

HIF1A Monoclonal Antibody (ESEE122), Biotin

Catalog NumberMA5-16016

Product data sheet

Details		Species Reactivity	
Size	100 µL	Species reactivity	Bovine, Dog, Human, Mouse, Rat
Host/Isotope	Mouse / IgG1	Tested Applications	Dilution *
Class	Monoclonal		
Type	Antibody		
Clone	ESEE122		
Immunogen	Human HIF-1 alpha amino acids 329-530.		
Conjugate	Biotin		
Form	Liquid	Flow Cytometry (Flow)	Assay-Dependent
Concentration	0.65 mg/mL	Immunohistochemistry (Frozen) (IHC (F))	1:100-1:5000
Purification	Protein G	Immunohistochemistry (Paraffin) (IHC (P))	1:100-1:5000
Storage buffer	PBS	Western Blot (WB)	1:500-1:1,000
Contains	0.05% sodium azide	Immunocytochemistry (ICC/IF)	Assay-Dependent
Storage Conditions	4° C, store in dark	* Suggested working dilutions are given as a guide only. It is recommended that the user titrate the product for use in their own experiment using appropriate negative and positive controls.	

Product specific information

Suggested positive control: Cos7 CoCl2-treated nuclear extract.

Background/Target Information

HIF1-alpha (HIF1A) is a subunit of HIF1, which is a transcription factor found in mammalian cells cultured under reduced oxygen tension. HIF-1 is a heterodimer consisting of an alpha and beta subunit, both belonging to the basic-helix-loop-helix Per-aryl hydrocarbon receptor nuclear translocator-Sim (PAS) family of transcription factors. HIF1 functions as a transcriptional regulator of the adaptive response to hypoxia. Under hypoxic conditions, HIF-1 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. HIF1-alpha regulates hypoxia-mediated apoptosis, cell proliferation and tumor angiogenesis. Hypoxia which induces p53 protein accumulation, directly interacts with HIF1-alpha and reduces hypoxia-induced expression of HIF1-alpha by promoting MDM2-mediated ubiquitination and proteasomal degradation under hypoxic conditions. Recent studies suggest that induction of NOX4 by HIF1-alpha contributes to maintain ROS levels after hypoxia and hypoxia-induced proliferation. In humans, it is located on the q arm of chromosome 14. The C-terminal of HIF1A binds to p300. p300/CBP-HIF complexes participate in the induction of hypoxia-responsive genes, including VEGF. Hypoxia contributes significantly to the pathophysiology of major categories of human disease, including myocardial and cerebral ischemia, cancer, pulmonary hypertension, congenital heart disease and chronic obstructive pulmonary disease.

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