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STK11 (LKB1) Antibody (N-term I29)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7239A

Product Information

Application	IHC-P, WB, FC, IF, E
Primary Accession	Q15831
Other Accession	Q91604, D4AE59, Q9WTK7, Q0GGW5
Reactivity	Human
Predicted	Chicken, Mouse, Rat, Xenopus
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	48636
Antigen Region	14-44

Additional Information

Gene ID	6794
Other Names	Serine/threonine-protein kinase STK11, Liver kinase B1, LKB1, hLKB1, Renal carcinoma antigen NY-REN-19, STK11, LKB1, PJS
Target/Specificity	This STK11 (LKB1) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 14-44 amino acids from the N-terminal region of human STK11 (LKB1).
Dilution	IHC-P~~1:100~500 WB~~1:1000 FC~~1:10~50 IF~~1:10~50 E~~Use at an assay dependent concentration.
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	STK11 (LKB1) Antibody (N-term I29) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	STK11 (HGNC:11389)
Synonyms	LKB1, PJS

Function	Tumor suppressor serine/threonine-protein kinase that controls the activity of AMP-activated protein kinase (AMPK) family members, thereby playing a role in various processes such as cell metabolism, cell polarity, apoptosis and DNA damage response. Acts by phosphorylating the T-loop of AMPK family proteins, thus promoting their activity: phosphorylates PRKAA1, PRKAA2, BRSK1, BRSK2, MARK1, MARK2, MARK3, MARK4, NUAK1, NUAK2, SIK1, SIK2, SIK3 and SNRK but not MELK. Also phosphorylates non-AMPK family proteins such as STRADA, PTEN and possibly p53/TP53. Acts as a key upstream regulator of AMPK by mediating phosphorylation and activation of AMPK catalytic subunits PRKAA1 and PRKAA2 and thereby regulates processes including: inhibition of signaling pathways that promote cell growth and proliferation when energy levels are low, glucose homeostasis in liver, activation of autophagy when cells undergo nutrient deprivation, and B-cell differentiation in the germinal center in response to DNA damage. Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton. Required for cortical neuron polarization by mediating phosphorylation and activation of BRSK1 and BRSK2, leading to axon initiation and specification. Involved in DNA damage response: interacts with p53/TP53 and recruited to the CDKN1A/WAF1 promoter to participate in transcription activation. Able to phosphorylate p53/TP53; the relevance of such result in vivo is however unclear and phosphorylation may be indirect and mediated by downstream STK11/LKB1 kinase NUAK1. Also acts as a mediator of p53/TP53-dependent apoptosis via interaction with p53/TP53: translocates to the mitochondrion during apoptosis and regulates p53/TP53-dependent apoptosis pathways. Regulates UV radiation-induced DNA damage response mediated by CDKN1A. In association with NUAK1, phosphorylates CDKN1A in response to UV radiation and contributes to its degradation which is necessary for optimal DNA repair (PubMed:25329316).
Cellular Location	Nucleus. Cytoplasm. Membrane. Mitochondrion. Note=A small fraction localizes at membranes (By similarity). Relocates to the cytoplasm when bound to STRAD (STRADA or STRADB) and CAB39/MO25 (CAB39/MO25alpha or CAB39L/MO25beta) Translocates to the mitochondrion during apoptosis. PTEN promotes cytoplasmic localization.
Tissue Location	Ubiquitously expressed. Strongest expression in testis and fetal liver

Background

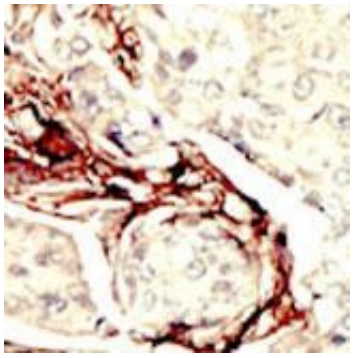
STK11, is a member of the serine/threonine kinase family, regulates cell polarity and functions as a tumor suppressor. Mutations in STK11 have been associated with Peutz-Jeghers syndrome, an autosomal dominant disorder characterized by the growth of polyps in the gastrointestinal tract, pigmented macules on the skin and mouth, and other neoplasms.

References

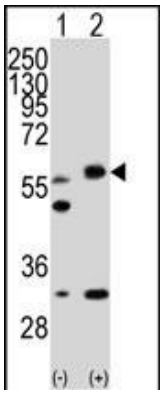
Mart, et al., J. Biotechnol. 115(1):23-34 (2005).
Shaw, R.J., et al., Proc. Natl. Acad. Sci. U.S.A. 101(10):3329-3335 (2004).
Suzuki, A., et al., Biochem. Biophys. Res. Commun. 324(3):986-992 (2004).
Hearle, N., et al., Genes Chromosomes Cancer 41(2):163-169 (2004).
Corradetti, M.N., et al., Genes Dev. 18(13):1533-1538 (2004).

Images

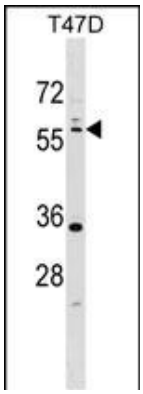
Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was



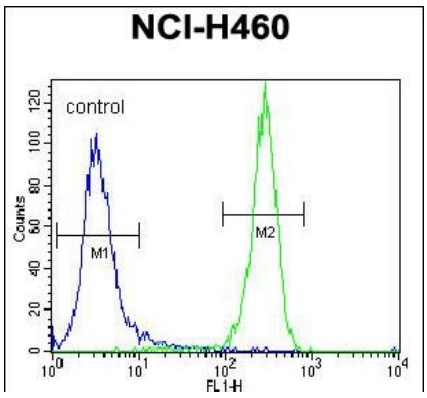
peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



Western blot analysis of STK11 (arrow) using rabbit polyclonal STK11 Antibody (Cat.#AP7239a). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the STK11 gene (Lane 2) (Origene Technologies).

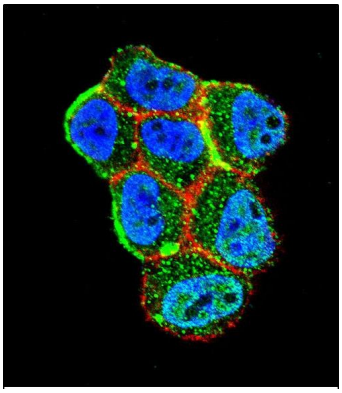


STK11 Antibody (I29) (Cat. #AP7239a) western blot analysis in T47D cell line lysates (35ug/lane).This demonstrates the STK11 antibody detected the STK11 protein (arrow).



STK11 (LKB1) Antibody (N-term I29) (Cat. #AP7239a) flow cytometric analysis of NCI-H460 cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

Confocal immunofluorescent analysis of STK11 (LKB1) Antibody (N-term I29)(Cat#AP7239a) with ZR-75-1 cell followed by Alexa Fluor 488-conjugated goat anti-rabbit lgG (green).Actin filaments have been labeled with Alexa Fluor 555 phalloidin (red).DAPI was used to stain the cell nuclear (blue).



Citations

- Upregulation of liver kinase B1 predicts poor prognosis in hepatocellular carcinoma.
- The methylenetetrahydrofolate reductase C677T mutation induces cell-specific changes in genomic DNA methylation and uracil misincorporation: a possible molecular basis for the site-specific cancer risk modification.