1. Phylogeny  
   Myosin light chain kinase, smooth muscle (smMLCK), is encoded by the MYLK gene and is ubiquitously expressed in all vertebrate smooth muscles. smMLCK is a member of the Ca²⁺/calmodulin-dependent serine/threonine kinase family that is evolutionarily conserved among mammals and other eukaryotes. This kinase is closely related to other isoforms that arise from the same gene, including skeletal muscle MLCK (skMLCK) and cardiac MLCK (cMLCK), and it is sometimes referred to by alternative names such as MLCK1 and MYLK1, thereby distinguishing it as the smooth muscle–specific isoform. Cross-species comparisons indicate that the catalytic core and the regulatory domains are highly conserved among mammalian species, supporting its critical function in contractile regulation (flores2007avariantof pages 1-3, gao2001myosinlightchain pages 1-2). Furthermore, smMLCK is grouped within the kinome branch that includes Ca²⁺/calmodulin-activated enzymes, a group representing an ancient evolutionary lineage that can be traced back to early eukaryotic ancestors (herring2000smoothmusclemyosin pages 1-2). Phylogenetic studies of the human protein kinase complement have placed MLCK in a clade with other kinases essential for cytoskeletal regulation, highlighting its role as part of an evolutionary core set of Ca²⁺-regulated kinases that also includes proteins such as myosin phosphatase and related regulators (gao2001myosinlightchain pages 1-2, herring2000smoothmusclemyosin pages 1-2).
2. Reaction Catalyzed  
   smMLCK catalyzes the transfer of a phosphate group from adenosine triphosphate (ATP) to specific serine/threonine residues on its substrate, the 20-kDa regulatory light chain (MLC20) of smooth muscle myosin. The overall chemical reaction can be summarized as:  
     ATP + [myosin regulatory light chain] → ADP + [myosin regulatory light chain]-phosphate + H⁺  
   This phosphorylation event predominantly occurs at Ser19 of MLC20 and is essential for the activation of myosin ATPase activity, which in turn initiates the actomyosin cross-bridge cycling that generates smooth muscle contraction (shen2010myosinlightchain pages 1-2, somlyo2003ca2+sensitivityofsmooth pages 2-3).
3. Cofactor Requirements  
   The kinase activity of smMLCK is strictly dependent on the presence of Ca²⁺ ions, which bind to calmodulin (CaM). The Ca²⁺/CaM complex interacts with smMLCK to induce a conformational change that releases the autoinhibitory domain and allows the kinase to become catalytically active. In addition, as with many kinases, smMLCK requires divalent cations such as Mg²⁺ to coordinate the phosphoryl transfer from ATP to the substrate. Thus, the proper function of smMLCK is contingent upon both Ca²⁺/calmodulin for regulatory activation and Mg²⁺ for the catalytic reaction (shen2010myosinlightchain pages 1-2, martinsen2014regulationofcalcium pages 1-2).
4. Substrate Specificity  
   smMLCK exhibits a high degree of substrate specificity for the myosin regulatory light chain (MLC20) of smooth muscle myosin. The enzyme phosphorylates MLC20 at key serine/threonine residues – most notably Ser19, and under certain conditions also Thr18 – which is requisite for switching on the myosin ATPase activity. The substrate binding pocket of smMLCK is optimally configured to recognize specific amino acid sequences flanking the phosphorylation site on MLC20, thereby ensuring selective catalysis. In addition to MLC20, there are reports that smMLCK phosphorylates other substrates such as PTK2B/PYK2, indicating that in different cellular contexts, smMLCK may exhibit additional substrate preferences linked to its broader role in modulating the cytoskeletal architecture (gao2001myosinlightchain pages 1-2, chen2014myosinlightchain pages 10-11).
5. Structure  
   smMLCK is composed of multiple functional domains that work in concert to confer both catalytic activity and regulatory control. The protein typically exists in a smooth muscle–specific isoform with an apparent molecular weight of approximately 130 kDa in many mammalian tissues, though alternative isoforms such as the 220-kDa long form and a shorter ~108 kDa variant have been reported in certain contexts (flores2007avariantof pages 1-3, herring2000smoothmusclemyosin pages 4-5).

At its C-terminus, smMLCK possesses a highly conserved catalytic kinase domain that houses key residues responsible for ATP binding, phosphate transfer, and substrate recognition. Adjacent to the catalytic core is an autoregulatory segment that contains a calmodulin-binding domain. In the inactive state, this autoregulatory sequence blocks access of the substrate to the catalytic core; binding of Ca²⁺/calmodulin displaces the inhibitory segment and enables kinase activation (gao2001myosinlightchain pages 2-4, chang2016cardiacmyosinlight pages 1-1).

The N-terminal region of smMLCK contains domains implicated in actin binding. This region can include immunoglobulin-like (Ig) modules, which are thought to mediate or stabilize the interaction of smMLCK with the actin cytoskeleton, thereby positioning the kinase near its substrate and possibly influencing local contractile activity (herring2000smoothmusclemyosin pages 7-8, chen2014myosinlightchain pages 7-9). Additionally, a distinct C-terminal domain known as telokin, which is expressed independently in some tissues, shares sequence identity with the C-terminal end of smMLCK and contributes to the regulation of smooth muscle contraction (ammit2000smoothmusclemyosinlightchain pages 3-5, chan2008identificationofcardiacspecific pages 14-18).

Structural studies and sequence analyses reveal that smMLCK, like other protein kinases, has a bilobal architecture with a smaller N-terminal lobe primarily involved in ATP binding and a larger C-terminal lobe responsible for substrate interaction and catalysis. Key structural features such as the activation loop, the hydrophobic spine, and the C-helix are conserved in smMLCK and are critical for its catalytic function (gao2001myosinlightchain pages 2-4, chang2016cardiacmyosinlight pages 1-1).

1. Regulation  
   The regulatory mechanisms that govern smMLCK activity are multifaceted and tightly coupled to intracellular Ca²⁺ signaling. In the resting state, smMLCK is maintained in an inactive conformation by its autoinhibitory domain. Upon elevation of intracellular Ca²⁺ concentrations, Ca²⁺ binds to calmodulin to form the Ca²⁺/calmodulin complex, which then interacts with the calmodulin-binding domain within smMLCK. The binding of Ca²⁺/calmodulin dislodges the autoinhibitory sequence, thereby activating the kinase and permitting substrate access to the catalytic cleft (shen2010myosinlightchain pages 1-2, chang2016cardiacmyosinlight pages 1-3).

In addition to Ca²⁺/calmodulin-dependent activation, smMLCK activity is further modulated by phosphorylation events. Specific serine residues within the regulatory domains, such as those identified between Ala796 and Ser815, can be phosphorylated by other kinases including protein kinase A (PKA) and Ca²⁺/calmodulin-dependent protein kinase II (CaMKII). Phosphorylation at these sites can alter the affinity of smMLCK for Ca²⁺/calmodulin or affect its catalytic efficiency. Autophosphorylation of smMLCK has also been reported to enhance its enzymatic activity (gao2001myosinlightchain pages 6-7, hirano2003proteinkinasenetwork pages 1-2).

Moreover, smMLCK exhibits a non-catalytic function through its actin-binding domains. Binding to actin not only assists in localizing the kinase within the cell but also plays a role in organizing the cytoskeletal network and modulating cell adhesion and motility. This kinase-independent mechanism has been implicated in the regulation of cell migration, where smMLCK serves as a scaffold linking integrins and the actin cytoskeleton (chen2014myosinlightchain pages 7-9). Thus, regulation of smMLCK occurs at both the biochemical level – via Ca²⁺/calmodulin-mediated activation and phosphorylation – and through spatial organization mediated by its interaction with cytoskeletal elements (chan2008identificationofcardiacspecific pages 9-10).

1. Function  
   smMLCK plays a central role in smooth muscle physiology by directly phosphorylating the regulatory light chain of myosin II, a modification that is essential for initiating contractile force generation. Phosphorylation of MLC20 induces conformational changes that increase myosin ATPase activity and promote actin-myosin interactions, thereby driving smooth muscle contraction. This activity is critical in a variety of tissues, including airway smooth muscle, vascular smooth muscle, and gastrointestinal smooth muscle (ammit2000smoothmusclemyosinlightchain pages 3-5, shen2010myosinlightchain pages 1-1).

In the respiratory system, increased smMLCK content has been observed in sensitized airways, which correlates with airway hyperreactivity, a hallmark of asthma. The abundance of smMLCK in airway smooth muscle suggests its importance in regulating tonic contraction and airway resistance under both physiological and pathophysiological conditions (ammit2000smoothmusclemyosinlightchain pages 3-5).

Beyond its canonical role in triggering contraction through MLC phosphorylation, smMLCK is also involved in modulating cytoskeletal dynamics independent of its kinase activity. Studies have demonstrated that smMLCK contributes to cell migration by serving as a scaffold that organizes actin filaments and forms complexes with integrins, vinculin, and other cytoskeletal proteins. This kinase-independent function affects cellular processes such as membrane tension, the formation of focal adhesions, and lamellipodial extension, thereby influencing cell motility and morphology (chen2014myosinlightchain pages 3-4, chen2014myosinlightchain pages 7-9).

smMLCK additionally has a role in the regulation of endothelial and vascular permeability. Through its influence on actomyosin contractility and cytoskeletal reorganization, smMLCK contributes to the maintenance of the endothelial barrier, and alterations in its activity have been implicated in inflammatory responses that lead to increased vascular permeability and leukocyte diapedesis (gao2001myosinlightchain pages 1-2, martinsen2014regulationofcalcium pages 8-9).

Furthermore, in the nervous system, smMLCK has been shown to control the growth initiation of astrocytic processes in culture and is involved in transmitter release at synapses in sympathetic ganglion cells, indicating a role in neuronal function and intercellular communication (information provided in the protein function description). Thus, smMLCK is a multifunctional enzyme whose primary activity in phosphorylating myosin regulatory light chains underlies smooth muscle contraction, while its non-catalytic interactions with cytoskeletal and adhesion proteins mediate additional cellular processes including migration, barrier function, and potentially neuronal modulation (chan2008identificationofcardiacspecific pages 1-2, chen2014myosinlightchain pages 11-12, milewicz2017alteredsmoothmuscle pages 1-2).

1. Other Comments  
   In addition to its well‐characterized roles in muscle contraction and cytoskeletal regulation, smMLCK is a target for a variety of pharmacological inhibitors, including small-molecule compounds such as ML-7, which have been used experimentally to attenuate its kinase activity. Inhibition of smMLCK can reduce contractile force, lower airway resistance, and affect cell migration by altering actomyosin kinetics (wilson2005integrinlinkedkinaseis pages 6-7). Furthermore, genetic studies have revealed that mutations or polymorphisms in the MYLK gene, particularly those that impair smMLCK function or expression, are associated with diseases such as thoracic aortic aneurysms and dissections as well as severe asthma, underscoring its clinical relevance in both cardiovascular and respiratory pathologies (milewicz2017alteredsmoothmuscle pages 4-5, ammit2000smoothmusclemyosinlightchain pages 3-5).

smMLCK’s dual functionality – its classical kinase-driven phosphorylation of myosin regulatory light chains and its kinase-independent role in organizing the actin cytoskeleton – makes it a critical participant in diverse physiological processes, ranging from the control of vascular tone and airway resistance to the regulation of cell migration and endothelial barrier integrity. In the context of inflammation, smMLCK activity influences leukocyte diapedesis and vascular permeability, further establishing its importance as an effector of the inflammatory response (information provided in the protein function description, chen2014myosinlightchain pages 11-12).

Additionally, beyond smooth muscle, smMLCK or its alternatively spliced variants have been detected in cardiac and skeletal muscles at lower levels, although their functional roles in these tissues differ considerably from those in smooth muscle. The independent expression of the telokin domain, a segment originally derived from the C-terminal end of smMLCK, also suggests that proteolytic processing and alternative splicing add further layers of regulation and functional specialization to the MYLK gene products (herring2000smoothmusclemyosin pages 8-9, chan2008identificationofcardiacspecific pages 10-14).

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