EPAS1 Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP10707a

Specification

EPAS1 Antibody (N-term) - Product Information

Application	**WB, FC,E**
Primary Accession	Q99814
Other Accession	NP_001421.2
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**96459**
Antigen Region	**124-150**

EPAS1 Antibody (N-term) - Additional Information

Gene ID 2034

Other Names
Endothelial PAS domain-containing protein 1, EPAS-1, Basic-helix-loop-helix-PAS protein MOP2, Class E basic helix-loop-helix protein 73, bHLHe73, HIF-1-alpha-like factor, HLF, Hypoxia-inducible factor 2-alpha, HIF-2-alpha, HIF2-alpha, Member of PAS protein 2, PAS domain-containing protein 2, EPAS1, BHLHE73, HIF2A, MOP2, PASD2

Target/Specificity
This EPAS1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 124-150 amino acids from the N-terminal region of human EPAS1.

Dilution
WB~~1:1000
FC~~1:10~50

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
EPAS1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

EPAS1 Antibody (N-term) - Protein Information

Name EPAS1

Synonyms BHLHE73, HIF2A, MOP2, PASD2

Function Transcription factor involved in the induction of oxygen regulated genes. Heterodimerizes with ARNT; heterodimer binds to core DNA sequence 5'-TACGTG-3' within the hypoxia response element (HRE) of target gene promoters (By similarity). Regulates the vascular endothelial growth factor (VEGF) expression and seems to be implicated in the development of blood vessels and the tubular system of lung. May also play a role in the formation of the endothelium that gives rise to the blood brain barrier. Potent activator of the Tie-2 tyrosine kinase expression. Activation requires recruitment of transcriptional coactivators such as CREBBP and probably EP300. Interaction with redox regulatory protein APEX1 seems to activate CTAD (By similarity).

Cellular Location
Nucleus {ECO:0000250|UniProtKB:P97481, ECO:0000255|PROSITE-ProRule:PRU00981}. Nucleus speckle {ECO:0000250|UniProtKB:P97481}. Note=Colocalizes with HIF3A in the nucleus and speckles. {ECO:0000250|UniProtKB:P97481}

Tissue Location
Expressed in most tissues, with highest levels in placenta, lung and heart. Selectively expressed in endothelial cells
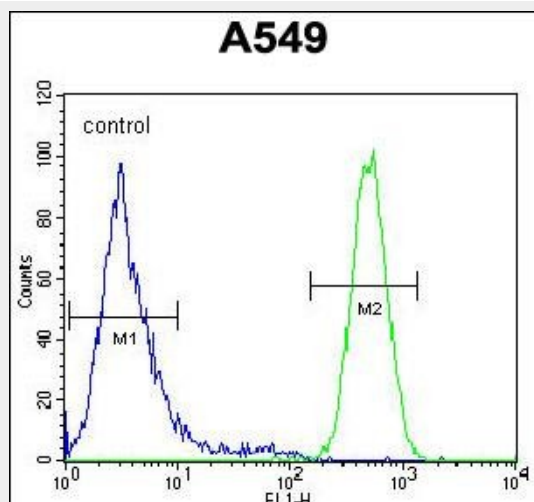
EPAS1 Antibody (N-term) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytomety](#)
- [Cell Culture](#)

EPAS1 Antibody (N-term) - Images
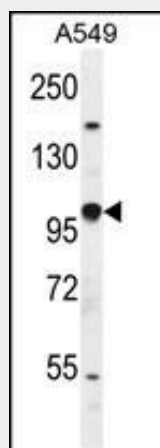
EPAS1 Antibody (N-term) (Cat. #AP10707a) western blot analysis in A549 cell line lysates (35ug/lane).This demonstrates the EPAS1 antibody detected the EPAS1 protein (arrow).

EPAS1 Antibody (N-term) (Cat. #AP10707a) flow cytometric analysis of A549 cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

EPAS1 Antibody (N-term) - Background

This gene encodes a transcription factor involved in the induction of genes regulated by oxygen, which is induced as oxygen levels fall. The encoded protein contains a basic-helix-loop-helix domain protein dimerization domain as well as a domain found in proteins in signal transduction pathways which respond to oxygen levels. Mutations in this gene are associated with erythrocytosis familial type 4.

EPAS1 Antibody (N-term) - References

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)
Yi, X., et al. Science 329(5987):75-78(2010)
Bougatef, F., et al. PLoS ONE 5 (8), E12265 (2010) :
Hossein Ghaderian, S.M., et al. Pathology 42(5):446-453(2010)
Mowat, F.M., et al. PLoS ONE 5 (6), E11103 (2010) :