ARG82980
Mouse HIF-1 alpha ELISA Kit

Package: 96 wells
Store at: 4°C

Summary

Product Description	ARG82980 Mouse HIF-1 alpha ELISA Kit is an Enzyme Immunoassay kit for the quantification of Mouse HIF-1 alpha in serum, plasma, cell culture supernatant samples.
Tested Reactivity	Ms
Tested Application	ELISA
Specificity	Cross-Reactivity: This kit reacts to native and recombinant Mouse HIF-1 alpha protein, it also cross-reacts to human, rat and monkey HIF-1 alpha.
Target Name	HIF-1 alpha
Conjugation	HRP
Conjugation Note	Substrate: TMB and read at 450 nm.
Sensitivity	62.5 pg/ml
Sample Type	Serum, plasma and cell culture supernatants.
Standard Range	125 - 8000 pg/ml
Sample Volume	100 µl
Alternate Names	Class E basic helix-loop-helix protein 78; Basic-helix-loop-helix-PAS protein MOP1; Hypoxia-inducible factor 1-alpha; PAS domain-containing protein 8; HIF1-alpha; HIF1-ALPHA; HIF1; MOP1; HIF-1-alpha; PASD8; HIF-1A; HIF-1alpha; Member of PAS protein 1; ARNT-interacting protein; bHLHe78

Application Instructions

Assay Time	~ 4 hours

Properties

Form	96 well
Storage instruction	Store the kit at 2-8°C. Keep microplate wells sealed in a dry bag with desiccants. Do not expose test reagents to heat, sun or strong light during storage and usage. Please refer to the product user manual for detail temperatures of the components.
Note	For laboratory research only, not for drug, diagnostic or other use.

Bioinformation

Gene Symbol	HIF1A
Gene Full Name	hypoxia inducible factor 1, alpha subunit (basic helix-loop-helix transcription factor)
Background	This gene encodes the alpha subunit of transcription factor hypoxia-inducible factor-1 (HIF-1), which is a heterodimer composed of an alpha and a beta subunit. HIF-1 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-1 thus plays an

	essential role in embryonic vascularization, tumor angiogenesis and pathophysiology of ischemic disease. Alternatively spliced transcript variants encoding different isoforms have been identified for this gene. [provided by RefSeq, Jul 2011]
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 CREBPB 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. [UniProt]
Highlight	Related products: HIF-1 alpha antibodies; HIF-1 alpha ELISA Kits; New ELISA data calculation tool: Simplify the ELISA analysis by GainData
Research Area	Cancer kit; Cell Biology and Cellular Response kit; Gene Regulation kit; Metabolism kit
PTM	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 (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. The iron and 2-oxoglutarate dependent 3-hydroxylation of asparagine is (S) stereospecific within HIF CTAD domains.