1. Phylogeny  
   RPS6KB2, encoding ribosomal protein S6 kinase beta‑2 (S6K2), is a member of the AGC kinase superfamily that emerged early in eukaryotic evolution. Orthologs of S6K2 are found throughout mammalian species, and its closest paralog is RPS6KB1 (S6K1). Both kinases share a common evolutionary origin with other TOR pathway effectors such as PDK1, PKB (AKT), p90 ribosomal S6 kinase (RSK) and SGK. The evolution of S6K enzymes is traced back to a gene duplication event in the common ancestor of animals and fungi, placing them within the evolutionary core of TOR signalling genes present in the Last Eukaryotic Common Ancestor (LECA) (Manning2002a, Manning2002b).
2. Reaction Catalyzed  
   RPS6KB2 catalyzes the phosphorylation reaction in which ATP and a protein substrate containing serine or threonine residues are converted to ADP and the phosphoprotein. The generalized reaction can be described as follows: ATP + [protein] – (L‑serine or L‑threonine) → ADP + [protein] – (L‑serine/threonine)‑phosphate + H⁺ (chrestensen2002characterizationofthe pages 3-5).
3. Cofactor Requirements  
   The catalytic activity of S6K2 depends on the presence of divalent cations, with Mg²⁺ acting as the essential cofactor for ATP binding and phosphoryl transfer (chrestensen2002characterizationofthe pages 3-5).
4. Substrate Specificity  
   RPS6KB2 exhibits substrate specificity that is characteristic of serine/threonine kinases within the AGC family. It preferentially recognizes substrates bearing an RxRxxS/T motif. In particular, S6K2 phosphorylates the ribosomal protein S6 on specific serine residues in its C-terminal cluster; these phosphorylation events are crucial for promoting mRNA translation. Substrate specificity studies based on large‐scale analyses of the serine/threonine kinome have identified a consensus motif of RxRxxp[ST] that S6K2 uses, aligning with its primary role in phosphorylating ribosomal protein S6 (Johnson2023, chrestensen2002characterizationofthe pages 3-5).
5. Structure  
   RPS6KB2 contains three major regions. The central kinase domain exhibits the conserved bi‐lobed structure typical of AGC kinases, including an activation loop (T-loop), a C-helix necessary for catalytic activity, and a hydrophobic spine that stabilizes its active conformation. Near the N-terminus, RPS6KB2 harbors a TOR signaling (TOS) motif that facilitates interaction with RAPTOR, an accessory component of the mTORC1 complex, and is essential for its mTOR-mediated phosphorylation and activation. In the C-terminal region, S6K2 is distinct from S6K1 as it possesses a proline-rich region followed immediately by a nuclear localization signal (NLS), rather than the PDZ-binding domain found in S6K1. This C-terminal arrangement is considered to be functionally important for directing subcellular localization and possibly for establishing interactions with specific substrates or regulatory proteins (huo2011investigationofthe pages 34-39, julich2008skaranovel pages 15-19, karlsson2014clinicalpotentialof pages 42-45). Structural studies and model predictions confirm that these domain features, including the TOS motif at the N-terminus and the proline-rich region with an embedded NLS in the C-terminus, underlie the unique regulation and substrate interactions of RPS6KB2 (magnuson2012regulationandfunction pages 2-3, thiriet2013cytoplasmicproteinserinethreonine pages 63-66).
6. Regulation  
   The activation of S6K2 is tightly controlled by multi-step phosphorylation events primarily downstream of mTORC1 signaling. Full activation requires the phosphorylation of the hydrophobic motif at Thr388 by mTORC1 and phosphorylation at the activation loop (T-loop) at Thr228 by PDK1. These events relieve autoinhibition and stabilize the active conformation of the kinase (chrestensen2002characterizationofthe pages 5-7, magnuson2012regulationandfunction pages 6-7). In addition to these key phosphorylation events, RPS6KB2 undergoes various post-translational modifications including phosphorylation at additional serine or threonine residues, acetylation, ubiquitination, and arginine methylation in its C-terminal regulatory domain. For example, phosphorylation by protein kinase C (PKC) at specific sites in the C-terminal region can modulate the nuclear localization signal, leading to changes in subcellular distribution (khalil2024s6k2infocus pages 9-11, julich2008skaranovel pages 15-19). These modifications collectively determine its interaction with upstream activators such as mTOR and PDK1 and downstream substrates including ribosomal protein S6 (fonseca2016evolutionoftor pages 37-40, sridharan2020distinctrolesof pages 3-5).
7. Function  
   As a serine/threonine kinase, RPS6KB2 is responsible for phosphorylating ribosomal protein S6, a key component of the 40S ribosomal subunit, thereby regulating mRNA translation and protein synthesis. Its activity is modulated in response to growth factors and nutrients, functioning downstream of mTORC1 to promote cell proliferation, cell growth, and cell cycle progression. RPS6KB2 operates through an alternative pathway regulated, in part, by MEAK7 and is implicated in the fine-tuning of translational control in various cellular contexts (fonseca2014theeverevolvingrole pages 7-7, mostafa2012hormonalandnutrient pages 59-64). Moreover, S6K2 may contribute to feedback regulation within the mTOR signaling network and has been linked to cellular processes relevant in oncogenic contexts as well as metabolic homeostasis (sridharan2020distinctrolesof pages 1-3, khalil2024s6k2infocus pages 24-25). Experimental studies have demonstrated that deletion or knockdown of S6K2 results in major reductions in ribosomal protein S6 phosphorylation, emphasizing its non-redundant role with S6K1 in the regulation of translational machinery (roux2018signalingpathwaysinvolved pages 11-13, ruvinsky2006ribosomalproteins6 pages 3-4).
8. Other Comments  
   Several inhibitors targeting S6 kinases in general have been identified in preclinical studies. Inhibitors such as PF‑4708671 and LY2584702 can reduce the phosphorylation of ribosomal protein S6 and downstream signaling events; however, these inhibitors tend to be more potent against S6K1 than S6K2 due to structural differences in their regulatory regions (karlsson2014clinicalpotentialof pages 49-52, majaeed2019s6kinasea pages 20-21). RPS6KB2 has been associated with cancer progression, and its differential expression and activity in various tumors have fueled interest in developing more selective therapeutic agents that target its unique C-terminal proline-rich region and nuclear localization features (tavares2015thes6kprotein pages 7-8, khalil2024s6k2infocus pages 7-9). RPS6KB2 is also under investigation as a potential mediator of chemoresistance and metabolic adaptation in cancer cells, indicating its relevance not only as a biomarker but also as a therapeutic target (lapenas2023ofthevulnerability pages 23-27, rebholz2006receptorassociationand pages 48-51).
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