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STK39 Antibody(Ascites)

Mouse Monoclonal Antibody (Mab)
Catalog # AM2027a

Specification

STK39 Antibody(Ascites) - Product Information

Application	**WB,E**
Primary Accession	Q9UEW8
Other Accession	O88506, Q9Z1W9, NP_037365.2
Reactivity	**Human**
Predicted	**Mouse, Rat**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**IgG1**
Calculated MW	**59474**

STK39 Antibody(Ascites) - Additional Information

Gene ID 27347

Other Names
STE20/SPS1-related proline-alanine-rich protein kinase, Ste-20-related kinase, DCHT, Serine/threonine-protein kinase 39, STK39, SPAK

Target/Specificity
Purified His-tagged STK39 protein(Fragment) was used to produced this monoclonal antibody.

Dilution
WB~~1:1000~4000
E~~Use at an assay dependent concentration.

Format
Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

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
STK39 Antibody(Ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

STK39 Antibody(Ascites) - Protein Information

Name STK39

Function Effector serine/threonine-protein kinase component of the WNK-SPAK/OSR1 kinase cascade, which is involved in various processes, such as ion transport, response to hypertonic

stress and blood pressure (PubMed:16669787, PubMed:18270262, PubMed:21321328, PubMed:34289367). Specifically recognizes and binds proteins with a RFXV motif (PubMed:16669787, PubMed:21321328). Acts downstream of WNK kinases (WNK1, WNK2, WNK3 or WNK4): following activation by WNK kinases, catalyzes phosphorylation of ion cotransporters, such as SLC12A1/NKCC2, SLC12A2/NKCC1, SLC12A3/NCC, SLC12A5/KCC2 or SLC12A6/KCC3, regulating their activity (PubMed:21321328). Mediates regulatory volume increase in response to hyperosmotic stress by catalyzing phosphorylation of ion cotransporters SLC12A1/NKCC2, SLC12A2/NKCC1 and SLC12A6/KCC3 downstream of WNK1 and WNK3 kinases (PubMed:12740379, PubMed:16669787, PubMed:21321328). Phosphorylation of Na-K-Cl cotransporters SLC12A2/NKCC1 and SLC12A2/NKCC1 promote their activation and ion influx; simultaneously, phosphorylation of K-Cl cotransporters SLC12A5/KCC2 and SLC12A6/KCC3 inhibit their activity, blocking ion efflux (PubMed:16669787, PubMed:19665974, PubMed:21321328). Acts as a regulator of NaCl reabsorption in the distal nephron by mediating phosphorylation and activation of the thiazide-sensitive Na-Cl cotransporter SLC12A3/NCC in distal convoluted tubule cells of kidney downstream of WNK4 (PubMed:18270262). Mediates the inhibition of SLC4A4, SLC26A6 as well as CFTR activities (By similarity). Phosphorylates RELT (By similarity).

Cellular Location
Cytoplasm. Nucleus. Note=Nucleus when caspase-cleaved.

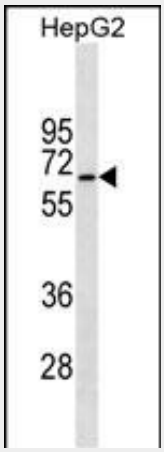
Tissue Location
Predominantly expressed in brain and pancreas followed by heart, lung, kidney, skeletal muscle, liver, placenta and testis.

STK39 Antibody(Ascites) - 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

STK39 Antibody(Ascites) - Images



STK39 Antibody (Cat. #AM2027a) western blot analysis in HepG2 cell line lysates (35µg/lane).This

demonstrates the STK39 antibody detected the STK39 protein (arrow).

STK39 Antibody(Ascites) - Background

This gene encodes a serine/threonine kinase that is thought to function in the cellular stress response pathway. The kinase is activated in response to hypotonic stress, leading to phosphorylation of several cation-chloride-coupled cotransporters. The catalytically active kinase specifically activates the p38 MAP kinase pathway, and its interaction with p38 decreases upon cellular stress, suggesting that this kinase may serve as an intermediate in the response to cellular stress. [provided by RefSeq].

STK39 Antibody(Ascites) - References

Duarte, J.D., et al. Pharmacogenet. Genomics 20(8):516-519(2010)
Sid, B., et al. J. Physiol. (Lond.) 588 (PT 13), 2315-2328 (2010) :
Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Balatoni, C.E., et al. Am. J. Pathol. 175(4):1653-1661(2009)
Cunnington, M.S., et al. BMC Med. Genet. 10, 135 (2009) :