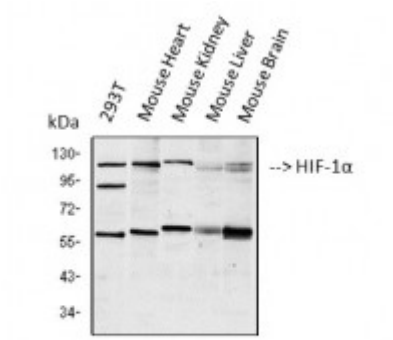
Anti-HIF- alpha antibody



Western Blot (WB) analysis of 1)293T, 2)mouse heart, 3)mouse kidney, 4)mouse liver, 5)mouse brain cell lysate using HIF-1α Antibody (STJ93498).

Description	HIF-1alpha is a protein encoded by the HIF1A gene which is approximately 92, 6 kDa. HIF-1alpha is localised to the cytoplasm and nucleus. It is involved in CDK-mediated phosphorylation, removal of Cdc6, HIF repressor pathways, signalling by PTK6 and the notch signalling pathway. It functions as a master regulator of cellular and systemic homeostatic response to hypoxia by activating transcription of many genes, including those involved in energy metabolism, angiogenesis, apoptosis, and other genes whose protein products increase oxygen delivery or facilitate metabolic adaptation to hypoxia. HIF-1alpha is expressed in most tissues with highest levels in kidney and heart. Mutations in the HIF1A gene may result in hypoxia and retinal ischemia. STJ93498 was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. This polyclonal antibody detects endogenous levels of HIF-1alpha protein.

Model	STJ93498
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IHC, WB
Immunogen	Synthesized peptide derived from human HIF-1alpha.
Immunogen Region	Internal
Gene ID	3091
Gene Symbol	HIF1A
Dilution range	WB 1:500-1:2000IHC 1:100-1:300ELISA 1:40000

Specificity	HIF-1alpha Polyclonal Antibody detects endogenous levels of HIF-1alpha protein.
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.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	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-c
Molecular Weight	110 kDa
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:4910OMIM:603348
Alternative 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-c
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.
Sequence and Domain Family	Contains two independent C-terminal transactivation domains, NTAD and CTAD, which function synergistically. Their transcriptional activity is repressed by an intervening inhibitory domain (ID).
Cellular Localization	Cytoplasm Nucleus Nucleus speckle. Colocalizes with HIF3A in the nucleus and speckles . Cytoplasmic in normoxia, nuclear translocation in response to hypoxia .

Post-translational Modifications	In normoxia, is hydroxylated on Pro-402 and Pro-564 in the oxygen-dependent degradation domain (ODD) by EGLN1/PHD2 and EGLN2/PHD1. EGLN3/PHD3 has also been shown to hydroxylate Pro-564. The hydroxylated prolines promote interaction with VHL, initiating rapid ubiquitination and subsequent proteasomal degradation. Deubiquitinated by USP20. Under hypoxia, proline hydroxylation is impaired and ubiquitination is attenuated, resulting in stabilization. In normoxia, is hydroxylated on Asn-803 by HIF1AN, thus abrogating interaction with CREBBP and EP300 and preventing transcriptional activation. This hydroxylation is inhibited by the Cu/Zn-chelator, Clioquinol. 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 . Deacetylation of Lys-709 by SIRT2 increases its interaction with and hydroxylation by EGLN1 thereby inactivating HIF1A activity by inducing its proteasomal degradation . 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. The iron and 2-oxoglutarate dependent 3-hydroxylation of asparagine is (S) stereospecific within HIF CTAD domains.