1. Phylogeny  
   SRPK2 is a member of the serine/arginine protein kinase family, which is placed within the larger CMGC group of serine/threonine kinases that also includes MAPKs, CDKs, GSKs, DYRKs, and CK2α. Orthologs of SRPK2 are evolutionarily conserved from yeast through invertebrates to mammals, and the duplication events that gave rise to SRPK1 and SRPK2 occurred early in the evolution of metazoans. SRPK2 and its paralog SRPK1 share a high degree of sequence conservation in their catalytic domains, yet diverge in their noncatalytic regions such as the unique spacer insert and N-terminal sequences that may contribute to tissue-specific regulation; for example, SRPK2 is highly expressed in neuronal tissues whereas SRPK1 has a more ubiquitous expression (wang1998srpk2adifferentially pages 1-2, zhou2013regulationofsplicing pages 16-18, long2019distinctmechanismsgovern pages 1-1). This conservation places SRPK2 within a core repertoire of splicing regulatory enzymes conserved across eukaryotes, reflecting its fundamental role in RNA processing.
2. Reaction Catalyzed  
   SRPK2 catalyzes the transfer of a phosphate group from ATP to specific serine residues within target proteins that contain arginine/serine-rich (RS) domains. The chemical reaction can be summarized as:  
     ATP + [protein]–(L-serine) → ADP + [protein]–(L-serine)-phosphate + H⁺  
   This phosphorylation reaction is critical for modulating the activity, protein–protein interactions, and subcellular localization of SR proteins, which are essential for proper spliceosome assembly and function (hong2011thenterminalfragment pages 1-2, zheng2023serinearginineproteinkinases pages 9-11).
3. Cofactor Requirements  
   The catalytic activity of SRPK2 is dependent on divalent cations, with Mg²⁺ being the principal cofactor that facilitates ATP binding and proper orientation of the phosphate group within the active site. Mg²⁺ is required to stabilize the transition state during the phosphoryl transfer reaction, a common feature shared by serine/threonine kinases (wang1998srpk2adifferentially pages 1-2, zheng2023serinearginineproteinkinases pages 9-11).
4. Substrate Specificity  
   SRPK2 displays high substrate specificity for proteins that contain RS domains, recognizing and phosphorylating serine residues arranged in arginine/serine (RS) dipeptide repeats. The kinase has a strong preference for serines that are immediately preceded or followed by arginine residues rather than lysine, as the steric and electrostatic requirements of the active site demand a specific basic context. Substrate recognition studies indicate that the enzyme efficiently phosphorylates key splicing factors such as ASF/SF2 (SRSF1), SRSF2, SRSF3, and ACIN1, among others. These splicing factors contain RS domains that serve as platforms for the assembly of spliceosomal components, and the phospho-modification alters their intranuclear distribution and binding affinities. Mutagenesis experiments have further demonstrated that substitution of the arginine residues with lysine or other noncanonical residues leads to a marked decrease in phosphorylation efficiency (hong2011thenterminalfragment pages 1-2, wang1998srpk2adifferentially pages 5-6, long2019distinctmechanismsgovern pages 2-3, zheng2023serinearginineproteinkinases pages 9-11).
5. Structure  
   The overall structure of SRPK2 is defined by a dual catalytic domain architecture that is separated by a unique spacer sequence. The two kinase domains share significant homology with those of SRPK1; however, the interdomain spacer in SRPK2 may function as an autonomous regulatory region that influences subcellular localization and interaction with other proteins. The N-terminal portion of the protein often contains a stretch of proline-rich sequences, which are postulated to mediate interactions with WW domain–containing regulatory proteins. In addition, high-resolution data from homologous kinases and structural models suggest that the catalytic domains harbor a conserved ATP-binding pocket with key residues—including a hinge region formed by Leu168 and Gly169 equivalents—that are critical for binding ATP and inhibitors. An electronegative docking groove is present within the catalytic domains, and this feature facilitates processive phosphorylation of RS substrates. Notably, subtle structural differences in the hydrophobic pocket region, when compared to SRPK1, may alter inhibitor binding and catalytic efficiency (wang1998srpk2adifferentially pages 1-2, wang1998srpk2adifferentially pages 11-12, zheng2023serinearginineproteinkinases pages 9-11, long2019distinctmechanismsgovern pages 1-1). This structural organization, including the conserved catalytic motifs and unique spacer insert, underpins the enzyme’s ability to phosphorylate multiple serine residues within a target RS domain in a processive manner (hong2011thenterminalfragment pages 1-2, long2019distinctmechanismsgovern pages 1-1, zhou2013regulationofsplicing pages 16-18).
6. Regulation  
   SRPK2 is subject to multifaceted regulatory mechanisms that include direct phosphorylation by upstream kinases, caspase-mediated cleavage, and interactions with regulatory proteins. One prominent regulatory pathway involves Akt, which phosphorylates SRPK2 at a specific threonine residue located in the variable spacer region (Thr-492). This phosphorylation event is crucial for modulating SRPK2’s activity and preventing its apoptotic cleavage; indeed, phosphorylated SRPK2 is stabilized by binding to 14-3-3 proteins, which in turn protect the kinase from degradation (jang2009interactionofaktphosphorylated pages 4-5, hong2011thenterminalfragment pages 1-2). In contrast, under apoptotic stimuli, SRPK2 is cleaved by caspases at Asp-139 and Asp-403, generating an N-terminal fragment that translocates into the nucleus and contributes to apoptotic signaling by affecting chromatin condensation and splicing factor redistribution (hong2011thenterminalfragment pages 8-9). Moreover, extracellular signals such as EGF activate the PI3K/Akt pathway, thereby indirectly increasing SRPK2 phosphorylation and promoting its nuclear translocation, which links growth factor signaling to alternative splicing regulation (zhou2012theaktsrpksraxis pages 4-5, zhou2013regulationofsplicing pages 16-18). Additional regulatory inputs may involve autophosphorylation events and dynamic interactions with molecular chaperones, which serve to modulate both kinase activity and subcellular compartmentalization (jang2009interactionofaktphosphorylated pages 4-5, pastor2021interplaybetweencmgc pages 5-6).
7. Function  
   SRPK2 plays a central role in the regulation of pre-mRNA splicing by phosphorylating serine residues within RS domains of a wide array of splicing factors. This phosphorylation event modulates the activity, localization, and protein–protein interactions of SR proteins, thereby influencing both constitutive and alternative splicing decisions. A crucial function of SRPK2 is its involvement in spliceosomal B complex formation; for instance, phosphorylation of DDX23/PRP28 by SRPK2 is essential for the proper integration of the U4/U6-U5 tri-snRNP into the functional spliceosome (hong2011thenterminalfragment pages 1-2, zhou2013regulationofsplicing pages 19-20). In neuronal cells, SRPK2 contributes to apoptotic pathways by phosphorylating SRSF2; this action suppresses p53 phosphorylation, resulting in the upregulation of cyclin-D1, which is a key event in promoting neuronal apoptosis. Similarly, SRPK2 phosphorylates ACIN1 and facilitates its redistribution from nuclear speckles to the nucleoplasm, selectively enhancing cyclin A1 expression without affecting cyclin A2 levels (hong2011thenterminalfragment pages 1-2, radhakrishnan2016dysregulationofsplicing pages 1-2, jang2009interactionofaktphosphorylated pages 4-5). Such functions underscore the dual regulatory roles of SRPK2 in both splicing regulation and cell cycle control. Expression analyses have revealed a tissue-specific pattern wherein SRPK2 is highly enriched in the brain relative to other tissues, indicative of specialized functions in neuronal RNA processing and apoptosis (wang1998srpk2adifferentially pages 1-2, caetano2022impairedexpressionof pages 1-2). Furthermore, by modulating the phosphorylation state of splicing factors, SRPK2 indirectly influences alternative splicing outcomes that are critical in a variety of biological contexts, including differentiation, development, and responses to stress (pastor2021interplaybetweencmgc pages 11-12, long2019distinctmechanismsgovern pages 1-1).
8. Other Comments  
   Several small molecule inhibitors targeting the SRPK family have been identified, including SRPIN340, SPHINX31, and the irreversible inhibitor SRPKIN-1, which inhibit kinase activity through competitive binding at the ATP pocket; these inhibitors have been shown to affect splicing patterns by altering the phosphorylation status of SR proteins (zheng2023serinearginineproteinkinases pages 13-14, caetano2022impairedexpressionof pages 1-2). Overexpression of SRPK2 has been linked to oncogenic processes, with elevated levels observed in various cancers such as pancreatic, non-small cell lung, and prostate cancer, and its activity is associated with poor prognosis in these settings (radhakrishnan2016dysregulationofsplicing pages 1-2, nikas2019serinearginineproteinkinase pages 12-14). Additionally, in neuronal contexts, aberrant SRPK2 activity has been implicated in apoptosis and neurodegeneration, particularly via dysregulation of cell cycle proteins such as cyclin-D1 and cyclin A1 (hong2011thenterminalfragment pages 1-2, jang2009interactionofaktphosphorylated pages 4-5). Emerging evidence also suggests that SRPK2 may play roles in the regulation of viral replication; phosphorylation of viral proteins by SRPK2 has been reported to influence viral RNA packaging and capsid assembly (zheng2023serinearginineproteinkinases pages 8-9). Continued efforts to develop more selective and potent inhibitors of SRPK2, along with detailed characterization of its substrate interactions and regulatory modifications, remain a priority in both cancer and antiviral research domains (zheng2023serinearginineproteinkinases pages 13-14, pastor2021interplaybetweencmgc pages 11-12).
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