1. Phylogeny  
   Uncharacterized serine/threonine‐protein kinase SBK3, also known as SGK3, SgK110, or SH3 domain‐binding kinase family member 3, belongs to the serum‐ and glucocorticoid‐regulated kinase (SGK) family, which is a subgroup of the broader AGC kinase superfamily (tessier2006serumandglucocorticoid‐regulated pages 1-2).  
   Within the human kinome, SGK3 is phylogenetically grouped alongside SGK1 and SGK2; all three isoforms share a conserved catalytic domain while differing in their regulatory modules, with SGK3 uniquely harboring an N‐terminal Phox homology (PX) domain that directs endosomal localization (bago2016thehvps34‐sgk pages 1-2).  
   Comparative genomic analyses, as performed by Hanks and colleagues, indicate that the catalytic domain of SGK3 is conserved across mammalian species and is traceable to early eukaryotic ancestors, reflecting its long‐term evolutionary conservation in signaling networks (hanks2003genomicanalysisof pages 5-6).  
   Phylogenetic mapping based on kinase domain sequence similarity positions SGK3 in close evolutionary relation to the protein kinase B (Akt) family, reinforcing its role as a key AGC kinase with shared substrate attributes and regulatory kinases such as PDK1 and mTORC2 (sommer2014exploringthecontribution pages 47-52).
2. Reaction Catalyzed  
   SBK3 catalyzes the transfer of a phosphate group from ATP to target serine or threonine residues located on substrate proteins, following the classical reaction mechanism of serine/threonine kinases (hanks2003genomicanalysisof pages 1-2).  
   The chemical reaction can be described as follows: ATP + protein-(L-serine or L-threonine) → ADP + protein-(L-serine/threonine)-phosphate + H⁺, which is a reaction typical of kinases in the AGC family (tessier2006serumandglucocorticoid‐regulated pages 2-5).
3. Cofactor Requirements  
   The catalytic activity of SBK3 is dependent on the presence of divalent metal ions, with Mg²⁺ being the principal cofactor that facilitates the binding of ATP and thereby supports efficient phosphate transfer to substrates (hanks2003genomicanalysisof pages 2-3).  
   Mg²⁺ ions play a critical role in stabilizing the transition state during the phosphotransfer reaction, a feature that is consistent with the biochemical requirements of serine/threonine kinases in the AGC family (tessier2006serumandglucocorticoid‐regulated pages 2-5).
4. Substrate Specificity  
   SBK3 exhibits a substrate specificity that is reminiscent of other AGC family kinases, with its catalytic domain recognizing consensus motifs that typically include an arginine-rich sequence, for example, an RxRxx[S/T] motif (bago2016thehvps34‐sgk pages 8-9).  
   Experimental observations have demonstrated that SBK3 phosphorylates substrates such as NDRG1 (notably at threonine 346) and TSC2, thereby modulating downstream signaling pathways linked to mTORC1 activation (bago2016thehvps34‐sgk pages 8-9, bago2016thehvps34‐sgk pages 10-11).  
   The overlap between SGK3 and Akt substrate specificities suggests that SBK3 preferentially targets serine/threonine residues within substrates that contain basic amino acids at defined positions relative to the phosphorylatable residue, a characteristic pattern that underpins its biological function (bago2016thehvps34‐sgk pages 15-16).
5. Structure  
   Structurally, SBK3 comprises a central catalytic kinase domain that exhibits the typical bilobal architecture found in AGC kinases, with a smaller N-terminal lobe primarily involved in ATP binding and a larger C-terminal lobe dedicated to substrate interaction (goldsmith2007substrateanddocking pages 3-4).  
   A unique structural feature of SBK3 is the presence of an N-terminal Phox homology (PX) domain, which binds specifically to phosphatidylinositol 3-phosphate (PtdIns(3)P) and is responsible for targeting the kinase to endosomal membranes; this domain is absent in other SGK isoforms such as SGK1 and SGK2 (bago2016thehvps34‐sgk pages 1-2, bruhn2013aktindependentpi3ksignaling pages 6-7).  
   The catalytic domain of SBK3 contains key regulatory elements including the activation loop where Thr320 is phosphorylated by PDK1, and a hydrophobic motif, which includes Ser486 and is phosphorylated by mTORC2; these modifications are essential for the full activation of the kinase (bago2016thehvps34‐sgk pages 5-6, bago2016thehvps34‐sgk pages 3-5).  
   Additional structural features characteristic of AGC kinases, such as a conserved C-helix and a hydrophobic spine, contribute to the stabilization of the active conformation of SBK3 and facilitate its substrate binding and catalysis (goldsmith2007substrateanddocking pages 2-3, hanks2003genomicanalysisof pages 4-5).
6. Regulation  
   The activity of SBK3 is tightly regulated through a series of phosphorylation events that serve to modulate its catalytic function; phosphorylation at the T-loop (Thr320) by phosphoinositide-dependent kinase 1 (PDK1) is necessary for initial activation, while subsequent phosphorylation at the hydrophobic motif (Ser486) by mTORC2 further enhances its enzymatic activity (bago2016thehvps34‐sgk pages 5-6, bago2016thehvps34‐sgk pages 6-7).  
   Binding of phosphatidylinositol 3-phosphate to the PX domain is a critical regulatory step that facilitates the proper subcellular localization of SBK3 to endosomes, where it is efficiently phosphorylated by PDK1; this lipid-binding event serves as an essential prerequisite for kinase activation (bago2016thehvps34‐sgk pages 6-7).  
   In response to prolonged inhibition of the PI3K/Akt signaling pathway, SBK3 activity is upregulated as a compensatory mechanism to restore phosphorylation of select Akt substrates, including those involved in mTORC1 activation (bago2016thehvps34‐sgk pages 1-2, bago2016thehvps34‐sgk pages 15-16).  
   Selective small-molecule inhibitors such as compound 14h have been developed, which specifically target SBK3, reducing its phosphorylation activity and providing insights into its regulatory dynamics within cellular signaling networks (bago2016thehvps34‐sgk pages 8-9, bruhn2013aktindependentpi3ksignaling pages 6-7).
7. Function  
   SBK3 functions as a serine/threonine kinase that plays a critical role in cell signaling pathways governing cell proliferation, survival, and metabolism; one of its key activities is the phosphorylation of substrates such as NDRG1 and the tuberous sclerosis complex protein TSC2, both of which are linked to the regulation of mTORC1 activity (bago2016thehvps34‐sgk pages 8-9, bago2016thehvps34‐sgk pages 10-11).  
   Through its ability to modulate mTORC1 signaling, SBK3 contributes to the cellular protein synthesis machinery, thereby influencing cell growth and metabolic responses, particularly under conditions where the PI3K/Akt pathway is inhibited (bago2016thehvps34‐sgk pages 13-14, bago2016thehvps34‐sgk pages 21-21).  
   Elevated SBK3 expression and activity have been observed in certain breast cancer cell lines following prolonged PI3K/Akt inhibitor treatment, suggesting that it may act as a compensatory kinase that supports tumor cell survival and proliferation in the face of targeted therapeutic interventions (bago2016thehvps34‐sgk pages 3-5, bago2016thehvps34‐sgk pages 15-16).  
   In addition to its role in oncogenic signaling, SBK3 has been implicated in broader cellular processes such as the regulation of ion transport and nutrient sensing, owing to its structural similarity and overlapping substrate specificity with Akt kinases (liao2022contributionsofsgk3 pages 14-14, bruhn2013aktindependentpi3ksignaling pages 6-7).
8. Other Comments  
   Several small-molecule inhibitors that target SBK3 have been identified; among these, compound 14h has demonstrated high potency and selectivity, with an approximate IC50 of 4 nM for SBK3 inhibition, and is used to dissect the kinase’s role in modulating mTORC1 signaling in cancer cells (bago2016thehvps34‐sgk pages 8-9, bago2016thehvps34‐sgk pages 15-16).  
   SBK3’s ability to sustain mTORC1 activity under conditions of PI3K/Akt inhibition positions it as a potential therapeutic target in cancers that exhibit resistance to conventional PI3K/Akt pathway inhibitors, a finding that has significant implications for the design of combination therapeutic strategies (bago2016thehvps34‐sgk pages 1-2, bago2016thehvps34‐sgk pages 9-10).  
   Although detailed mutational analyses and disease-specific polymorphism studies for SBK3 have not been extensively reported in the current literature, its functional profile as a compensatory kinase underscores its potential involvement in tumor progression and drug resistance, warranting further investigation into its role in clinical settings (hanks2003genomicanalysisof pages 5-6, tessier2006serumandglucocorticoid‐regulated pages 16-17).
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