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Mouse Lats2 Antibody(C-term) Blocking peptide

Synthetic peptide
Catalog # BP19422b

Specification

Mouse Lats2 Antibody(C-term) Blocking peptide - Product Information

Primary Accession Q7TSJ6

Mouse Lats2 Antibody(C-term) Blocking peptide - Additional Information

Gene ID 50523

Other Names
Serine/threonine-protein kinase LATS2, Kinase phosphorylated during mitosis protein, Large tumor suppressor homolog 2, Serine/threonine-protein kinase kpm, Lats2 {ECO:0000312|EMBL:AAH530281}

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.

Mouse Lats2 Antibody(C-term) Blocking peptide - Protein Information

Name Lats2 {ECO:0000312|EMBL:AAH53028.1}

Function
Negative regulator of YAP1 in the Hippo signaling pathway that plays a pivotal role in organ size control and tumor suppression by restricting proliferation and promoting apoptosis. The core of this pathway is composed of a kinase cascade wherein STK3/MST2 and STK4/MST1, in complex with its regulatory protein SAV1, phosphorylates and activates LATS1/2 in complex with its regulatory protein MOB1, which in turn phosphorylates and inactivates YAP1 oncoprotein and WWTR1/TAZ. Phosphorylation of YAP1 by LATS2 inhibits its translocation into the nucleus to regulate cellular genes important for cell proliferation, cell death, and cell migration. Acts as a tumor suppressor which plays a critical role in centrosome duplication, maintenance of mitotic fidelity and genomic stability. Negatively regulates G1/S transition by down-regulating cyclin E/CDK2 kinase activity. Negative regulator of the androgen receptor. Phosphorylates SNAI1 in the nucleus leading to its nuclear retention and stabilization, which enhances its epithelial-mesenchymal transition and tumor cell invasion/migration activities. This tumor-promoting activity is independent of its effects upon YAP1 or WWTR1/TAZ (By similarity).

Cellular Location
Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm. Cytoplasm,

cytoskeleton, spindle pole. Nucleus. Note=Colocalizes with AURKA at the centrosomes during interphase, early prophase and cytokinesis (By similarity). Migrates to the spindle poles during mitosis, and to the midbody during cytokinesis. Translocates to the nucleus upon mitotic stress by nocodazole treatment (By similarity)

Tissue Location
Expressed at high levels in ovary and testis and at lower levels in all other tissues examined

Mouse Lats2 Antibody(C-term) Blocking peptide - Protocols

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

- Blocking Peptides

Mouse Lats2 Antibody(C-term) Blocking peptide - Images

Mouse Lats2 Antibody(C-term) Blocking peptide - Background

Negative regulator of YAP1 in the Hippo signaling pathway that plays a pivotal role in organ size control and tumor suppression by restricting proliferation and promoting apoptosis. The core of this pathway is composed of a kinase cascade wherein MST1/MST2, in complex with its regulatory protein SAV1, phosphorylates and activates LATS1/2 in complex with its regulatory protein MOB1, which in turn phosphorylates and inactivates YAP1 oncoprotein and WWTR1/TAZ. Phosphorylation of YAP1 by LATS2 inhibits its translocation into the nucleus to regulate cellular genes important for cell proliferation, cell death, and cell migration. Acts as a tumor suppressor which plays a critical role in centrosome duplication, maintenance of mitotic fidelity and genomic stability. Negatively regulates G1/S transition by down-regulating cyclin E/CDK2 kinase activity. Negative regulator of the androgen receptor (By similarity).

Mouse Lats2 Antibody(C-term) Blocking peptide - References

Zhang, N., et al. Dev. Cell 19(1):27-38(2010)Oh, S., et al. Mol. Cell. Biol. 29(23):6309-6320(2009)Quina, L.A., et al. J. Neurosci. 29(45):14309-14322(2009)Nishioka, N., et al. Dev. Cell 16(3):398-410(2009)Matsui, Y., et al. Circ. Res. 103(11):1309-1318(2008)