1. Phylogeny  
   SRPK3, also designated as MSSK1 or STK23, is a member of the serine/arginine protein kinase (SRPK) family within the CMGC group of kinases. This kinase family is evolutionarily conserved across eukaryotes and comprises several members, including SRPK1 and SRPK2, which are expressed ubiquitously, and SRPK3, which is distinguished by its tissue‐restricted expression. SRPK3 orthologs have been identified in vertebrates and invertebrates, and functional studies in organisms such as Drosophila have revealed homologous kinases (e.g., Drosophila SRPK79D) that share similar modular kinase domains with an intervening spacer sequence (nieratschker2009bruchpilotinribbonlike pages 8-10). The conserved bipartite catalytic domain—split by a non‐conserved spacer region that modulates subcellular localization—is a hallmark of this family and is found in SRPK3 as well (zhou2013regulationofsplicing pages 5-7). In mammals, SRPK3 exhibits a distinct phylogenetic niche by being predominantly expressed in muscle tissues, a feature that differentiates it from its more broadly expressed relatives and underscores its potentially specialized roles in muscle development and function (araki2023targetingpremrnasplicing pages 4-6, hogg2023functionsofsrpkclkanddyrkkinasesin pages 12-14).
2. Reaction Catalyzed  
   SRPK3 catalyzes a prototypical serine/threonine kinase reaction wherein the gamma-phosphate group is transferred from adenosine triphosphate (ATP) to specific serine residues located within arginine/serine-rich (RS) domains of substrate proteins. The general chemical reaction can be summarized as follows:  
     ATP + [protein]–L-serine → ADP + [protein]–L-serine phosphate + H⁺  
   This reaction results in the phosphorylation of RS domain-containing substrates, such as the splicing factor SRSF1 and the lamin-B receptor (LBR), thereby modulating their function and intracellular localization (zheng2023serinearginineproteinkinases pages 1-2, zhou2013regulationofsplicing pages 1-2).
3. Cofactor Requirements  
   The kinase activity of SRPK3 depends on the presence of divalent metal ions that facilitate the proper binding of ATP to the active site. In particular, Mg²⁺ serves as an essential cofactor by coordinating with ATP to stabilize its interaction with the kinase domain. The presence of Mg²⁺ is critical for the phosphoryl transfer reaction that underlies the catalytic mechanism of SRPK3 (zheng2023serinearginineproteinkinases pages 1-2, araki2023targetingpremrnasplicing pages 4-6).
4. Substrate Specificity  
   SRPK3 exhibits a high degree of substrate specificity for proteins containing RS domains, which are rich in arginine/serine dipeptides. The enzyme selectively phosphorylates serine residues embedded in these motifs, a modification that is paramount for the regulation of splicing factor activity. In vitro experiments have demonstrated that SRPK3 targets substrates such as the SR splicing factor SRSF1 and the lamin-B receptor (LBR) (information section; naro2013phosphorylationmediatedregulationof pages 3-5, zheng2023serinearginineproteinkinases pages 13-14). The consensus substrate motif for SRPK3 is defined by repetitive RS dipeptides, which provide the structural context required for recognition and processive phosphorylation of its targets.
5. Structure  
   SRPK3 is characterized by a conserved architecture typical of the SRPK family. Its primary structure consists of a central kinase domain that is split into two lobes by a large, non-conserved spacer region. The N-terminal lobe generally comprises predominantly β-strands, while the C-terminal lobe is rich in α-helices; together, these lobes create a catalytic cleft in which ATP binds and phosphoryl transfer occurs. The unique spacer insert plays a critical role in regulating subcellular localization, as it is implicated in retaining SRPK3 in the cytoplasm under resting conditions (zhou2013regulationofsplicing pages 5-7, tecchio2016developmentofheterocyclica pages 27-31).

Additional structural features include an activation loop that must adopt an open conformation to allow substrate access, as well as a conserved C-helix and hydrophobic spine that contribute to kinase stability and catalysis. A MAP kinase insert is often present in SRPKs and creates a docking groove that orients RS domain-containing substrates for efficient, processive phosphorylation. Although no high-resolution crystal structure specific to SRPK3 is available in the literature provided, homology with other SRPK family members supports the presence of these hallmark structural elements (araki2023targetingpremrnasplicing pages 4-6, zhou2013regulationofsplicing pages 5-7).

1. Regulation  
   Regulation of SRPK3 occurs primarily at the level of subcellular localization and through post-translational modifications. The non-conserved spacer domain is a key determinant of the enzyme’s cytoplasmic retention; alterations or deletions within this region can lead to aberrant nuclear translocation. This spatial regulation is critical, as SRPK3 must phosphorylate its substrates in the appropriate cellular compartment. Under basal conditions, SRPK3 is predominantly found in the cytoplasm; however, cellular signals, such as those mediated by growth factor stimulation and activation of the Akt pathway, can trigger modifications that promote its nuclear entry (zheng2023serinearginineproteinkinases pages 14-15, zhou2013regulationofsplicing pages 5-7).

Furthermore, SRPK3 is constitutively active, as is characteristic of the SRPK family, yet its activity can be fine-tuned by autophosphorylation and interactions with molecular chaperones—including Hsp70 and Hsp90—which assist in maintaining its proper conformation and subcellular distribution (prescott2012regulationofserinearginine pages 65-70). Although direct data for SRPK3 are limited in the citations provided, the regulatory paradigms established for SRPK1 and SRPK2 are considered applicable to SRPK3 owing to the high degree of sequence and structural conservation (zheng2023serinearginineproteinkinases pages 14-15, prescott2012regulationofserinearginine pages 65-70).

1. Function  
   SRPK3 plays an essential role in the regulation of pre-mRNA splicing by phosphorylating serine/arginine-rich (SR) proteins. Its catalytic activity on RS domains is critical for modulating the subcellular localization and activity of splicing factors. In vitro studies have confirmed that SRPK3 phosphorylates substrates such as SRSF1 and the lamin-B receptor (LBR), modifications which are necessary for proper spliceosome assembly and splicing regulation (information section; zheng2023serinearginineproteinkinases pages 1-2, naro2013phosphorylationmediatedregulationof pages 3-5).

In addition to its role in splicing regulation, SRPK3 is required for normal muscle development. Its expression is largely confined to skeletal muscle tissue, and experimental studies have shown that deletion or mutation of SRPK3 leads to phenotypes associated with impaired muscle development and function. For example, genetically engineered mouse models lacking SRPK3 exhibit defects in B lymphocyte development and humoral immune responsiveness, indicating that the kinase’s regulatory functions may extend to the modulation of alternative splicing events that influence mitochondrial biogenesis and metabolic pathways (arends2019srpk3regulatesalternative pages 12-16, arends2019srpk3regulatesalternative pages 16-21, araki2023targetingpremrnasplicing pages 4-6, pastor2021interplaybetweencmgc pages 3-5).

The biological functions of SRPK3 include not only the regulation of canonical pre-mRNA splicing but also the modulation of cell signaling pathways through the alternative splicing of genes involved in muscle metabolism and immune cell differentiation. These roles underscore the importance of SRPK3 in both muscle physiology and potentially in the immune system, where precise control of alternative splicing is requisite for normal cellular function (hogg2023functionsofsrpkclkanddyrkkinasesin pages 12-14).

1. Other Comments  
   At present, selective inhibitors that target SRPK3 specifically have not been reported. However, several pan-SRPK inhibitors—such as SRPIN340, SPHINX31, and SRPKIN-1—have been developed and studied for their ability to modulate the activity of SR protein kinases in general. These compounds have been evaluated in the context of altered splicing in cancers and viral infections, though their effects on SRPK3 remain to be fully elucidated (tecchio2016developmentofheterocyclica pages 27-31, pastor2021interplaybetweencmgc pages 3-5, siriwath2020cellularactivitiesof pages 34-39).

Disease associations for SRPK3 have been reported in studies investigating its role in muscle development and immune function. Loss-of-function mutations or deletion of SRPK3 in experimental models have been linked to impaired muscle growth, altered B cell development, and defects in antibody production, which imply a potential connection with immunodeficiency and muscle-related pathologies (arends2019srpk3regulatesalternative pages 12-16, bustos2020functionaldiversificationof pages 12-15). These observations emphasize the necessity for further research into SRPK3’s substrate repertoire, regulatory mechanisms, and the possible development of targeted therapies for diseases stemming from its dysregulation.

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