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Sik1 Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP18232b

Specification

Sik1 Antibody (C-term) Blocking peptide - Product Information

Primary Accession Q60670

Sik1 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 17691

Other Names
Serine/threonine-protein kinase SIK1, HRT-20, Myocardial SNF1-like kinase, Salt-inducible kinase 1, SIK-1, Serine/threonine-protein kinase SNF1-like kinase 1, Serine/threonine-protein kinase SNF1LK, Sik1, Msk, Sik, Snf1lk

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Sik1 Antibody (C-term) Blocking peptide - Protein Information

Name Sik1

Synonyms Msk, Sik, Snf1lk

Function
Serine/threonine-protein kinase involved in various processes such as cell cycle regulation, gluconeogenesis and lipogenesis regulation, muscle growth and differentiation and tumor suppression. Phosphorylates HDAC4, HDAC5, PPME1, SREBF1, CRTC1/TORC1 and CRTC2/TORC2. Acts as a tumor suppressor and plays a key role in p53/TP53-dependent anoikis, a type of apoptosis triggered by cell detachment: required for phosphorylation of p53/TP53 in response to loss of adhesion and is able to suppress metastasis. Part of a sodium- sensing signaling network, probably by mediating phosphorylation of PPME1: following increases in intracellular sodium, SIK1 is activated by CaMK1 and phosphorylates PPME1 subunit of protein phosphatase 2A (PP2A), leading to dephosphorylation of sodium/potassium-transporting ATPase ATP1A1 and subsequent increase activity of ATP1A1. Acts as a regulator of muscle cells by phosphorylating and inhibiting class II histone deacetylases HDAC4 and HDAC5, leading to promote expression of MEF2 target genes in myocytes. Also required during cardiomyogenesis by regulating the exit of cardiomyoblasts from the cell cycle via down- regulation of CDKN1C/p57Kip2. Acts as a regulator of hepatic gluconeogenesis by phosphorylating and repressing the CREB-specific coactivators

CRTC1/TORC1 and CRTC2/TORC2, leading to inhibit CREB activity. Also regulates hepatic lipogenesis by phosphorylating and inhibiting SREBF1. In concert with CRTC1/TORC1, regulates the light- induced entrainment of the circadian clock by attenuating PER1 induction; represses CREB-mediated transcription of PER1 by phosphorylating and deactivating CRTC1/TORC1.

Cellular Location
Cytoplasm. Nucleus. Note=Following ACTH (adrenocorticotropic hormone) treatment and subsequent phosphorylation by PKA, translocates to the cytoplasm, where it binds to YWHAZ

Tissue Location
Expressed in lung, skin, ovary, heart and stomach. No expression in brain, liver or adult skeletal muscle but is present in skeletal muscle progenitor cells of the somite beginning at 9.5 dpc Present at 8.0 dpc in the monolayer of presumptive myocardial cells but rapidly down-regulated at 8.5 dpc upon primitive ventricle formation, although still present in myocardial cells that will populate the primitive atrium and bulbus cordis. At 9.5 dpc expression is down- regulated in the primitive atrium but observed in the sinus venosus and truncus arteriosus.

Sik1 Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

Sik1 Antibody (C-term) Blocking peptide - Images

Sik1 Antibody (C-term) Blocking peptide - Background

Transient role during the earliest stages of myocardial cell differentiation and/or primitive chamber formation and may also be important for the earliest stages of skeletal muscle growth and/or differentiation. Potential role in G2/M cell cycle regulation. Inhibits CREB activity by phosphorylating and repressing the CREB-specific coactivators, CRTC1-3.

Sik1 Antibody (C-term) Blocking peptide - References

Romito, A., et al. PLoS ONE 5 (2), E9029 (2010) :Takemori, H., et al. Endocr. J. 56(1):121-130(2009)Berdeaux, R., et al. Nat. Med. 13(5):597-603(2007)Katoh, Y., et al. FEBS J. 273(12):2730-2748(2006)Cobellis, G., et al. Nucleic Acids Res. 33 (4), E44 (2005) :