1. Phylogeny  
   Ribosomal protein S6 kinase beta-1 (RPS6KB1), commonly designated as S6K1, is a member of the AGC kinase family, a group that includes protein kinase A (PKA), protein kinase G (PKG), and protein kinase C (PKC). Phylogenetic analysis indicates that S6K1 is conserved throughout eukaryotic evolution and is present in all mammalian species, as part of an evolutionarily ancient core of TOR pathway genes that date back to the Last Eukaryotic Common Ancestor (LECA). Moreover, S6K1 shares evolutionary relationships with related kinases such as p90 ribosomal S6 kinase (RSK) and serum- and glucocorticoid-regulated kinase (SGK), with evidence indicating that both S6K and RSK originated from a gene duplication event in the common ancestor of animals and fungi (magnuson2012regulationandfunction pages 2-3, sridharan2020distinctrolesof pages 1-3). Comparative analyses demonstrate that orthologs of S6K1 exist across diverse species including humans, mice, Xenopus, and yeast, underscoring its essential and conserved role in coupling nutrient and growth factor signals to cell proliferation and metabolism (pende2014ribosomalproteins6 pages 1-3).
2. Reaction Catalyzed  
   RPS6KB1 catalyzes the transfer of a phosphate group from ATP to protein substrates containing serine or threonine residues. The reaction can be summarized as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (template). This phosphorylation reaction is vital for initiating a cascade of events that regulate protein synthesis and cell proliferation (magnuson2012regulationandfunction pages 6-7).
3. Cofactor Requirements  
   The catalytic activity of S6K1 is dependent on divalent metal ions. In particular, Mg²⁺ is required as a cofactor to facilitate ATP binding and proper orientation of the phosphate groups for transfer to the substrate (template, fumagalli2022s6kinase1 pages 3-4).
4. Substrate Specificity  
   S6K1 exhibits a substrate specificity that is defined by a consensus sequence motif. The typical motif recognized by S6K1 is RxRxxp[ST], where “p[ST]” represents the phosphorylated serine or threonine residue. This motif guides S6K1 to phosphorylate various key substrates involved in translation and signal transduction, such as the ribosomal protein S6, eukaryotic initiation factor 4B (eIF4B), and eukaryotic elongation factor 2 kinase (eEF2K) (template, roux2018signalingpathwaysinvolved pages 11-13).
5. Structure  
   S6K1 contains a central catalytic kinase domain that is flanked by regulatory regions which are largely intrinsically disordered. One key structural feature is the conserved TOR signaling (TOS) motif located near the N-terminus; this motif, typically an FDIDL sequence in human S6K1, is crucial for binding to the RAPTOR subunit of mTORC1, thereby facilitating its activation by mTORC1 (magnuson2012regulationandfunction pages 3-4, sunami2010structuralbasisof pages 1-2). The kinase domain itself is organized in a bilobal fold with an N-terminal lobe that predominantly binds ATP and a C-terminal lobe that coordinates substrate binding; within this enzyme, the activation loop requires phosphorylation for full activation. Crystal structural data reveal that phosphorylation of residues in the activation loop, such as Thr252 (in the case of human p70S6K1), drives conformational changes that allow proper substrate access, while the hydrophobic motif is phosphorylated at residues such as Thr389, stabilizing the active conformation (sunami2010structuralbasisof pages 7-8, magnuson2012regulationandfunction pages 10-11). In addition, S6K1 features a C-terminal region which, in some isoforms, contains a PDZ-binding domain, and in the longer p85 isoform, a nuclear localization signal (NLS) is present; these structural variations contribute to the differential subcellular localization of its isoforms (malanchuk2024investigatingtheregulation pages 1-2, pende2014ribosomalproteins6 pages 7-10). Residues involved in coordinating ATP and substrate contacts are highly conserved, and the active site displays a DFG-in conformation typical of serine/threonine kinases, which is imperative for catalytic activity (sunami2010structuralbasisof pages 8-9).
6. Regulation  
   S6K1 activation is regulated by a multi-step hierarchical phosphorylation mechanism. Under conditions of growth factor stimulation and nutrient abundance, mTORC1 phosphorylates S6K1 at a critical hydrophobic motif residue—Thr389 in S6K1—which acts as a priming event that creates a docking site for phosphoinositide-dependent kinase-1 (PDK1). Subsequent phosphorylation of the activation loop residue Thr229 by PDK1 completes the activation process (magnuson2012regulationandfunction pages 3-4, sridharan2020distinctrolesof pages 3-5). Additional phosphorylation events occur at sites within the C-terminal autoinhibitory region, including modifications by proline-directed kinases such as cyclin-dependent kinases and MAPK, which relieve autoinhibition and can modulate substrate specificity (fumagalli2022s6kinase1 pages 3-4, sridhar2022targetingrps6k1for pages 1-4). S6K1 is also subject to negative regulation by phosphatases such as PP2A, which dephosphorylate key activating residues, and by ubiquitination events that target S6K1 for proteasomal degradation; acetylation has also been reported to affect its protein stability (magnuson2012regulationandfunction pages 15-16, thiriet2013cytoplasmicproteinserinethreonine pages 63-66). The specificity of mTORC1-mediated phosphorylation is mediated by the TOS motif interaction with RAPTOR, ensuring that S6K1 activation is tightly coupled to upstream signals such as the availability of amino acids and energy status (pende2014ribosomalproteins6 pages 3-5, kim2009regulationandlocalization pages 10-11). Furthermore, differential phosphorylation and subcellular shuttling of distinct isoforms (p70 versus p85) modulate the precise outcomes of S6K1 signaling in different cellular contexts (sridhar2022targetingrps6k1for pages 7-11, malanchuk2024investigatingtheregulation pages 1-2).
7. Function  
   S6K1 plays a central role in mediating translational control, cell growth, and cell cycle progression through its action downstream of the mTORC1 pathway. By phosphorylating substrates such as ribosomal protein S6 (RPS6), eIF4B, and eEF2K, S6K1 promotes mRNA translation, thereby enhancing protein synthesis and cell growth (fumagalli2022s6kinase1 pages 3-4, pende2014ribosomalproteins6 pages 5-7). In addition to ribosome biogenesis, S6K1 phosphorylates the translational repressor PDCD4, leading to its ubiquitination and subsequent degradation, which facilitates cap-dependent translation initiation. The kinase further phosphorylates components involved in the pioneer round of translation, such as POLDIP3/SKAR, thereby ensuring efficient mRNA processing and translation of newly spliced transcripts (sridhar2022targetingrps6k1for pages 7-11). Beyond its canonical role in protein synthesis, S6K1 contributes to cell survival through its phosphorylation of the pro-apoptotic protein BAD, thereby impairing its ability to induce apoptosis (fumagalli2022s6kinase1 pages 3-4). Furthermore, S6K1 is implicated in metabolic regulation, for example by phosphorylating IRS1 on multiple serine residues and promoting its degradation—this regulation is associated with the development of insulin resistance (fumagalli2022s6kinase1 pages 10-10, sridhar2022targetingrps6k1for pages 4-7). S6K1 also plays a part in feedback inhibition of mTOR signaling; it phosphorylates components such as mTOR, RICTOR, and SIN1, thereby modulating the activity of mTORC2 and downstream AKT signaling (magnuson2012regulationandfunction pages 17-17, sridhar2022targetingrps6k1for pages 11-13). Additionally, S6K1 phosphorylates the pyrimidine biosynthesis enzyme CAD to regulate de novo nucleotide synthesis, underscoring its role in metabolic reprogramming (fumagalli2022s6kinase1 pages 10-10). In various cell types, the distinct isoforms of S6K1 (p70 and p85) exhibit differential subcellular localizations that allow S6K1 to integrate signals emanating from both cytosolic and nuclear compartments, thereby affecting processes such as cell proliferation, migration, and differentiation (malanchuk2024investigatingtheregulation pages 1-2, sridhar2022targetingrps6k1for pages 7-11).
8. Other Comments  
   Multiple inhibitors targeting the S6K1 signaling axis have been developed; among the most characterized is the ATP-competitive inhibitor PF-4708671, which has been shown to selectively inhibit S6K1 activity with approximately 20-fold greater potency relative to closely related kinases such as RSK1 and MSK1 (sridhar2022targetingrps6k1for pages 1-4). Other investigational compounds, including LY2584702 and TAS0612, exhibit the capacity to inhibit S6K1 and have entered clinical trials for cancer therapy, particularly in settings of aberrant mTOR pathway activation and PTEN loss (sridhar2022targetingrps6k1for pages 11-13). S6K1 dysregulation has been associated with oncogenesis, metabolic disorders, and insulin resistance as a consequence of its role in phosphorylating IRS1, BAD, and other regulators of cell survival and metabolic homeostasis (fumagalli2022s6kinase1 pages 10-10, yi2021ribosomalproteins6 pages 17-18). Disease associations also extend to the context of cardiac hypertrophy, renal hypertrophy, and aging, with genetic ablation studies in mice demonstrating phenotypes that mimic aspects of caloric restriction and improved insulin sensitivity (pende2014ribosomalproteins6 pages 15-17, magnuson2012regulationandfunction pages 15-16). No specific disease-causing missense mutations in RPS6KB1 have been uniformly reported; however, alterations in gene copy number or upstream signaling components affecting S6K1 activity have been linked to various cancer types (yi2021ribosomalproteins6 pages 41-43). In addition, the protein’s involvement in feedback inhibition of mTORC2 and modulation of cytoskeletal dynamics via neurabin interaction highlights its broader significance in both oncogenic processes and cell motility (fumagalli2022s6kinase1 pages 3-4, lapenas2023ofthevulnerability pages 173-176).
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