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LKB1 (phospho Thr189) Polyclonal Antibody

Catalog # AP67260

Product Information

Application	WB, E
Primary Accession	Q15831
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	48636

Additional Information

Gene ID	6794
Other Names	STK11; LKB1; PJS; Serine/threonine-protein kinase STK11; Liver kinase B1; LKB1; hLKB1; Renal carcinoma antigen NY-REN-19
Dilution	WB~~Western Blot: 1/500 - 1/2000. ELISA: 1/5000. Not yet tested in other applications. E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20℃

Protein Information

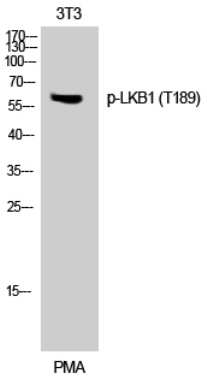
Name	STK11 (HGNC:11389)
Synonyms	LKB1, PJS
Function	Tumor suppressor serine/threonine-protein kinase that controls the activity of AMP-activated protein kinase (AMPK) family members, thereby playing a role in various processes such as cell metabolism, cell polarity, apoptosis and DNA damage response (PubMed:12805220, PubMed:14517248, PubMed:14976552, PubMed:15016379, PubMed:15733851). 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 (PubMed:12805220, PubMed:14517248, PubMed:14976552, PubMed:15016379, PubMed:15733851). Also phosphorylates non-AMPK family proteins such as STRADA, PTEN and possibly p53/TP53 (PubMed:11430832, PubMed:15987703). 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 (PubMed:14976552). Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton (PubMed:15016379). Required for cortical neuron polarization by mediating phosphorylation and activation of BRSK1 and BRSK2, leading to axon initiation and specification (PubMed:14976552). Involved in DNA damage response: interacts with p53/TP53 and recruited to the CDKN1A/WAF1 promoter to participate in transcription activation (PubMed:11430832, PubMed:17108107, PubMed:21317932). 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 (PubMed:11430832, PubMed:17108107, PubMed:21317932). 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 (PubMed:11430832, PubMed:17108107, PubMed:21317932). Regulates UV radiation-induced DNA damage response mediated by CDKN1A (PubMed:25329316). 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 {ECO:0000250|UniProtKB:Q9WTK7} Mitochondrion. Late endosome {ECO:0000250|UniProtKB:Q9WTK7}. 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) (PubMed:18854309). Translocates to the mitochondrion during apoptosis. PTEN promotes cytoplasmic localization (PubMed:11430832). Translocates to late endosomes in response to calorie restriction (By similarity). {ECO:0000250|UniProtKB:Q9WTK7, ECO:0000269|PubMed:11430832, ECO:0000269|PubMed:18854309}
Tissue Location	Ubiquitously expressed. Strongest expression in testis and fetal liver

Background

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. In vein endothelial cells, inhibits PI3K/Akt signaling activity and thus induces apoptosis in response to the oxidant peroxynitrite (in vitro). 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).

Images



Western Blot analysis of 3T3 cells using Phospho-LKB1 (T189) Polyclonal Antibody

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.