1. Phylogeny  
   RSK2, encoded by the RPS6KA3 gene and alternatively known as pp90RSK2, MAPKAPK1B, or ISPK1, belongs to the p90 ribosomal S6 kinase (RSK) family, a subgroup of the MAPK-activated protein kinases within the AGC kinase family. This family comprises four human isoforms—RSK1, RSK2, RSK3, and RSK4—that share approximately 73–80% amino acid identity yet differ in their N- and C-terminal regulatory regions (romeo2012regulationandfunction pages 2-4, youn2021rskisoformsin pages 1-3). Phylogenetic analyses of the kinome have demonstrated that RSK2 arose early in eukaryotic evolution, and orthologs of RSK2 are present across metazoans; this conservation underscores its fundamental role in cellular signaling. RSK2 is evolutionarily related not only to its family members but also to other AGC kinases such as PDK1, AKT, and SGK, which together form a conserved regulatory network that dates back to the Last Eukaryotic Common Ancestor (LECA) (romeo2012regulationandfunction pages 2-4). Gene duplication events in early metazoan ancestors are believed to have given rise to the distinct RSK isoforms, allowing for divergence in regulation and substrate specificity while preserving the core catalytic mechanism (romeo2012regulationandfunction pages 2-4). As a member of the MAPK-activated protein kinases, RSK2 is positioned downstream of ERK1/2, which is pivotal for transmitting mitogenic and stress signals from the extracellular environment to intracellular targets. The evolutionary conservation of RSK2 across species and its close structural relationship with other kinases within the AGC group indicate that it plays an essential role in translating extracellular cues into specific cellular responses (youn2021rskisoformsin pages 1-3).
2. Reaction Catalyzed  
   RSK2 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine and, in some cases, threonine residues on its substrate proteins. The general chemical reaction can be represented as follows:  
     ATP + [protein]–(L-serine or L-threonine) → ADP + [protein]–(L-serine/threonine)-phosphate + H⁺  
   This reaction is characteristic of serine/threonine protein kinases and is essential for the modulation of the biological activity of target substrates through phosphorylation (smith2005identificationofthe pages 1-1, romeo2012regulationandfunction pages 9-10). The enzymatic activity of RSK2 thereby plays a critical role in initiating downstream signaling cascades that regulate various aspects of cell growth, proliferation, and survival without altering the overall amino acid sequence of the substrate proteins (smith2005identificationofthe pages 1-1).
3. Cofactor Requirements  
   The catalytic activity of RSK2 is dependent on the presence of Mg²⁺ ions, which serve as essential cofactors for the kinase reaction. Magnesium ions facilitate the proper binding of ATP in the active site and are thereby crucial for the efficient transfer of the phosphate group to substrate proteins (smith2005identificationofthe pages 2-3, anti2009nonspecificserinethreonineprotein pages 19-22). In addition to Mg²⁺, the reaction requires ATP as the phosphate donor, and the coordination of Mg²⁺ with ATP ensures the correct orientation and stabilization of the nucleotide for efficient catalysis (smith2005identificationofthe pages 2-3).
4. Substrate Specificity  
   RSK2 exhibits a defined substrate specificity that is governed by its preference for phosphorylating serine/threonine residues within consensus motifs enriched in basic amino acids. In particular, substrates commonly contain positively charged residues, such as arginine or lysine, positioned appropriately relative to the phosphorylation site. The consensus motif often involves basic residues at critical positions—for example, the −3 position relative to the target serine or threonine—which facilitate electrostatic interactions with the catalytic residues of RSK2 (romeo2012regulationandfunction pages 9-10, tchevkina2012proteinphosphorylationas pages 26-29). Documented substrates of RSK2 include key transcription factors and signaling proteins; among them are CREB1, which is phosphorylated to promote transcriptional activation of immediate-early genes, and ETV1/ER81 and NR4A1/NUR77, whose phosphorylation events are associated with the regulation of cell growth and differentiation (romeo2012regulationandfunction pages 1-2, youn2021rskisoformsin pages 9-10). Additional substrates include ribosomal protein S6 and eukaryotic initiation factor 4B (EIF4B), both of which are phosphorylated to enhance mRNA translation and thereby facilitate protein synthesis (information provided, romeo2012regulationandfunction pages 11-12). RSK2 also phosphorylates pro-apoptotic regulators such as BAD and DAPK1, leading to repression of their apoptotic functions; such modifications contribute to the survival and proliferation of cells in response to mitogenic or stress-induced signals (information provided, romeo2012regulationandfunction pages 11-12). The substrate specificity of RSK2 is intricately linked to its structural conformation and regulated by dynamic interactions with substrate proteins, ensuring that the phosphorylation events occur in a spatially and temporally controlled manner (tchevkina2012proteinphosphorylationas pages 26-29).
5. Structure  
   RSK2 is characterized by a dual kinase domain architecture that underpins its unique catalytic and regulatory functions. The protein contains an N-terminal kinase domain (NTKD) and a C-terminal kinase domain (CTKD), which are separated by a linker region of approximately 100 amino acids. The NTKD belongs to the AGC kinase family and is primarily responsible for phosphorylating downstream substrates, while the CTKD is structurally related to Ca²⁺/calmodulin-dependent kinases and plays an essential role in autophosphorylation events that regulate overall kinase activity (romeo2012regulationandfunction pages 2-4, kurinov2009structuraldiversityof pages 1-2).

Detailed structural studies, including crystallographic analyses, have revealed that the N-terminal kinase domain of RSK2 adopts an active conformation complicated by a unique structural adaptation. Notably, the NTD contains a three-stranded βB-sheet insertion within its N-lobe that displaces the canonical αC-helix. This displacement results in an altered positioning of the αC-helix such that the typical salt bridge formation between a conserved lysine (usually involved in ATP coordination) and a glutamate residue is disrupted. Instead, an alternative interaction is established involving Lys216, located on the novel βB-sheet, which directly contacts the β-phosphate of ATP or its analogs (kurinov2009structuraldiversityof pages 3-4, kurinov2009structuraldiversityof pages 4-6). Mutation studies that replace Lys216 with an alanine residue (K216A) have demonstrated a severe reduction in kinase activity both in vitro and ex vivo, confirming the critical role of this residue in the catalytic mechanism (kurinov2009structuraldiversityof pages 3-4).

The crystallographic structure of the NTKD, solved in complex with the non-hydrolyzable ATP analog AMP-PNP at a resolution of approximately 1.8 Å, reveals a well-defined ATP-binding pocket that shares significant structural similarity with other kinases such as protein kinase A (PKA), despite possessing unique deviations due to its β-sheet insertion (kurinov2009structuraldiversityof pages 8-10). Key catalytic motifs, including the DFG (Asp-Phe-Gly) motif and the RD (Arg-Asp) motif, are conserved and play vital roles in substrate coordination and phosphate transfer. The overall structural organization ensures that RSK2 is capable of accommodating ATP and phosphorylating substrates in a manner that is both efficient and highly regulated (kurinov2009structuraldiversityof pages 8-10).

In contrast to the NTKD, the C-terminal kinase domain exhibits a structure more similar to that of Ca²⁺/calmodulin-dependent kinases. The CTKD is responsible for autophosphorylation events that lead to the creation of a hydrophobic docking motif within the linker region. This phosphorylation event is a prerequisite for the recruitment of 3-phosphoinositide-dependent kinase-1 (PDK1), which further phosphorylates the activation loop within the NTKD, culminating in full activation of the enzyme (romeo2012regulationandfunction pages 5-7). The linker region itself is not merely a passive connector; it contains several regulatory phosphorylation sites that are critical for the controlled activation and proper subcellular localization of RSK2. Thus, the bipartite structural organization of RSK2—comprising the NTKD with its unique catalytic adaptations and the CTKD serving as a regulatory module—provides a framework for its sophisticated mechanism of action (romeo2012regulationandfunction pages 5-7, kurinov2009structuraldiversityof pages 1-2).

The 3D structural models of RSK2, derived from both experimental crystallography and predictive algorithms such as AlphaFold, consistently show that the kinase domains are flanked by flexible regions that likely mediate interactions with substrates and regulatory proteins. This structural flexibility may contribute to the enzyme’s ability to respond to a diverse array of extracellular stimuli and to integrate signals from multiple upstream pathways (kurinov2009structuraldiversityof pages 8-10, romeo2012regulationandfunction pages 11-12). Overall, its unique structural features—including the noncanonical arrangement of the βB-sheet insertion and the dual kinase domain configuration—are essential for the enzyme’s role in relaying mitogenic and stress signals to a variety of downstream effectors (kurinov2009structuraldiversityof pages 3-4, romeo2012regulationandfunction pages 11-12).

1. Regulation  
   RSK2 is subject to a complex multi-step regulatory mechanism that ensures its precise activation in response to extracellular signals. Its regulation is predominantly orchestrated by the Ras/MAPK pathway, in which extracellular stimuli such as growth factors (e.g., EGF) activate ERK1/2, the immediate upstream kinases of RSK2. ERK1/2 binds to RSK2 via a specialized docking domain and phosphorylates specific residues within the C-terminal kinase domain (CTKD), including Thr573 in the activation loop. This initial phosphorylation event is critical for the conformational rearrangement of the CTKD and its subsequent autophosphorylation events (romeo2012regulationandfunction pages 7-8, youn2021rskisoformsin pages 3-5).

Following the activation of the CTKD, RSK2 undergoes autophosphorylation at residues such as Ser380 in the linker region, a modification that creates a high-affinity docking site for PDK1. PDK1 is a constitutively active kinase that then phosphorylates Ser221 within the activation loop of the N-terminal kinase domain (NTKD), thereby fully activating the catalytic function of RSK2 (romeo2012regulationandfunction pages 7-8, youn2021rskisoformsin pages 3-5). In addition to these central phosphorylation events, further modifications occur that fine-tune the activity, substrate specificity, and subcellular localization of RSK2. Phosphorylation at Ser227, although its precise role is less clear, is recognized as a key activation marker that contributes to the optimal catalytic performance of the enzyme (kurinov2009structuraldiversityof pages 1-2, romeo2012regulationandfunction pages 10-11).

RSK2 regulation is also modulated by feedback mechanisms. For instance, phosphorylation events at sites such as Ser732 have been implicated in reducing the affinity of RSK2 for ERK, thereby serving as a negative feedback loop to limit prolonged signaling (romeo2012regulationandfunction pages 16-17). In some cellular contexts, RSK2 can interact with regulatory proteins such as 14-3-3, which bind to phosphorylated serine residues and sequester RSK2 in specific subcellular compartments, further modulating its activity and access to substrates (romeo2012regulationandfunction pages 14-14).

Alternate regulatory pathways may also contribute to RSK2 activation. In addition to the canonical ERK-mediated cascade, RSK2 may be subject to modulation by other kinases—for example, p38 MAPK or FGFR3-mediated tyrosine phosphorylations have been reported to influence its activation state in a context-dependent manner (youn2021rskisoformsin pages 3-5). These additional phosphorylation events can alter both the catalytic efficiency and the substrate interaction profile of RSK2, thereby expanding its functional repertoire in response to diverse extracellular cues (youn2021rskisoformsin pages 3-5, romeo2012regulationandfunction pages 10-11).

The dynamic interplay of these phosphorylation events, autophosphorylation processes, and protein–protein interactions ensures that RSK2 activity is tightly controlled. Such regulation is essential to prevent aberrant signaling that could lead to inappropriate cell proliferation or survival, as is observed in various pathological contexts (wright2023therapeutictargetingof pages 3-4, romeo2012regulationandfunction pages 16-17). Overall, the precise regulation of RSK2 involves a combination of sequential phosphorylation by ERK1/2 and PDK1, autophosphorylation events within distinct domains, and interactions with regulatory partners that together modulate catalytic activity, substrate specificity, and subcellular distribution (romeo2012regulationandfunction pages 7-8, youn2021rskisoformsin pages 3-5).

1. Function  
   RSK2 functions as a critical mediator of cellular responses to extracellular signals and plays a central role in a variety of biological processes. As a serine/threonine protein kinase acting downstream of the ERK1/2 MAPK cascade, RSK2 orchestrates a range of cellular programs that include cell growth, proliferation, survival, and differentiation. One of the principal mechanisms through which RSK2 exerts its biological effects is by phosphorylating key transcription factors and regulatory proteins. For example, the phosphorylation of CREB1 by RSK2 leads to the activation of immediate-early gene transcription, a process that is fundamental for mitogenic responses in fibroblasts upon EGF stimulation (kurinov2009structuraldiversityof pages 1-2, romeo2012regulationandfunction pages 1-2). In addition to CREB1, RSK2 phosphorylates transcription factors such as ETV1/ER81 and NR4A1/NUR77, thereby playing a role in the regulation of gene expression patterns that govern cellular differentiation and stress responses (romeo2012regulationandfunction pages 1-2, youn2021rskisoformsin pages 9-10).

RSK2 also modulates protein synthesis by phosphorylating proteins involved in the translation machinery. Among its substrates are ribosomal protein S6 and eukaryotic initiation factor 4B (EIF4B), whose phosphorylation facilitates cap-dependent translation and thus supports the synthesis of proteins required for cell growth and proliferation (romeo2012regulationandfunction pages 11-12, youn2021rskisoformsin pages 9-10). In the context of survival signaling, RSK2 phosphorylates pro-apoptotic molecules such as BAD and DAPK1, resulting in the repression of apoptosis and promoting cell survival under stress conditions (romeo2012regulationandfunction pages 11-12, youn2021rskisoformsin pages 12-13).

Beyond its roles in transcription and translation, RSK2 is involved in the modulation of mTOR signaling—a key pathway that integrates nutrient availability and growth signals to control cellular metabolism. By influencing mTOR activity, RSK2 indirectly affects cellular anabolic processes and the regulation of autophagy. This additional level of control highlights the kinase’s position as a central node in the integration of multiple signaling cascades (romeo2012regulationandfunction pages 12-13, wright2023therapeutictargetingof pages 11-12).

RSK2’s functional roles are not limited to proliferative processes; it is also implicated in differentiation and stress responses across various cell types. For instance, in fibroblasts and neuronal cells, RSK2 activity is linked to cytoskeletal reorganization and the control of cell motility, which are essential for processes such as wound healing and neurite outgrowth (lin2019roleofp90rsk pages 1-3, cuello2011novelrolefor pages 1-2). Moreover, the kinase’s involvement in the phosphorylation of histone H3 at serine 10 has been associated with chromatin remodeling and transcriptional activation, further underscoring its role in gene regulation (kurinov2009structuraldiversityof pages 1-2).

In the context of disease, RSK2 is of significant clinical interest due to its dual role in developmental processes and oncogenic signaling. Mutations in RPS6KA3, which encodes RSK2, are directly linked to Coffin–Lowry syndrome—a rare X-linked developmental disorder characterized by intellectual disability and skeletal abnormalities (romeo2012regulationandfunction pages 17-17, youn2021rskisoformsin pages 12-13). Furthermore, aberrant activation and overexpression of RSK2 have been observed in various cancers, including breast cancer, melanoma, and hematologic malignancies; such dysregulation contributes to uncontrolled cell proliferation and survival, marking RSK2 as a promising therapeutic target (smith2005identificationofthe pages 5-6, wright2023therapeutictargetingof pages 14-15).  
The tissue-specific expression patterns of RSK2 indicate elevated mRNA and protein levels in proliferative tissues such as skeletal muscle, heart, and certain regions of the brain where its activity influences differentiation and survival pathways (romeo2012regulationandfunction pages 4-5, lin2019roleofp90rsk pages 1-3). In addition, RSK2 is involved in the regulation of metabolic processes and stress response pathways, thereby impacting cellular homeostasis and contributing to the overall control of cell cycle progression (romeo2012regulationandfunction pages 12-13, youn2021rskisoformsin pages 3-5).

1. Other Comments  
   RSK2 has been the subject of extensive studies aimed at developing small-molecule inhibitors that can modulate its activity for potential therapeutic applications. Among the most notable inhibitors is SL0101, a naturally derived flavonol glycoside that has been shown to selectively inhibit RSK isoforms, particularly RSK2, resulting in cell cycle arrest in malignant cells without significantly affecting normal cell proliferation (smith2005identificationofthe pages 2-3, wright2023therapeutictargetingof pages 15-15). Additional ATP-competitive inhibitors, such as BI-D1870, have been routinely employed in both in vitro and in vivo studies to dissect the functional role of RSK2 in various signaling pathways. These inhibitors target the ATP-binding pocket within the NTKD but frequently encounter challenges in achieving absolute isoform specificity due to the high sequence conservation among RSK family members (wright2023therapeutictargetingof pages 14-14).

Moreover, irreversible inhibitors like FMK have been developed to target the CTKD of RSK2 by covalently modifying a reactive cysteine residue within the ATP-binding site, thereby exerting potent inhibition. Such agents provide valuable chemical tools for elucidating the contributions of RSK2 to cellular signaling and oncogenesis (wright2023therapeutictargetingof pages 9-11). Ongoing drug discovery efforts, including structure-based virtual screening and the development of proteolysis-targeting chimeras (PROTACs), continue to refine the selectivity and efficacy of RSK2 inhibitors, with some compounds progressing into early-phase clinical trials for the treatment of advanced cancers (wright2023therapeutictargetingof pages 15-15, youn2021rskisoformsin pages 14-15).

RSK2 is further associated with several disease states beyond cancer. Mutations in the RPS6KA3 gene cause Coffin–Lowry syndrome, indicating that loss-of-function or hypomorphic alleles of RSK2 disrupt normal neurodevelopmental and skeletal processes (romeo2012regulationandfunction pages 17-17, youn2021rskisoformsin pages 12-13). In oncological contexts, hyperactivation of RSK2 has been implicated in promoting tumorigenesis by enhancing cell proliferation, survival, and metastasis, making it a target of substantial interest in cancer biology (smith2005identificationofthe pages 8-8, wright2023therapeutictargetingof pages 14-15). In addition, RSK2-mediated phosphorylation events play a role in the regulation of mTOR signaling and translational control, suggesting that aberrations in its activity may contribute to metabolic dysregulation in various diseases.  
Collectively, the extensive research on RSK2 supports its candidacy as an attractive therapeutic target, and the continued development and refinement of selective inhibitors promise to advance clinical applications in oncology and potentially in the treatment of genetic disorders linked to impaired RSK2 activity (wright2023therapeutictargetingof pages 13-13, youn2021rskisoformsin pages 14-15).

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