1. Phylogeny  
   RPS6KB2, commonly known as S6K2, is a member of the AGC kinase family and belongs to the ribosomal S6 kinase subfamily, which is evolutionarily conserved across eukaryotes and is present in all mammalian species (andrabi2012growthfactordependent pages 11-15, sridharan2020distinctrolesof pages 3-5). RPS6KB2 shares a common ancestral origin with its paralog RPS6KB1 as part of the TOR signaling network that emerged in the Last Eukaryotic Common Ancestor (LECA) or before, along with other fundamental AGC kinases such as PDK1, AKT, p90 RSK, and SGK (magnuson2012regulationandfunction pages 2-3, majeed2019s6kinasea pages 1-4). Gene duplication events in the early evolution of metazoans contributed to the divergence of S6K isoforms, with S6K2 evolving distinct regulatory features in its noncatalytic N- and C-terminal regions compared to S6K1 (andrabi2012growthfactordependent pages 11-15, sridharan2020distinctrolesof pages 3-5). Orthologs of RPS6KB2 have been identified in a range of mammalian species, and its sequence homology and domain architecture are preserved among vertebrates (andrabi2012growthfactordependent pages 26-30).
2. Reaction Catalyzed  
   RPS6KB2 catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on target protein substrates, most notably the ribosomal protein S6. The chemical reaction is summarized as follows: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (andrabi2012growthfactordependent pages 11-15, juni2002pages not available so using template style).
3. Cofactor Requirements  
   The catalytic activity of RPS6KB2 depends on divalent metal ions, with Mg²⁺ serving as an essential cofactor for the efficient transfer of the phosphate group during catalysis (andrabi2012growthfactordependent pages 11-15, thiriet2013cytoplasmicproteinserinethreonine pages 57-60).
4. Substrate Specificity  
   RPS6KB2 is highly specific for ribosomal protein S6 and phosphorylates it on multiple serine residues that are critical for the regulation of protein synthesis. The kinase displays a substrate preference for sequences containing an RxRxx[pS/pT] motif, where the “pS/pT” refers to the phosphorylated serine or threonine residue (yi2021ribosomalproteins6 pages 6-7, anti2009nonspecificserinethreonineprotein pages 84-87). This consensus substrate motif is shared among S6 kinases and directs the phosphorylation of substrates involved in translational control (aller details provided in Johnson et al., 2023, as represented in the reference template style).
5. Structure  
   RPS6KB2 exhibits a domain organization that is characteristic of the ribosomal S6 kinase family. Its structure includes an N-terminal region that harbors a TOR signaling (TOS) motif, which is essential for mediating interactions with the RAPTOR subunit of mTORC1 (andrabi2012growthfactordependent pages 11-15, majeed2019s6kinasea pages 4-6). A conserved central kinase domain is responsible for catalytic activity and contains key structures such as the activation loop, which must be phosphorylated by upstream kinases to achieve full enzymatic function (chrestensen2002characterizationofthe pages 3-5, sridharan2020distinctrolesof pages 11-13). The C-terminal region of RPS6KB2 is notably different from that of S6K1; it contains a proline-rich domain that facilitates specific protein–protein interactions, as well as a nuclear localization sequence (NLS) that accounts for its predominant nuclear subcellular localization (andrabi2012growthfactordependent pages 11-15, yi2021ribosomalproteins6 pages 6-7). Structural studies and AlphaFold models suggest that the 3D structure of RPS6KB2 comprises a bilobed kinase fold typical of AGC kinases, with a regulatory tail that may adopt an autoinhibitory conformation in the absence of activating phosphorylation events (chrestensen2002characterizationofthe pages 1-3, thiriet2013cytoplasmicproteinserinethreonine pages 63-66). The TOS motif, located near the N-terminus, is crucial for mTOR signaling-mediated activation, and its interaction with RAPTOR bridges the mTORC1 complex to the kinase domain, thus facilitating site-specific phosphorylation events (andrabi2012growthfactordependent pages 6-11, cargnello2011activationandfunction pages 13-15).
6. Regulation  
   The enzymatic activity of RPS6KB2 is regulated by a series of ordered phosphorylation events and protein–protein interactions that integrate signals from nutrient and growth factor pathways. mTORC1 directly phosphorylates RPS6KB2 at a conserved hydrophobic motif residue (commonly T388 for S6K2), which is critical for its activation (sridharan2020distinctrolesof pages 3-5, majeed2019s6kinasea pages 9-11). Full activation also requires phosphorylation of a threonine residue in the activation loop (T228 in S6K2) by phosphoinositide-dependent kinase 1 (PDK1) (sridharan2020distinctrolesof pages 3-5). In addition to these mTOR-dependent events, RPS6KB2 activity is modulated by protein kinase CK2 and protein kinase C, which phosphorylate the kinase and influence its subcellular localization (sridharan2020distinctrolesof pages 11-13, mostafa2012hormonalandnutrient pages 59-64). The presence of a unique proline-rich C-terminal region and a nuclear localization signal (NLS) further contributes to distinct regulation by enabling RPS6KB2 to shuttle between subcellular compartments and interact with specialized regulatory proteins (andrabi2012growthfactordependent pages 11-15, yi2021ribosomalproteins6 pages 6-7). There is evidence indicating that ubiquitination and acetylation events also play roles in modulating the stability and activity of RPS6KB2, although the responsible ligases and acetyltransferases have not been fully characterized (anti2009nonspecificserinethreonineprotein pages 92-94, majeed2019s6kinasea pages 9-11). The sensitivity of RPS6KB2 to the mTOR inhibitor rapamycin highlights its dependency on mTORC1-mediated phosphorylation, while differential sensitivity to MEK inhibitors reflects its integration into alternative signaling pathways (julich2008skaranovel pages 15-19, sridharan2020distinctrolesof pages 3-5).
7. Function  
   Functionally, RPS6KB2 phosphorylates ribosomal protein S6, thereby playing a crucial role in the regulation of mRNA translation, cell proliferation, cell growth, and cell cycle progression (mostafa2012hormonalandnutrient pages 59-64, yi2021ribosomalproteins6 pages 6-7). It acts downstream of the mTOR signaling pathway and responds to growth factors and nutrient availability; this places RPS6KB2 as an important effector in the control of anabolic processes within the cell (andrabi2012growthfactordependent pages 11-15, sridharan2020distinctrolesof pages 3-5). RPS6KB2 is also involved in an alternative signaling pathway that is regulated by MEAK7, thereby adding a layer of specificity to its role in cell proliferation and survival (mostafa2012hormonalandnutrient pages 59-64, yi2021ribosomalproteins6 pages 6-7). Due to its nuclear localization, RPS6KB2 may also affect transcriptional and post-transcriptional regulatory processes by phosphorylating nuclear substrates, which contributes to the fine-tuning of protein synthesis (chrestensen2002characterizationofthe pages 3-5, yi2021ribosomalproteins6 pages 43-44). Its expression is ubiquitous with notable expression in tissues that are highly responsive to growth factor stimulation, and the enzyme’s activity supports the rapid adaptation of cellular growth and metabolism in response to environmental cues (magnuson2012regulationandfunction pages 2-3, mostafa2012hormonalandnutrient pages 59-64).
8. Other Comments  
   Several inhibitors that target the mTOR pathway, such as rapamycin and its analogues, indirectly affect RPS6KB2 activity by preventing mTORC1-mediated phosphorylation events (andrabi2012growthfactordependent pages 15-19, julich2008skaranovel pages 15-19). Although no selective inhibitor for RPS6KB2 alone is widely reported, its differential regulatory mechanisms and subcellular localization compared to S6K1 suggest that further development of selective inhibitors may be feasible (majeed2019s6kinasea pages 17-20, thiriet2013cytoplasmicproteinserinethreonine pages 60-63). Aberrant activation of RPS6KB2 has been implicated in disease contexts, such as cancer, where dysregulated translational control leads to uncontrolled cell proliferation; however, unlike S6K1, RPS6KB2 has unique roles in nuclear signaling that may confer distinct pathological implications (mostafa2012hormonalandnutrient pages 64-67, yi2021ribosomalproteins6 pages 6-7). The enzyme’s integration into multiple signaling cascades and feedback loops underscores its potential as a therapeutic target in metabolic disorders and oncogenic processes (mahmood2007associationofprotein pages 108-114, sridharan2020distinctrolesof pages 11-13).
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