1. Phylogeny  
   Serine/threonine‐protein kinase SIK2 belongs to the salt‐inducible kinase (SIK) subfamily within the larger AMP‐activated protein kinase (AMPK)–related kinase family. In mammals three SIK isoforms have been identified – SIK1, SIK2, and SIK3 – with SIK2 sharing high sequence and structural conservation with its paralogs. Orthologs of SIK2 are present throughout vertebrates and invertebrates, with the single SIK orthologue in Caenorhabditis elegans, known as KIN‐29, showing the closest similarity to SIK2, and insect species expressing both SIK2 and SIK3 (babbe2024identificationofhighly pages 1-2, darling2021nutsandbolts pages 1-2). Phylogenetic analyses based on the conserved catalytic domain and the overall architecture place SIK2 in an evolutionary core set of AMPK‐related kinases that have their origins in early eukaryotic evolution, and these kinases are integral members of the kinome found in almost every eukaryote (darling2021nutsandbolts pages 1-2, babbe2024identificationofhighly pages 1-2).
2. Reaction Catalyzed  
   SIK2 catalyzes the transfer of the γ‐phosphate group from ATP to the hydroxyl group of a serine or threonine residue on a protein substrate. The general chemical reaction can be described as:  
     ATP + [protein]–(L‐serine or L‐threonine) → ADP + [protein]–(L‐serine/threonine)‐phosphate + H⁺  
   This ATP‐dependent phosphorylation reaction is fundamental to its function as a serine/threonine kinase (mannion2016identifyingnovelsubstrates pages 22-25).
3. Cofactor Requirements  
   The catalytic activity of SIK2 requires the presence of divalent cations, most notably Mg²⁺, which is essential for proper alignment and binding of ATP in the catalytic pocket. As with other serine/threonine kinases, the magnesium ion serves as a critical cofactor ensuring efficient phosphoryl transfer (mannion2016identifyingnovelsubstrates pages 22-25).
4. Substrate Specificity  
   SIK2 exhibits substrate specificity for serine/threonine residues situated within preferred consensus sequences. In particular, SIK2 phosphorylates substrates that frequently contain motifs characterized by a hydrophobic or basic residue preceding the serine/threonine to be phosphorylated; one defined consensus motif in related kinases of this family is LX(R/K/H)(S/T)XSXXXL. Among its well‐characterized substrates, SIK2 phosphorylates insulin receptor substrate‐1 (IRS1) at serine 794 in insulin‐stimulated adipocytes, which may modulate insulin signal transduction (information provided, PubMed:23322770, PubMed:26983400). SIK2 also phosphorylates the CREB‐regulated transcription coactivators (TORCs) thereby inhibiting CREB activity, and it phosphorylates EP300, consequently inhibiting its histone acetyltransferase function; both events ultimately regulate the DNA binding ability of transcription factors such as PPARA and MLXIPL (information provided, babbe2024identificationofhighly pages 1-2, darling2021nutsandbolts pages 12-14).
5. Structure  
   SIK2 contains a highly conserved N‐terminal kinase domain typical of AMPK‐related kinases, which is responsible for its catalytic activity. Within this domain, critical features include an activation loop that requires phosphorylation by the upstream kinase LKB1 at Thr175, a small threonine gatekeeper residue (Thr96) positioned near the ATP‐binding site, and a charged glutamic acid (Glu103) that contributes to substrate and inhibitor specificity (babbe2024identificationofhighly pages 2-3). Beyond the kinase domain, SIK2 harbors a centrally located ubiquitin‐associated (UBA) domain whose primary role is believed to be in maintaining proper protein conformation and possibly modulating catalytic activity through domain‐domain interactions. The extensive, largely unstructured C‐terminal tail of SIK2 contains multiple phosphorylation sites that serve as regulatory motifs for interaction with 14-3-3 proteins and other signaling proteins. Although high-resolution crystallographic data for SIK2 itself are limited, homology models based on available structures of other SIK isoforms (notably SIK3) and structure‐based design studies have delineated key determinants of inhibitor binding in the ATP‐binding pocket and surrounding regions (babbe2024identificationofhighly pages 2-3, tesch2021structurebaseddesignof pages 22-26, oster2024thestructuresof pages 1-2).
6. Regulation  
   The activity of SIK2 is tightly regulated by multiple post-translational modifications. Full activation of SIK2 is achieved through phosphorylation of the activation loop residue Thr175 by the upstream kinase LKB1, a modification that is necessary for productive catalytic activity (babbe2024identificationofhighly pages 1-2, darling2021nutsandbolts pages 2-4). In contrast, elevated levels of cyclic AMP lead to activation of protein kinase A (PKA), which phosphorylates SIK2 at several sites—including Ser343, Ser358, Thr484, and Ser587—in its C-terminal tail. Phosphorylation at these sites induces binding of 14-3-3 proteins, resulting in a conformational change that sequesters SIK2 in the cytoplasm and inhibits its access to substrates (shi2024understandingtheroles pages 2-3, darling2021nutsandbolts pages 2-4, babbe2024identificationofhighly pages 1-2). Additional modifications, such as autophosphorylation events (for example at Ser179, as observed in related isoforms), may further fine-tune the kinase’s activity and substrate recognition, although the full range of such events has not been completely elucidated (darling2021nutsandbolts pages 2-4). Thus, SIK2 operates as a nodal point where upstream signals mediated by LKB1 and PKA converge to modulate its activity and subcellular localization.
7. Function  
   SIK2 is ubiquitously expressed in multiple tissues and plays diverse roles in cellular metabolism, immune responses, and transcriptional regulation. In the context of glucose metabolism, SIK2 phosphorylates IRS1 at serine 794 in insulin‐stimulated adipocytes, thereby potentially modulating the efficiency of insulin signal transduction and influencing downstream metabolic pathways (information provided, PubMed:23322770, PubMed:26983400). Through its action on the CREB‐regulated transcription coactivators (TORCs), SIK2 inhibits CREB activity, which in turn affects the expression of genes involved in gluconeogenesis and lipogenesis. Moreover, its phosphorylation of EP300 leads to suppressed histone acetyltransferase activity, thereby altering the DNA-binding capacity of transcription factors such as PPARA and MLXIPL. In addition to these roles in metabolic regulation, SIK2 is implicated in the control of autophagy and fatty acid oxidation, contributing to overall cellular energy homeostasis. Emerging evidence also supports its involvement in the modulation of innate immune responses through the regulation of cytokine expression, in part via its effects on transcriptional coactivators that control gene expression programs related to inflammation (babbe2024identificationofhighly pages 1-2, darling2021nutsandbolts pages 12-14, feng2023rolesofsalt‑inducible pages 1-2). By phosphorylating a variety of substrates that serve as mediators of transcription and metabolic flux, SIK2 functions as a critical integrator, coordinating signals from metabolic, hormonal, and immune pathways. Additionally, by similarity to other SIK family members, SIK2 is implicated in thymic T-cell development. Its broad substrate repertoire and regulatory capacity thus underscore its importance in maintaining cellular homeostasis and in orchestrating responses to metabolic and environmental stimuli.
8. Other Comments  
   Several small-molecule inhibitors targeting SIK2 have been developed through structure-based design and high-throughput screening approaches. Notable compounds include pan-SIK inhibitors such as HG-9-91-01 and its more pharmacokinetically favorable derivative YKL-05-099, as well as inhibitors like ARN-3236, which have demonstrated the capacity to modulate SIK2 activity in experimental models of inflammation and cancer (sundberg2016developmentofchemical pages 1-3, tesch2021structurebaseddesignof pages 1-4). Clinically approved tyrosine kinase inhibitors such as dasatinib and bosutinib have also been reported to inhibit SIK2 effectively at nanomolar concentrations, thereby influencing cytokine secretion profiles and contributing to anti-inflammatory effects (darling2021nutsandbolts pages 6-8, oster2024thestructuresof pages 7-8). Dysregulation of SIK2 has been associated with various disease states including metabolic disorders (such as obesity-induced insulin resistance and type 2 diabetes), cancer (notably ovarian and prostate malignancies where altered SIK2 activity affects cell cycle progression and metabolic reprogramming), and immune dysfunctions. As such, SIK2 represents a potential therapeutic target for a spectrum of diseases involving aberrant metabolic signaling and inflammatory responses (babbe2024identificationofhighly pages 1-2, sun2020thepotentroles pages 9-11, feng2023rolesofsalt‑inducible pages 1-2).
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