1. Phylogeny  
   Serine/threonine-protein kinase SGK1 belongs to the AGC kinase family and is evolutionarily conserved across eukaryotes. Orthologs have been identified from yeast to mammals, and its presence in a wide range of species indicates that it is part of the core set of kinases that emerged early during eukaryotic evolution (jeyarajUnknownyeardierolleder pages 27-30, daniels2010investigationofthe pages 31-35). Within the human kinome, SGK1, along with SGK2 and SGK3, forms a subfamily that displays significant sequence identity in the catalytic domain, with about 80% amino acid identity between SGK1 and its paralogues. Phylogenetic analyses place SGK1 alongside other AGC kinases such as Akt/PKB, PKA, and p70S6K, underscoring its conservation and the shared mechanisms of regulation through upstream kinases like PDK1 and mTOR complexes (daniels2010investigationofthe pages 31-35, firestone2003stimulusdependentregulationof pages 5-6).
2. Reaction Catalyzed  
   SGK1 catalyzes the phosphorylation of serine or threonine residues on substrate proteins using ATP as a phosphate donor. The overall reaction can be summarized as:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction is typical of serine/threonine kinases in the AGC family (jeyarajUnknownyeardierolleder pages 27-30).
3. Cofactor Requirements  
   The enzymatic activity of SGK1 depends on the presence of divalent cations, with Mg²⁺ being the primary cofactor required to coordinate ATP within the kinase active site. This cofactor requirement is consistent with its classification among serine/threonine protein kinases (daniels2010investigationofthe pages 35-39).
4. Substrate Specificity  
   SGK1 exhibits a substrate specificity that recognizes a consensus sequence motif that is typically described as R-X-R-X-X-(S/T)-φ, where “φ” denotes a hydrophobic amino acid. This motif is shared with other kinases in the AGC family and underlies SGK1’s ability to phosphorylate substrates involved in regulating ion channels, transporters, and transcription factors. Such specificity contributes to its role in modulating protein stability and activity, notably through the phosphorylation of regulators like the ubiquitin ligase NEDD4L, thereby affecting membrane retention of channels such as ENaC (daniels2010investigationofthe pages 35-39, jang2022serumandglucocorticoidregulated pages 13-14).
5. Structure  
   The 3D structure of SGK1 consists of a relatively conserved catalytic domain of approximately 330 amino acids flanked by regulatory sequences. The N-terminal region is variable and is involved in subcellular localization, while the catalytic domain encompasses key structural features such as the ATP-binding site (including a conserved lysine residue analogous to lysine 127 in other kinases), the activation loop, and a hydrophobic motif that is phosphorylated by mTORC2 (serine 422 in SGK1) to induce an open, active conformation (daniels2010investigationofthe pages 35-39, jang2022serumandglucocorticoidregulated pages 1-3). Additional regulatory features include a nuclear localization signal (NLS) present in the N-terminal portion that mediates stimulus-dependent nuclear–cytoplasmic shuttling as well as a six–amino-acid ubiquitin-binding motif that facilitates proteasomal degradation (firestone2003stimulusdependentregulationof pages 5-6, maestro2020serumandglucocorticoidinduced pages 3-4). The overall three-dimensional organization is consistent with a bilobal kinase fold, with an N-terminal β-sheet domain and a C-terminal α-helical domain, features typical for AGC kinases (daniels2010investigationofthe pages 31-35, jang2022serumandglucocorticoidregulated pages 17-17).
6. Regulation  
   SGK1 is regulated both transcriptionally and post-translationally. Transcriptional regulation occurs under the influence of serum, glucocorticoids, mineralocorticoids, cytokines, and various stressors such as hyperosmotic shock, high extracellular glucose, and oxidative stress. Its promoter contains response elements for glucocorticoid and mineralocorticoid receptors, enabling rapid induction in response to hormonal stimuli (firestone2003stimulusdependentregulationof pages 11-11, daniels2010investigationofthe pages 194-196).

Post-translationally, SGK1 activation is achieved through phosphorylation at multiple key residues. Phosphoinositide-dependent kinase-1 (PDK1) phosphorylates the activation loop (threonine 256), while mTOR complex 2 (mTORC2) phosphorylates the hydrophobic motif (serine 422) which is required for the proper alignment of the active site, thus enabling subsequent phosphorylation events (daniels2010investigationofthe pages 35-39, jang2022serumandglucocorticoidregulated pages 1-3). Additional phosphorylation sites have been identified (e.g., serine 78, threonine 369) that further modulate its activity through inputs from other pathways such as PKA and MAPK (firestone2003stimulusdependentregulationof pages 9-10, jang2022serumandglucocorticoidregulated pages 14-15). Moreover, SGK1 activity is intricately regulated by protein-protein interactions; binding to scaffold proteins such as NHERF2 helps localize SGK1 to the plasma membrane where it encounters its substrates. SGK1 also undergoes rapid ubiquitination via E3 ligases like Nedd4-2, which targets it for proteasomal degradation, thereby conferring a short half-life and dynamic control over its cellular levels (lou2016serumandglucocorticoid pages 14-15, maestro2020serumandglucocorticoidinduced pages 7-8).

1. Function  
   SGK1 plays a significant role in a diverse array of physiological processes. It is expressed in multiple tissues including kidney, brain, lung, and muscle, and orchestrates a number of functions such as the regulation of ion channels, membrane transporters, and transcription factors. In epithelial cells, SGK1 enhances sodium reabsorption by up-regulating the epithelial sodium channel (ENaC). This is achieved indirectly via phosphorylation of NEDD4L, whereby the binding of 14-3-3 proteins prevents the ubiquitin-dependent degradation of ENaC, resulting in increased channel stability and plasma membrane abundance (daniels2010investigationofthe pages 196-199, firestone2003stimulusdependentregulationof pages 11-11).

In addition to sodium transport, SGK1 phosphorylates and modulates the activity of an extensive list of ion channels and transporters. It up-regulates voltage-gated sodium channels (e.g., SCN5A), potassium channels such as ROMK1 (KCNJ1) and members of the KCNA and KCNQ families, epithelial calcium channels (TRPV5 and TRPV6), various chloride channels (e.g., CFTR), as well as several glutamate and amino acid transporters. SGK1 also directly phosphorylates regulators of the Na⁺/K⁺-ATPase and participates in the modulation of glucose transporters (GLUT1 and SGLT1) and other nutrient carriers, thereby influencing cellular metabolic processes (daniels2010investigationofthe pages 196-199, daniels2010investigationofthe pages 199-202, lou2016serumandglucocorticoid pages 3-5).

Beyond its classical roles in ion transport, SGK1 is involved in cell survival, proliferation, and apoptosis. It acts on transcription factors such as FOXO1 and FOXO3, causing their relocalization out of the nucleus and thereby diminishing their pro-apoptotic transcriptional programs. In neuronal cells, SGK1 phosphorylates MAPT/TAU, contributing to neurite formation and microtubule dynamics, and it regulates memory consolidation by modulating CREB1 activity (firestone2003stimulusdependentregulationof pages 11-12, lang2009thephysiologicalimpact pages 12-13). Furthermore, SGK1 phosphorylates MDM2, which in turn leads to the ubiquitination and degradation of the tumor suppressor p53, linking SGK1 activity to cell cycle regulation and oncogenic processes (daniels2010investigationofthe pages 199-202, lang2009targetingsgk1in pages 1-2).

1. Other Comments  
   Several small-molecule inhibitors targeting SGK1 have been developed in preclinical studies. Compounds such as GSK650394, EMD638683, and various pyrazolopyrazine derivatives have been identified as SGK1 inhibitors with potential applications in hypertensive, diabetic, and cancerous conditions (jang2022serumandglucocorticoidregulated pages 14-15, lang2009targetingsgk1in pages 7-8, guerriero2020serumandglucocorticoidinducible pages 25-25). SGK1 has been implicated in the pathophysiology of hypertension, diabetic nephropathy, and salt-sensitive glucose uptake, and its dysregulation is associated with fibrotic processes within the kidney and heart. Overexpression or increased activity of SGK1 due to gain-of-function polymorphisms correlates with elevated blood pressure and enhanced sodium retention. In addition, its role in mediating anti-apoptotic and pro-survival signals renders SGK1 a potential contributor to cancer progression, and it has been examined as a target in non-small cell lung cancer (guerriero2020serumandglucocorticoidinducible pages 15-17, lang2009targetingsgk1in pages 6-7). Notably, alternative isoforms of SGK1 exhibit distinct cellular localizations and functional impacts—for example, isoform 2 has been reported to have a greater effect on plasma membrane expression of ENaC than isoform 1 (daniels2010investigationofthe pages 194-196). These diverse functional roles underscore the therapeutic interest in SGK1 inhibition, with approaches aimed at achieving selective blockade without interfering with closely related kinases such as Akt/PKB (jang2022serumandglucocorticoidregulated pages 14-15).
2. References  
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