**1. Phylogeny:**  
Ribosomal protein S6 kinase beta-1 (RPS6KB1), commonly referred to as p70 ribosomal S6 kinase 1, belongs to the AGC family of serine/threonine kinases and is a central component of the TOR signaling pathway. It is evolutionarily conserved across eukaryotes and can be traced to the Last Eukaryotic Common Ancestor (LECA) or earlier, with orthologs present in all mammalian species and broadly distributed among metazoans (bradley2019evolutionofprotein pages 1-2). RPS6KB1 is part of an ancestral core set of TOR pathway genes that also includes other AGC kinases such as phosphoinositide‐dependent kinase 1 (PDK1), protein kinase B (PKB/AKT), p90 ribosomal S6 kinase (RSK), and serum‐ and glucocorticoid‐regulated kinase (SGK) (andrade2011eukaryoticproteinkinases pages 14-15). Gene duplication events in the common ancestor of animals and fungi led to the emergence of both S6 kinases and their closely related RSK kinases, with both groups being regulated by PDK1, while the activation of S6K1 is further controlled by the mechanistic target of rapamycin complex 1 (mTORC1) (briedis2008thedistributionand pages 26-31).

**2. Reaction Catalyzed:**  
RPS6KB1 catalyzes the phosphorylation process in which the gamma-phosphate group of ATP is transferred to specific serine or threonine residues on substrate proteins. The chemical reaction is represented as:  
ATP + [protein]-(L-serine or L-threonine) = ADP + [protein]-(L-serine/threonine)-phosphate + H+ (briedis2008thedistributionand pages 205-208).

**3. Cofactor Requirements:**  
The catalytic activity of RPS6KB1 is dependent on Mg²⁺ ions. These divalent metal ions coordinate with the ATP molecule to facilitate the proper transfer of the phosphate group to the substrate (briedis2008thedistributionand pages 189-193).

**4. Substrate Specificity:**  
RPS6KB1 displays a substrate specificity characterized by a preference for protein substrates containing an RxRxxp[ST] consensus sequence. In this motif the phospho-acceptor residue can be either serine or threonine, and the residues at the –5 and –3 positions are most frequently arginine. This pattern of substrate recognition is well established for serine/threonine kinases within the human kinome based on studies such as the substrate specificity atlas for serine/threonine kinases (bradley2019evolutionofprotein pages 8-9, bdzhola2025coexpressionofthe pages 9-10).

**5. Structure:**  
RPS6KB1 is composed of a central kinase catalytic domain that is surrounded by intrinsically disordered regulatory regions. A key structural element is the conserved TOR signaling (TOS) motif located near the N-terminus; in human S6K1 this motif, consisting of the FDIDL sequence at residues 5–9, is essential for binding to the RAPTOR subunit of mTORC1, a step crucial for its phosphorylation and activation (ikuta2007crystalstructuresof pages 1-2, briedis2008thedistributionand pages 26-31). The kinase domain of RPS6KB1 includes well-defined regulatory features such as the activation loop (T-loop), which is phosphorylated by PDK1 at T229, and a hydrophobic motif that is phosphorylated at T389 by mTORC1, events that together are required for full enzymatic activity (pearce2010characterizationofpf4708671 pages 14-16). Additionally, RPS6KB1 contains a C-terminal PDZ-binding domain that distinguishes it from its paralog S6K2, which instead possesses a proline-rich region followed by a nuclear localization signal (bradley2019evolutionofprotein pages 25-25). Structural evidence obtained from experimental crystallography and supported by AlphaFold modeling confirms the presence of a catalytic core featuring a well-defined C-helix and hydrophobic spine, both of which are critical for substrate binding and catalysis (ikuta2007crystalstructuresof pages 1-2, pearce2010characterizationofpf4708671 pages 14-16).

**6. Regulation:**  
The regulation of RPS6KB1 is predominantly achieved through a series of well-orchestrated phosphorylation events. Full activation of the kinase requires sequential phosphorylation steps. First, mTORC1 phosphorylates the hydrophobic motif (specifically at T389 in S6K1), creating a docking site for PDK1. Subsequently, PDK1 phosphorylates the activation loop (at T229 in S6K1) to relieve autoinhibition and initiate catalytic activity (ikuta2007crystalstructuresof pages 10-10, lai2015investigationsofthe pages 9-17). Under nutrient-deprived conditions, the inactive form of RPS6KB1 remains sequestered in a complex with the EIF3 translation initiation machinery; upon mitogenic stimulation and consequent mTORC1-mediated phosphorylation, RPS6KB1 dissociates from the EIF3 complex and becomes active (ahmed2019directimagingof pages 13-13, murphy2021theroleof pages 111-114). In addition to these primary regulatory phosphorylations, RPS6KB1 is involved in feedback regulation mechanisms within the mTOR signaling network. The kinase phosphorylates several mTOR complex components, including MAPKAP1/SIN1, MTOR, and RICTOR, which results in the inhibition of mTORC2 activity and downregulation of AKT1 signaling (bradley2019evolutionofprotein pages 18-19, cronin2023amechanisticapproach pages 27-34). Moreover, phosphorylation of DEPTOR by RPS6KB1 further contributes to the feedback regulation between mTORC1 and mTORC2 (bradley2019evolutionofprotein pages 21-22). RPS6KB1 also catalyzes phosphorylation of IRS1 on multiple serine residues, leading to its accelerated degradation and thereby mediating TNF-alpha-induced insulin resistance (murphy2021theroleof pages 15-21). Furthermore, phosphorylation of the pro-apoptotic protein BAD by RPS6KB1 represses its activity to promote cell survival, and phosphorylation of mitochondrial URI1 leads to its dissociation from the PPP1CC complex; the liberated PPP1CC is then able to dephosphorylate RPS6KB1 at Thr-412, providing a negative feedback mechanism on its anti-apoptotic function (ahmed2019directimagingof pages 13-13). In cells that lack a functional TSC1/2 complex, RPS6KB1 is constitutively active, resulting in the phosphorylation and inhibition of GSK3B (murphy2021theroleof pages 15-21).

**7. Function:**  
RPS6KB1 functions as a pivotal downstream effector of mTORC1 signaling in response to growth factors and nutrient availability. The kinase plays a central role in promoting cell proliferation, growth, and cell cycle progression primarily by regulating protein synthesis. It achieves this by phosphorylating several key substrates involved in translation initiation and elongation. These substrates include EIF4B, which is a component of the cap-binding complex, ribosomal protein S6 (RPS6), a component of the 40S ribosomal subunit, and eukaryotic elongation factor 2 kinase (EEF2K), whose inhibition augments the activity of eukaryotic elongation factor 2 (EEF2) for protein elongation (ahmed2019directimagingof pages 13-13, murphy2021theroleof pages 15-21). In addition to its role in protein synthesis, RPS6KB1 phosphorylates PDCD4, a negative regulator of EIF4A, thereby targeting PDCD4 for ubiquitination and proteolysis; it also phosphorylates POLDIP3/SKAR to promote the pioneer round of protein synthesis (ahmed2019directimagingof pages 13-13). Through phosphorylation of the pro-apoptotic protein BAD, RPS6KB1 diminishes apoptotic signaling, which supports cell survival. Moreover, the kinase mediates feedback inhibition of the mTOR signaling pathway by phosphorylating elements of the mTORC2 complex (MAPKAP1/SIN1, MTOR, RICTOR), resulting in the downregulation of AKT1 signaling (bradley2019evolutionofprotein pages 18-19, sridhar2022targetingrps6k1for pages 11-13). RPS6KB1 also targets IRS1, phosphorylating it on serine residues that trigger its degradation and contribute to insulin resistance. Beyond translational control and survival, RPS6KB1 influences metabolic pathways via phosphorylation of the pyrimidine biosynthesis enzyme CAD and by regulating fatty acid uptake through phosphorylation of EPRS (ahmed2019directimagingof pages 13-13).

**8. Other Comments:**  
Selective inhibition of RPS6KB1 has been a significant focus of drug discovery efforts owing to the kinase’s central role in cell growth, metabolism, and survival. One of the most notable inhibitors is PF-4708671, which has demonstrated approximately 20-fold selectivity for S6K1 over related kinases such as RSK and MSK; PF-4708671 inhibits S6K1 activity without affecting upstream phosphorylation events mediated by mTORC1 or PDK1 (pearce2010characterizationofpf4708671 pages 14-16, pearce2010characterizationofpf4708671 pages 16-17). TAS0612 is another compound that inhibits S6K along with other kinases including RSK and PKB and has shown growth inhibition effects in preclinical cancer models (pearce2010characterizationofpf4708671 pages 16-17). Dysregulation of RPS6KB1 activity is associated with pathological conditions, including various types of cancer, metabolic syndromes such as insulin resistance, and obesity (murphy2021theroleof pages 15-21). In addition, there is evidence linking RPS6KB1 function with cytoskeletal rearrangements via its potential interaction with neurabin, and its broad involvement in several cellular signaling cascades makes it a prime target for further therapeutic development. Several inhibitors such as LY2584702 have progressed into clinical trials, underscoring the ongoing interest in targeting RPS6KB1 for therapeutic benefit (sridhar2022targetingrps6k1for pages 11-13).

**9. References:**  
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