1. Phylogeny  
   Serine/threonine‐protein kinase SGK1 belongs to the AGC kinase family, a conserved group of kinases that also includes protein kinase A (PKA), protein kinase G (PKG), protein kinase C (PKC) isoforms, Akt (also called protein kinase B) and S6 kinases. SGK1 is evolutionarily conserved across eukaryotes and is present in all mammalian species. In vertebrates the SGK family comprises three paralogs – SGK1, SGK2 and SGK3 – which are encoded by distinct genes and exhibit partially overlapping yet divergent tissue expression patterns and substrate specificities. SGK1 is ubiquitously expressed, with high transcript levels detected in tissues such as kidney, heart, liver, pancreas and brain, while SGK2 shows more tissue-restricted expression and SGK3 possesses an additional Phox homology (PX) domain that targets it to endosomal membranes. Phylogenetic analyses based on the protein kinase complement of the human genome indicate that SGK1, along with other AGC kinases, arose from common ancestral eukaryotic genes and forms part of an evolutionarily conserved core set of kinases whose origins predate the divergence of yeast and mammals (jang2022serumandglucocorticoidregulated pages 1-3, lang2020theenigmaticrole pages 1-2, Manning2022).
2. Reaction Catalyzed  
   SGK1 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues on its substrate proteins. The chemical reaction can be represented as follows:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (jang2022serumandglucocorticoidregulated pages 3-4).
3. Cofactor Requirements  
   The catalytic activity of SGK1 requires the presence of divalent cations. In particular, Mg²⁺ acts as an essential cofactor that facilitates ATP binding and phosphoryl transfer during the enzymatic reaction (lou2016serumandglucocorticoid pages 3-5).
4. Substrate Specificity  
   SGK1 phosphorylates a wide spectrum of substrates involved in ion transport, metabolism, transcription regulation, cell survival, and cytoskeletal dynamics. Experimentally, SGK1 has been shown to modify proteins such as NDRG1, FOXO3a, NEDD4-2, TSC2, ULK1, and β-catenin (jang2022serumandglucocorticoidregulated pages 1-3). In addition, SGK1 regulates numerous ion channels and transporters by phosphorylating them directly or by modifying their regulatory proteins. For example, phosphorylation of the ubiquitin ligase NEDD4L by SGK1 interferes with its interaction with the epithelial sodium channel (ENaC), resulting in enhanced channel stability and function (jang2022serumandglucocorticoidregulated pages 3-4, lou2016serumandglucocorticoid pages 14-15). Published substrate specificity studies for serine/threonine kinases indicate that kinases in this family frequently preferentially phosphorylate substrates that display a basic residue-rich motif – for instance an RxRxxS/T consensus motif – although SGK1 appears capable of accommodating a broad substrate spectrum due to its versatile active site (jang2022serumandglucocorticoidregulated pages 13-14, lang2020theenigmaticrole pages 2-3).
5. Structure  
   SGK1 is a 431–amino acid protein that is organized into several distinct domains. The N-terminal region includes a PX-like motif that may contribute to subcellular localization, while the central kinase domain constitutes the catalytic core responsible for its enzymatic activity. This kinase domain contains conserved features shared among AGC kinases, including a catalytic lysine (Lys127) essential for ATP binding, a conserved Asp-Phe-Gly (DFG) motif necessary for magnesium coordination and catalysis, and a hinge region that forms hydrogen bonds with ATP (jang2022serumandglucocorticoidregulated pages 3-4). A critical leucine residue (Leu176) in the catalytic domain acts as a gatekeeper in the ATP-binding pocket. Moreover, the C-terminal hydrophobic motif (including Ser422) is crucial for full activation, as phosphorylation of this region by mTOR complex 2 (mTORC2) promotes conformational changes required for subsequent activation loop phosphorylation by PDK1 at Thr256 (jang2022serumandglucocorticoidregulated pages 1-3, maestro2020serumandglucocorticoidinduced pages 3-4). The overall three-dimensional structure is thus characterized by a bilobal kinase core with flexible regulatory regions that allow integration of upstream signals and determine substrate interactions (jang2022serumandglucocorticoidregulated pages 3-4, maestro2020serumandglucocorticoidinduced pages 4-6).
6. Regulation  
   SGK1 activity is tightly regulated both at the transcriptional and post-translational levels. Transcriptionally, SGK1 is rapidly induced by extracellular stimuli such as serum, glucocorticoids, mineralocorticoids, and cytokines (jang2022serumandglucocorticoidregulated pages 1-3, lang2020theenigmaticrole pages 1-2). Following synthesis, SGK1 undergoes critical phosphorylation events for activation. First, mTOR complex 2 phosphorylates the hydrophobic motif at Ser422, which induces a conformational change that allows PDK1 to subsequently phosphorylate the activation loop at Thr256. These phosphorylation events are essential for full kinase activation and enable SGK1 to phosphorylate its downstream substrates (jang2022serumandglucocorticoidregulated pages 1-3, maestro2020serumandglucocorticoidinduced pages 11-12). In addition to phosphorylation, SGK1 is subject to regulatory modifications such as ubiquitination that control its short half-life (approximately 30 minutes) and cellular abundance (maestro2020serumandglucocorticoidinduced pages 3-4, jang2022serumandglucocorticoidregulated pages 17-17). Conformational regulation via subcellular localization also plays a role; for instance, serum stimulation can promote nuclear translocation while glucocorticoids and hyperosmotic stress favor cytoplasmic retention (maestro2020serumandglucocorticoidinduced pages 3-4, jang2022serumandglucocorticoidregulated pages 3-4).
7. Function  
   SGK1 plays diverse roles in cellular physiology via its ability to phosphorylate an extensive array of target proteins. Functionally, SGK1 is a critical regulator of ion channels and transporters. It enhances sodium uptake and retention in epithelial cells by phosphorylating NEDD4L, thereby stabilizing the epithelial sodium channel (ENaC) and preventing its ubiquitin‐dependent degradation (jang2022serumandglucocorticoidregulated pages 3-4, lou2016serumandglucocorticoid pages 14-15). Through its actions on ion channels, SGK1 contributes to the regulation of renal sodium reabsorption, renal potassium excretion, salt appetite, and overall electrolyte homeostasis (lou2016serumandglucocorticoid pages 15-17, maestro2020serumandglucocorticoidinduced pages 6-7).  
   Beyond ion transport, SGK1 influences multiple cellular processes such as cell proliferation, survival, migration, and apoptosis. It phosphorylates transcription factors like FOXO1 and FOXO3, modulating their subcellular localization and transcriptional activity, and thereby influencing gene expression programs involved in cell survival and metabolic regulation (jang2022serumandglucocorticoidregulated pages 3-4, maestro2020serumandglucocorticoidinduced pages 12-13). SGK1 also phosphorylates regulators of cell cycle progression and mediators of cellular stress response, playing a role in autophagy modulation and cellular adaptation to environmental stress (jang2022serumandglucocorticoidregulated pages 6-8, maestro2020serumandglucocorticoidinduced pages 8-10). In the nervous system, SGK1 phosphorylates MAPT/TAU which is linked to microtubule dynamics and neurite formation in hippocampal neurons, contributing to aspects of memory consolidation and neuronal survival (jang2022serumandglucocorticoidregulated pages 1-3, lang2020theenigmaticrole pages 1-2). SGK1’s involvement in signal transduction pathways extends to modulation of enzymes such as GSK3α/β, regulation of MDM2-dependent p53 ubiquitination, and stimulation of ERK2 via strengthening its interaction with upstream kinases (jang2022serumandglucocorticoidregulated pages 14-15, maestro2020serumandglucocorticoidinduced pages 13-14). Collectively, these functions situate SGK1 as a multifunctional regulator that integrates hormonal and stress signals to control ion transport, metabolism, and cell survival (jang2022serumandglucocorticoidregulated pages 6-8, lang2020theenigmaticrole pages 2-3, lou2016serumandglucocorticoid pages 15-17).
8. Other Comments  
   Several small-molecule inhibitors targeting SGK1 have been developed and characterized in preclinical studies. For example, inhibitors such as SI113 and azaindole analogs exhibit potent inhibition in the nanomolar range and have been evaluated for their roles in modulating cancer cell proliferation and cardiac hypertrophy (jang2022serumandglucocorticoidregulated pages 13-14, jang2022serumandglucocorticoidregulated pages 14-15). Natural compounds like herbacetin have also been identified as SGK1 inhibitors, showing effects on cardiomyocyte hypertrophy and other cellular processes (jang2022serumandglucocorticoidregulated pages 13-14). In addition, experimental agents like EMD638683 have been used to explore the role of SGK1 in hypertension by reducing phosphorylation of downstream targets such as NDRG1 and by lowering blood pressure in animal models (lou2016serumandglucocorticoid pages 5-8). Clearly, aberrant SGK1 activity is implicated in diverse pathologies including hypertension, diabetic nephropathy, certain cancers (such as breast, prostate and colorectal cancer) and neurodegenerative disorders. Genetic variants or dysregulated expression of SGK1 have been associated with increased salt sensitivity, altered blood pressure regulation, and metabolic dysfunction (jang2022serumandglucocorticoidregulated pages 17-17, lou2016serumandglucocorticoid pages 27-27). There remains ongoing interest in developing more selective and potent SGK1 inhibitors for therapeutic applications, and such inhibitors are being actively studied in both cellular and animal models (sherk2008developmentofa pages 5-6, guerriero2020serumandglucocorticoidinducible pages 15-17).
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