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MLCK Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP7966a

Specification

MLCK Antibody (N-term) Blocking peptide - Product Information

Primary Accession	Q15746
Other Accession	Q9C0L5

MLCK Antibody (N-term) Blocking peptide - Additional Information

Gene ID 4638

Other Names
Myosin light chain kinase, smooth muscle, MLCK, smMLCK, Kinase-related protein, KRP, Telokin, Myosin light chain kinase, smooth muscle, deglutamylated form, MYLK, MLCK, MLCK1, MYLK1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7966a was selected from the N-term region of human MLCK-long . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

MLCK Antibody (N-term) Blocking peptide - Protein Information

Name MYLK (HGNC:7590)

Synonyms MLCK, MLCK1, MYLK1

Function
Calcium/calmodulin-dependent myosin light chain kinase implicated in smooth muscle contraction via phosphorylation of myosin light chains (MLC). Also regulates actin-myosin interaction through a non-kinase activity. Phosphorylates PTK2B/PYK2 and myosin light-chains. Involved in the inflammatory response (e.g. apoptosis, vascular permeability, leukocyte diapedesis), cell motility and morphology, airway hyperreactivity and other activities relevant to asthma. Required for tonic airway smooth muscle contraction that is necessary for physiological and asthmatic airway resistance. Necessary for gastrointestinal motility. Implicated in the regulation of endothelial as well as vascular permeability, probably via the regulation of cytoskeletal rearrangements. In the

nervous system it has been shown to control the growth initiation of astrocytic processes in culture and to participate in transmitter release at synapses formed between cultured sympathetic ganglion cells. Critical participant in signaling sequences that result in fibroblast apoptosis. Plays a role in the regulation of epithelial cell survival. Required for epithelial wound healing, especially during actomyosin ring contraction during purse-string wound closure. Mediates RhoA-dependent membrane blebbing. Triggers TRPC5 channel activity in a calcium-dependent signaling, by inducing its subcellular localization at the plasma membrane. Promotes cell migration (including tumor cells) and tumor metastasis. PTK2B/PYK2 activation by phosphorylation mediates ITGB2 activation and is thus essential to trigger neutrophil transmigration during acute lung injury (ALI). May regulate optic nerve head astrocyte migration. Probably involved in mitotic cytoskeletal regulation. Regulates tight junction probably by modulating ZO-1 exchange in the perijunctional actomyosin ring. Mediates burn-induced microvascular barrier injury; triggers endothelial contraction in the development of microvascular hyperpermeability by phosphorylating MLC. Essential for intestinal barrier dysfunction. Mediates Giardia spp.-mediated reduced epithelial barrier function during giardiasis intestinal infection via reorganization of cytoskeletal F-actin and tight junctional ZO-1. Necessary for hypotonicity-induced Ca(2+) entry and subsequent activation of volume-sensitive organic osmolyte/anion channels (VSOAC) in cervical cancer cells. Responsible for high proliferative ability of breast cancer cells through anti-apoptosis.

Cellular Location
Cytoplasm. Cell projection, lamellipodium. Cleavage furrow. Cytoplasm, cytoskeleton, stress fiber. Note=Localized to stress fibers during interphase and to the cleavage furrow during mitosis

Tissue Location
Smooth muscle and non-muscle isozymes are expressed in a wide variety of adult and fetal tissues and in cultured endothelium with qualitative expression appearing to be neither tissue- nor development-specific. Non-muscle isoform 2 is the dominant splice variant expressed in various tissues. Telokin has been found in a wide variety of adult and fetal tissues. Accumulates in well differentiated enterocytes of the intestinal epithelium in response to tumor necrosis factor (TNF).

MLCK Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

MLCK Antibody (N-term) Blocking peptide - Images

MLCK Antibody (N-term) Blocking peptide - Background

MLCK, a member of the Ser/Thr protein kinase family, is a calcium/calmodulin-dependent enzyme responsible for smooth muscle contraction via phosphorylation of a specific serine in the N-terminus of myosin light chains (MLC), an event that facilitates myosin interaction with actin filaments. It is a central determinant in the development of vascular permeability and tissue edema formation. In the nervous system it has been shown to control the growth initiation of astrocytic processes in culture and to participate in transmitter release at synapses formed between cultured sympathetic ganglion cells. MLCK acts as a critical participant in signaling sequences that result in fibroblast apoptosis. Smooth muscle and non-muscle isozymes are expressed in a wide variety of adult and fetal tissues and in cultured endothelium with qualitative expression appearing to be neither tissue- nor development-specific. Non-muscle isoform 2 is the dominant splice variant expressed in various tissues. The Telokin isoform, which binds calmodulin, has been found in a wide variety of adult and fetal tissues. MLCK is probably down-regulated by phosphorylation. The protein contains 1 fibronectin type III domain and 9 immunoglobulin-like C2-type domains.

MLCK Antibody (N-term) Blocking peptide - References

Lazar, V., et al., Genomics 57(2):256-267 (1999).Watterson, D.M., et al., J. Cell. Biochem. 75(3):481-491 (1999).Garcia, J.G., et al., Am. J. Respir. Cell Mol. Biol. 16(5):489-494 (1997).Potier, M.C., et al., Genomics 29(3):562-570 (1995).