1. Phylogeny – Ribosomal protein S6 kinase alpha‑2 (RSK3), encoded by the gene RPS6KA2 and also known as pp90RSK3, belongs to the p90 ribosomal S6 kinase (RSK) family, a subgroup within the larger AGC kinase superfamily. RSK family members, which include RSK1, RSK2, RSK3, and RSK4, are evolutionarily conserved serine/threonine kinases that trace their origins to an early gene duplication event in the common ancestor of animals and fungi. Orthologs of RSK3 are found across a broad range of species – from vertebrates to invertebrates such as Drosophila and Caenorhabditis elegans – thus indicating that the fundamental mechanisms of RSK-mediated signal transduction have been preserved throughout eukaryotic evolution (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 2-4). The conservation extends not only to the catalytic core but also to critical regulatory domains, which include the N‑terminal kinase domain (NTKD) responsible for substrate phosphorylation and the C‑terminal kinase domain (CTKD) involved in autophosphorylation and regulatory functions. In comparison to its paralogs, RSK3 exhibits unique sequence variations in its non‐catalytic regions that may underlie subtle differences in subcellular localization and signal modulation. Collectively, phylogenetic analysis supports a model wherein the RSK family has maintained an essential role in bridging extracellular cues with intracellular responses, thus positioning RSK3 as a critical node in the MAPK pathway (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 2-4).

RSK3 is situated within an ancient kinase network that includes other AGC kinases such as PDK1 and Akt, all of which are central to the regulation of cell growth, proliferation, and stress responses. The evolutionary retention of these kinases underscores their importance in maintaining cellular homeostasis over extensive phylogenetic periods. Studies have shown that the RSK family, as part of this larger signaling module, participates in relaying signals from the Ras/MAPK cascade to downstream effectors that regulate gene expression, protein synthesis, and cell cycle progression (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 2-4). Moreover, the phylogenetic grouping of RSK3 with its RSK family counterparts emphasizes both the conservation and functional specialization that have evolved to meet the diverse demands of different cell types and tissues.

1. Reaction Catalyzed – RSK3 functions as a serine/threonine protein kinase that catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues in substrate proteins. The catalytic reaction can be formally represented as: ATP + [protein]‑(L‑serine or L‑threonine) → ADP + [protein]‑(phospho‑L‑serine/threonine) + H⁺. This ATP‐dependent phosphorylation event forms the biochemical basis through which RSK3 modulates the structure and activity of its substrates, thereby influencing key cellular processes such as transcription, translation, and cell cycle progression (anjum2008therskfamily pages 4-4, roux2003phosphorylationofp90 pages 8-9). The efficiency and selectivity of this reaction are critical for ensuring that the appropriate downstream signals are transduced in response to mitogenic or stress stimuli received by the cell.
2. Cofactor Requirements – The kinase activity of RSK3 strictly depends on the presence of divalent metal ions, with magnesium (Mg²⁺) serving as the essential cofactor. Mg²⁺ coordinates with ATP in the active site, thereby stabilizing the binding of the nucleotide and orienting its γ‑phosphate precisely for transfer to the target substrate. As with many serine/threonine kinases within the AGC family, this dependence on Mg²⁺ is a hallmark of RSK3’s catalytic mechanism, ensuring the fidelity and efficiency of the phosphorylation reaction (anjum2008therskfamily pages 4-4, jain2015discoveryofpotent pages 1-2).
3. Substrate Specificity – RSK3 phosphorylates serine/threonine residues on target proteins that contain specific consensus recognition motifs. The substrates of RSK3 include a variety of transcription factors—including CREB, ATF4, and NFATc4—as well as proteins involved in translation regulation, such as eukaryotic initiation factor 4B and ribosomal protein S6. These proteins typically harbor motif sequences enriched in basic residues; for instance, a frequently observed consensus is characterized by an arginine-rich motif, commonly exemplified as RxRxxp[serine/threonine], where ‘p’ denotes the phosphorylated residue. This recognition motif ensures that RSK3 selectively engages substrates that are integral to mediating diverse cellular responses, thereby linking extracellular signaling events to changes in gene expression and protein synthesis (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 8-9). The substrate specificity of RSK3 is further refined by docking interactions provided by ancillary protein domains that promote the formation of a productive enzyme–substrate complex in appropriate cellular contexts (jain2015discoveryofpotent pages 1-2).
4. Structure – RSK3 is a multidomain protein with an approximate molecular weight of 90 kDa. Its two main catalytic units are arranged in a tandem fashion: the N‑terminal kinase domain (NTKD) and the C‑terminal kinase domain (CTKD). The NTKD, which is responsible for phosphorylating downstream substrates, exhibits characteristic AGC kinase features such as a glycine-rich loop for ATP binding, a catalytic loop containing essential aspartate residues, and an activation segment that undergoes conformational changes upon phosphorylation. The CTKD, in contrast, is largely involved in regulatory autophosphorylation events required for subsequent activation of the NTKD (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 4-5).

Between these catalytic domains lies a flexible linker region that contains multiple phosphorylation sites critical for full enzymatic activation. One key regulatory element is the kinase interaction motif (KIM), which serves as the binding site for extracellular signal‑regulated kinases (ERK1/2). ERK binding facilitates initial phosphorylation events that trigger a cascade of intramolecular autophosphorylation, ultimately culminating in the activation of the NTKD by PDK1. Additionally, RSK3 possesses a nuclear localization signal (NLS) that directs its import into the nucleus, thereby positioning it to phosphorylate nuclear targets that regulate transcription (romeo2012regulationandfunction pages 2-4, romeo2012regulationandfunction pages 13-14).

Structural models based on crystallographic studies of related RSK isoforms reveal a conserved bilobal kinase fold comprising a smaller N‑terminal lobe and a larger C‑terminal lobe, with the active site located in the inter-lobal cleft. Within this architecture, the activation loop is a critical structural element whose phosphorylation drives rearrangements of the αC‑helix and the assembly of a hydrophobic regulatory spine that stabilizes the active conformation. Moreover, the dual kinase domains enable RSK3 to integrate intramolecular regulatory signals; phosphorylation within the CTKD can allosterically modulate the NTKD’s catalytic efficiency, ensuring that the enzyme’s activity is tightly controlled by upstream mitogenic cues (romeo2012regulationandfunction pages 2-4, somale2020activationofrsk pages 10-11).

Unique structural features of RSK3, as compared to other RSK isoforms, include variations in its C‑terminal tail regions, which may harbor proline‑rich motifs that contribute to specific protein–protein interactions and subcellular targeting. These structural variations likely underpin differences in localization, substrate recognition, and signaling output for RSK3 relative to RSK1, RSK2, and RSK4. Collectively, the modular domain organization and defined catalytic elements of RSK3 provide the structural basis for its function as a signal transducer activated downstream of the MAPK pathway (romeo2012regulationandfunction pages 4-5, roux2003phosphorylationofp90 pages 8-9, romeo2012regulationandfunction pages 16-17).

1. Regulation – The activity of RSK3 is controlled by a multi‑step phosphorylation cascade that integrates signals received from upstream MAPK/ERK pathways. Initially, ERK1/2 binds to a conserved docking motif on RSK3 and phosphorylates specific residues on the CTKD. This ERK‑mediated phosphorylation not only primes RSK3 for further modifications but also triggers intramolecular autophosphorylation events that are necessary for forming a docking site for 3‑phosphoinositide‑dependent protein kinase 1 (PDK1). Subsequent phosphorylation by PDK1 at the activation loop within the NTKD is critical for relieving autoinhibition and fully activating the kinase (anjum2008therskfamily pages 1-2, romeo2012regulationandfunction pages 5-7).

The regulatory process further involves additional phosphorylation events at both the linker region and the hydrophobic motif, which contribute to conformational stabilization and amplification of catalytic activity. For instance, phosphorylation-induced structural changes facilitate the repositioning of the αC‑helix and the establishment of a hydrophobic regulatory spine, both of which are imperative for the enzymatic function of the NTKD. Moreover, RSK3 is subject to dephosphorylation by specific phosphatases such as MKP1 and MKP2, which help terminate the signal and restore the kinase to its basal state (romeo2012regulationandfunction pages 16-16, roux2003phosphorylationofp90 pages 9-9).

A notable feature of RSK3 regulation is its prolonged association with ERK even after the initial activation event, a characteristic that distinguishes it from other RSK isoforms. This sustained interaction may impart extended signaling duration, thereby affecting the temporal dynamics of downstream target phosphorylation. Furthermore, the presence of nuclear localization signals within RSK3 facilitates its translocation to the nucleus upon activation, ensuring that key transcriptional regulators are targeted appropriately. In parallel, interactions with scaffold proteins further refine spatial and temporal control over RSK3 activity, linking extracellular stimuli to specific intracellular responses (romeo2012regulationandfunction pages 16-17, somale2020activationofrsk pages 1-2).

Pharmacological agents, including the inhibitors BI‑D1870 and SL0101, have been used experimentally to disrupt RSK kinase activity by interfering with ATP binding to the NTKD. Although these inhibitors affect multiple RSK isoforms, their application in cellular and biochemical assays has provided insights into the regulatory mechanisms underlying RSK3 activation and inactivation (jain2015discoveryofpotent pages 16-17, serra2013rsk34mediateresistance pages 8-10). Overall, the regulation of RSK3 is characterized by a precisely orchestrated series of phosphorylation and dephosphorylation events, combined with subcellular localization signals and protein–protein interaction motifs that collectively ensure robust control over its kinase activity.

1. Function – RSK3 functions as an effector kinase downstream of the extracellular signal‑regulated kinase (ERK) signaling cascade, thereby translating extracellular mitogenic and stress signals into specific intracellular responses. As a serine/threonine kinase, RSK3 plays a crucial role in modulating the activity of transcription factors—including CREB, ATF4, and NFAT family members—that regulate immediate‑early gene expression. Through the phosphorylation of such transcription factors, RSK3 influences the expression of genes involved in cell proliferation, differentiation, and survival (anjum2008therskfamily pages 1-2, jain2015discoveryofpotent pages 1-2).

In addition to its role in transcriptional regulation, RSK3 is also pivotal in the control of protein synthesis. It phosphorylates components of the translational machinery, such as eukaryotic initiation factors and ribosomal protein S6, thereby promoting cap‑dependent translation and ensuring appropriate cell growth responses following stimulation by growth factors. This dual role in regulating both transcription and translation places RSK3 at a central node in coordinating cellular responses to extracellular signals (romeo2012regulationandfunction pages 7-8, romeo2012regulationandfunction pages 8-9).

Cell cycle progression and survival pathways are also modulated by RSK3. Its kinase activity contributes to the regulation of cell cycle checkpoints and is involved in the suppression or promotion of apoptotic pathways, depending on the cellular context. Notably, in epithelial ovarian cancer cells, RSK3 has been implicated as a potential tumor suppressor; alterations in its expression or activity may lead to dysregulated cell proliferation and contribute to oncogenic transformation (anjum2008therskfamily pages 1-2, serra2013rsk34mediateresistance pages 13-13).

Tissue‑specific expression patterns of RSK3 have been documented, with expression observed in the central nervous system, heart, skeletal muscle, and pancreas. These patterns are consistent with its roles in processes such as neuronal differentiation, cardiovascular function, and metabolic regulation. Through its diverse substrate repertoire, RSK3 integrates signals from upstream mitogenic pathways to affect a wide range of cellular processes, thereby ensuring that cell growth, survival, and differentiation are tightly coordinated in response to external stimuli (romeo2012regulationandfunction pages 12-13, romeo2012regulationandfunction pages 16-17).

1. Other Comments – Although several inhibitors targeting the broader p90 RSK family have been developed, including BI‑D1870 and SL0101, the development of highly selective inhibitors for RSK3 remains an area of ongoing research. Current pharmacological studies often employ these inhibitors to explore the functional roles of RSK family members in cellular processes; however, the specificity of these compounds for RSK3 versus other isoforms is not absolute (jain2015discoveryofpotent pages 16-17, serra2013rsk34mediateresistance pages 8-10). In the context of disease, dysregulation of RSK3 activity has been linked to oncogenic processes, and altered levels or activity of RSK3 have been observed in various cancers, including epithelial ovarian cancer and certain subtypes of breast cancer. The ability of RSK3 to mediate stress responses and to regulate transcription factors and components of the translational machinery positions it as a potential tumor suppressor as well as a mediator of resistance to targeted therapies (anjum2008therskfamily pages 1-2, serra2013rsk34mediateresistance pages 13-13). Furthermore, the unique regulatory characteristics of RSK3, such as its sustained association with ERK and its distinct subcellular localization patterns, offer promising avenues for the development of novel therapeutic strategies aimed at modulating its activity in disease states (romeo2012regulationandfunction pages 16-17, somale2020activationofrsk pages 12-13). Continued research into isoform‑specific properties and the interplay between RSK3 and its substrates will be essential for the future design of targeted interventions that selectively modulate this kinase without affecting the wider RSK family.
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