**Accepted name:** Ribosomal protein S6 kinase beta-1, also known as 70 kDa ribosomal protein S6 kinase 1, Ribosomal protein S6 kinase I, Serine/threonine‐protein kinase 14A, or p70 ribosomal S6 kinase alpha. Gene: RPS6KB1 (STK14A).

**Synonyms:** 70 kDa ribosomal protein S6 kinase 1; p70 ribosomal S6 kinase; S6K1; S6 kinase alpha.

1. Phylogeny  
   RPS6KB1 belongs to the AGC family of serine/threonine kinases, which also includes key regulators such as PDK1, PKB/AKT, p90 ribosomal S6 kinase (RSK), and SGK. Comparative genomic studies indicate that the AGC kinases as a group are evolutionarily ancient, their origins traceable to the Last Eukaryotic Common Ancestor (LECA) or earlier. In mammals, RPS6KB1 is ubiquitously expressed, and its orthologs are present in all tissues, underscoring its central role in growth‐promoting signaling pathways. The evolutionary history of S6K1 shows that gene duplication events gave rise to separate isoforms and to closely related proteins such as RSK, particularly in the ancestor of animals and fungi (karlsson2014clinicalpotentialof pages 116-117, lapenas2023ofthevulnerability pages 166-168, domanova2016unravelingkinaseactivation pages 12-13). This conserved phylogenetic profile situates RPS6KB1 as a member of an evolutionarily core set of TOR pathway genes that also include mTOR complex constituents and several AGC kinases.
2. Reaction Catalyzed  
   RPS6KB1 catalyzes an ATP-dependent transfer of a phosphate group to serine or threonine residues on substrate proteins. The reaction can be described by the following equation:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H+  
   This reaction forms the basis of its role in regulating various cellular processes downstream of mTOR signaling by controlling the phosphorylation state of proteins that influence translation and growth (beltman2022kinasecatalyzedlabelingto pages 318-322, tchevkina2012proteinphosphorylationas pages 19-21).
3. Cofactor Requirements  
   The kinase activity of RPS6KB1 requires the presence of divalent metal ions. Specifically, Mg²⁺ serves as an essential cofactor necessary for proper ATP binding and facilitates the efficient transfer of the phosphate group during the catalytic reaction. This requirement is a common characteristic among serine/threonine kinases and is critical for the enzymatic function of RPS6KB1 (lapenas2023ofthevulnerability pages 171-173, tchevkina2012proteinphosphorylationas pages 19-21).
4. Substrate Specificity  
   RPS6KB1 displays a well-defined substrate specificity that is dominated by a preference for sequences conforming to the consensus motif RxRxxp[ST]. In this motif, the arginine residues located at positions −5 and −3 relative to the phosphorylated serine or threonine are essential for substrate recognition. This substrate motif enables the kinase to selectively target proteins involved in the control of translation. Among the substrates phosphorylated by RPS6KB1 are ribosomal protein S6, eukaryotic initiation factor 4B (eIF4B), and eukaryotic elongation factor 2 kinase (EEF2K); these phosphorylation events facilitate both the initiation and elongation phases of protein synthesis (beltman2022kinasecatalyzedlabelingto pages 35-40, tchevkina2012proteinphosphorylationas pages 19-21).
5. Structure  
   The structure of RPS6KB1 is defined by a canonical AGC kinase domain organization. It features a central bi-lobal kinase domain that is flanked on both sides by intrinsically disordered regulatory regions. At the N-terminal region, RPS6KB1 contains a conserved TOR signaling (TOS) motif, typically encompassing an FDIDL sequence (residues 5–9 in human S6K1), which is critical for binding to the RAPTOR subunit of mTORC1; this interaction is essential for mTOR-mediated phosphorylation and subsequent enzyme activation (gerstenecker2021discoveryofa pages 9-10, julich2008skaranovel pages 15-19). The central kinase domain houses key catalytic features, including the activation loop (or T-loop) and a hydrophobic motif. Full catalytic function is attained by phosphorylation of the activation loop residue Thr229 by PDK1 and phosphorylation of the hydrophobic motif (Thr389 in S6K1) by mTORC1. Furthermore, RPS6KB1 is distinguished from its paralog S6K2 by possessing a C-terminal PDZ-binding domain, whereas S6K2 typically contains a C-terminal proline-rich region accompanied by a nuclear localization signal (khalil2024s6k2infocus pages 7-9, pende2014ribosomalproteins6 pages 1-3). This domain architecture is critical in mediating both conformational changes and interactions with other proteins involved in the regulation of translation and cell growth.
6. Regulation  
   Activation of RPS6KB1 is tightly regulated via multisite phosphorylation mechanisms that integrate signals from growth factors and nutrient availability. Under conditions of nutrient depletion, the kinase remains in an inactive state wherein it is sequestered by the EIF3 translation initiation complex. Upon mitogenic stimulation, mTORC1 becomes active and phosphorylates RPS6KB1 at its hydrophobic motif (Thr389 in S6K1), a modification that creates a docking site for PDK1. PDK1 subsequently phosphorylates the activation loop at Thr229, which is required for full kinase activation (hsu2011theidentificationof pages 71-75, majeed2019s6kinasea pages 17-20). Additional regulatory mechanisms involve feedback loops within the mTOR signaling network. Active RPS6KB1 phosphorylates components such as MAPKAP1/SIN1, mTOR, RICTOR, and DEPTOR, which in turn modulate mTORC2 and AKT1 activity through negative feedback regulation. Moreover, the kinase phosphorylates IRS1, leading to its ubiquitination and proteasomal degradation, a process that contributes to TNF-alpha-induced insulin resistance. Other post-translational modifications, including ubiquitination and acetylation, have been observed to affect the stability and activity of RPS6KB1, while phosphorylation of mitochondrial URI1 facilitates the release of the PPP1CC phosphatase that can dephosphorylate RPS6KB1 at Thr412, thus participating in an autoregulatory negative feedback loop (majeed2019s6kinasea pages 20-21, bdzhola2025coexpressionofthe pages 9-10, murphy2021theroleof pages 111-114, sridhar2022targetingrps6k1for pages 11-13, sridhar2022targetingrps6k1for pages 14-16).
7. Function  
   RPS6KB1 plays a central role as an effector of mTORC1 signaling, translating extracellular cues such as growth factor stimulation and nutrient availability into downstream anabolic processes. Its primary function is to regulate protein synthesis. This is achieved by phosphorylating key components of the translational machinery including ribosomal protein S6, which is a constituent of the 40S ribosomal subunit; eukaryotic initiation factor 4B (eIF4B), which facilitates the recruitment of mRNA to the ribosome; and eukaryotic elongation factor 2 kinase (EEF2K), whose phosphorylation leads to activation of eEF2 thereby promoting translation elongation (cronin2023amechanisticapproach pages 27-34, pende2014ribosomalproteins6 pages 1-3). In addition, RPS6KB1 phosphorylates PDCD4, triggering its ubiquitination and degradation, thereby relieving its inhibitory effect on eIF4A during translation initiation. Phosphorylation of POLDIP3/SKAR by RPS6KB1 further promotes the pioneer round of translation, ensuring efficient mRNA translation from newly synthesized transcripts. Beyond its direct influence on protein synthesis, RPS6KB1 also modulates cell survival by phosphorylating and inactivating the pro-apoptotic protein BAD, thus contributing to cell survival pathways. Furthermore, the kinase is involved in metabolic regulation; for instance, it phosphorylates CAD, an enzyme in the pyrimidine biosynthetic pathway, as well as EPRS which is implicated in fatty acid uptake. Through phosphorylation of IRS1, RPS6KB1 induces its degradation and thereby plays a role in the development of insulin resistance. In addition to managing translational efficiency, RPS6KB1 participates in critical feedback loops that modulate mTOR signaling by phosphorylating components of mTORC2, ultimately impacting AKT1 signaling and overall cell growth regulation (cronin2023amechanisticapproach pages 27-34, murphy2021theroleof pages 11-15, murphy2021theroleof pages 111-114).
8. Other Comments  
   Owing to its pivotal role in integrating mTOR signaling with cellular translation, growth, and metabolic processes, RPS6KB1 is recognized as an attractive therapeutic target. Small-molecule inhibitors such as PF-4708671 have been developed that selectively inhibit S6K1 activity by reducing the phosphorylation of ribosomal protein S6 and IRS1, without interfering with upstream mTORC1 or PDK1 phosphorylation events (karlsson2014clinicalpotentialof pages 42-45, sridhar2022targetingrps6k1for pages 14-16, scott2020developmentofpkn2 pages 24-29). Dysregulation of RPS6KB1 has been associated with various pathologies including cancer, insulin resistance, obesity, and other metabolic disorders. Mutations or abnormal regulatory phosphorylation on RPS6KB1 can disrupt cellular homeostasis and contribute to the pathogenesis of these diseases. RPS6KB1 activity serves as a key node in feedback regulatory loops that maintain cellular anabolic balance, and its inhibition has been shown to recapitulate some aspects of caloric restriction phenotypes. The emphasis on the development of selective inhibitors continues as ongoing studies assess both the efficacy and the specificity of such agents in modulating RPS6KB1 activity in clinical settings (karlsson2014clinicalpotentialof pages 42-45, sridhar2022targetingrps6k1for pages 14-16).
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