue Specificity	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.
Predicted Cross Reactivity	**Pig, Bovine, Horse, Rabbit**
Similarity	Pig (100%), Bovine (100%), Horse (100%), Rabbit (100%)
Conjugation	Unconjugated
Preparation and Storage	Store at -20 degree C. Stable for 15 months from date of receipt.

IMAGES

IHC (Immunohistochemistry)
Colon tissue HIF1A antibody used at 1/200 on formalin-fixed paraffin embedded tissue.

WB (Western Blot)
Western blot analysis of extracts of various sample, using hif1a antibody.

IF (Immunofluorescence)
AAA321279 staining HeLa cells by IF/ICC. The sample were fixed with PFA and permeabilized in 0.1% Triton X-100, then blocked in 10% serum for 45 minutes at 25 degree C. The primary antibody was diluted at 1/200 and incubated with the sample for 1 hour at 37 degree C. An Alexa Fluor 594 conjugated goat anti-rabbit IgG (H+L) antibody, diluted at 1/600, was used as secondary antibody.

WB (Western Blot)
Western blot analysis on LOVO using HIF1a antibody, The lane on the left is blocked with the antigen-specific peptide.

ADDITIONAL INFORMATION

Related Product Information for anti-HIF1A antibody

Description: Cell growth and viability is compromised by oxygen deprivation (hypoxia). Hypoxia-inducible factors, including HIF-1alpha, Arnt 1 (also designated HIF-1beta), EPAS-1 (also designated HIF-2alpha) and HIF-3alpha, induce glycolysis, erythropoiesis and angiogenesis in order to restore oxygen homeostasis. Hypoxia-inducible factors are members of the Per-Arnt-Sim (PAS) domain transcription factor family.
Function: Functions as a master transcriptional regulator of the adaptive response to hypoxia. 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. Plays an essential role in embryonic vascularization, tumor angiogenesis and pathophysiology of ischemic disease. Binds to core DNA sequence 5'-[AG]CGTG-3' within the hypoxia response element (HRE) of target gene promoters. Activation requires recruitment of transcriptional coactivators such as CREBBP and EP300. Activity is enhanced by interaction with both, NCOA1 or NCOA2. Interaction with redox regulatory protein APEX seems to activate CTAD and potentiates activation by NCOA1 and CREBBP. Involved in the axonal distribution and transport of mitochondria in neurons during hypoxia.
Subunit Structure: Interacts with the HIF1A beta/ARNT subunit; heterodimerization is required for DNA binding. Interacts with COPS5; the interaction increases the transcriptional activity of HIF1A through increased stability (By similarity). Interacts with EP300 (via TAZ-type 1 domains); the interaction is stimulated in response to hypoxia and inhibited by CITED2. Interacts with CREBBP (via TAZ-type 1 domains). Interacts with NCOA1, NCOA2, APEX and HSP90. Interacts (hydroxylated within the ODD domain) with VHLL (via beta domain); the interaction, leads to polyubiquitination and subsequent HIF1A proteasomal degradation. During hypoxia, sumoylated HIF1A also binds VHL; the interaction promotes the ubiquitination of HIF1A. Interacts with SENP1; the interaction desumoylates HIF1A resulting in stabilization and activation of transcription. Interacts (Via the ODD domain) with ARD1A; the interaction appears not to acetylate HIF1A nor have any affect on protein stability, during hypoxia. Interacts with RWDD3; the interaction enhances HIF1A sumoylation. Interacts with TSGA10 (By similarity). Interacts with HIF3A (By similarity). Interacts with RORA (via the DNA binding domain); the interaction enhances HIF1A transcription under hypoxia through increasing protein stability. Interaction with PSMA7 inhibits the transactivation activity of HIF1A under both normoxic and hypoxia-mimicking conditions. Interacts with USP20. Interacts with RACK1; promotes HIF1A ubiquitination and proteasome-mediated degradation. Interacts (via N-terminus) with USP19. Interacts with SIRT2. Interacts (deacetylated form) with EGLN1. Interacts with CBFA2T3. Interacts with HSP90AA1 and HSP90AB1 (PubMed:26517842).
Post-translational Modifications: S-nitrosylation of Cys-800 may be responsible for increased recruitment of p300 coactivator necessary for transcriptional activity of HIF-1 complex.Requires phosphorylation for DNA-binding. Phosphorylation at Ser-247 by CSNK1D/CK1 represses kinase activity and impairs ARNT binding. Phosphorylation by GSK3-beta and PLK3 promote degradation by the proteasome. Sumoylated; with SUMO1 under hypoxia. Sumoylation is enhanced through interaction with RWDD3. Both sumoylation and desumoylation seem to be involved in the regulation of its stability during hypoxia. Sumoylation can promote either its stabilization or its VHL-dependent degradation by promoting hydroxyproline-independent HIF1A-VHL complex binding, thus leading to HIF1A ubiquitination and proteasomal degradation. Desumoylation by SENP1 increases its stability amd transcriptional activity. There is a disaccord between various publications on the effect of sumoylation and desumoylation on its stability and transcriptional activity. Acetylation of Lys-532 by ARD1 increases interaction with VHL and stimulates subsequent proteasomal degradation (PubMed:12464182). Deacetylation of Lys-709 by SIRT2 increases its interaction with and hydroxylation by EGLN1 thereby inactivating HIF1A activity by inducing its proteasomal degradation (PubMed:24681946). Polyubiquitinated; in normoxia, following hydroxylation and interaction with VHL. Lys-532 appears to be the principal site of ubiquitination. Clioquinol, the Cu/Zn-chelator, inhibits ubiquitination through preventing hydroxylation at Asn-803. Ubiquitinated by a CUL2-based E3 ligase. In normoxia, is hydroxylated on Pro-402 and Pro-564 in the oxygen-dependent degradation domain (ODD) by EGLN1/PHD2 and EGLN2/PHD1 (PubMed:11292861, PubMed:11566883, PubMed:12351678, PubMed:15776016, PubMed:25974097). EGLN3/PHD3 has also been shown to hydroxylate Pro-564 (PubMed:11292861, PubMed:11566883, PubMed:12351678, PubMed:15776016, PubMed:25974097). The hydroxylated prolines promote interaction with VHL, initiating rapid ubiquitination and subsequent proteasomal degradation (PubMed:11292861, PubMed:11566883, PubMed:12351678, PubMed:15776016, PubMed:25974097). Deubiquitinated by USP20 (PubMed:11292861, PubMed:11566883, PubMed:12351678, PubMed:15776016, PubMed:25974097). Under hypoxia, proline hydroxylation is impaired and ubiquitination is attenuated, resulting in stabilization (PubMed:11292861, PubMed:11566883, PubMed:12351678, PubMed:15776016, PubMed:25974097). In normoxia, is hydroxylated on Asn-803 by HIF1AN, thus abrogating interaction with CREBBP and EP300 and preventing transcriptional activation (PubMed:12080085). This hydroxylation is inhibited by the Cu/Zn-chelator, Clioquinol (PubMed:12080085). Repressed by iron ion, via Fe2+ prolyl hydroxylase (PHD) enzymes-mediated hydroxylation and subsequent proteasomal degradation (PubMed:28296633). The iron and 2-oxoglutarate dependent 3-hydroxylation of asparagine is (S) stereospecific within HIF CTAD domains.
Similarity: Contains two independent C-terminal transactivation domains, NTAD and CTAD, which function synergistically. Their transcriptional activity is repressed by an intervening inhibitory domain (ID).

References	Dong J, Zheng N, Wang X, Tang C, Yan P, Zhou HB, Huang J; Journal: Eur J Pharmacol. A novel HDAC6 inhibitor exerts an anti-cancer effect by triggering cell cycle arrest and apoptosis in gastric cancer. Zhang L, Xiong W, Li N, Liu H, He H, Du Y, Zhang Z, Liu Y; Journal: Fertil Steril. Estrogen stabilizes hypoxia-inducible factor 1a through G protein-coupled estrogen receptor 1 in eutopic endometrium of endometriosis. Liu H, Du Y, Zhang Z, Lv L, Xiong W, Zhang L, Li N, He H, Li Q, Liu Y; Journal: Biol Reprod. Autophagy contributes to hypoxia-induced epithelial to mesenchymal transition of endometrial epithelial cells in endometriosis. Xiong Y, Liu Y, Xiong W, Zhang L, Liu H, Du Y, Li N; Journal: Hum Reprod. Hypoxia-inducible factor 1a-induced epithelial-mesenchymal transition of endometrial epithelial cells may contribute to the development of endometriosis. Liu H, Zhang Z, Xiong W, Zhang L, Xiong Y, Li N, He H, Du Y, Liu Y; Journal: Reproduction. Hypoxia-inducible factor-1a promotes endometrial stromal cells migration and invasion by upregulating autophagy in endometriosis.

NCBI AND UNIPROT INFORMATION

NCBI GI #	340545531
NCBI GeneID	3091
NCBI Accession #	NP_001230013.1
NCBI GenBank Nucleotide #	NM_001243084.1
UniProt Accession #	Q16665; Q53XP6; Q96PT9; Q9UPB1; C0LZJ3
Molecular Weight	Observed: 120 kDa Predicted: 93 kDa
NCBI Official Full Name	hypoxia-inducible factor 1-alpha isoform 3
NCBI Official Synonym Full Names	hypoxia inducible factor 1 subunit alpha
NCBI Official Symbol	HIF1A
NCBI Official Synonym Symbols	HIF1; MOP1; PASD8; HIF-1A; bHLHe78; HIF-1alpha; HIF1-ALPHA; HIF-1-alpha
NCBI Protein Information	hypoxia-inducible factor 1-alpha
UniProt Protein Name	Hypoxia-inducible factor 1-alpha
UniProt Gene Name	**HIF1A**
UniProt Synonym Gene Names	BHLHE78; MOP11 PublicationManual assertion based on opinion iniRef.2; HIF-1-alpha1 PublicationManual assertion based on opinion iniRef.1; HIF1-alpha1 PublicationManual assertion based on opinion iniRef.1; bHLHe78

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