1. Phylogeny  
   Death‐associated protein kinase 3 (DAPK3), also known as ZIP kinase, belongs to the death‐associated protein kinase (DAPK) family, a conserved group of serine/threonine kinases present across metazoans. DAPK3 shares approximately 80% identity in its catalytic domain with other family members such as DAPK1 and DAPK2, and its evolutionary conservation is supported by orthologs found in a range of vertebrate species (elbadawy2018novelfunctionsof pages 1-3, gozuacik2006dapkproteinfamily pages 1-2). As part of the broader kinome, DAPK3 is categorized within the Ca²⁺/calmodulin‐dependent protein kinase group despite distinct regulatory differences, particularly due to the absence of a Ca²⁺/calmodulin binding domain in DAPK3 compared to DAPK1 and DAPK2 (shani2004deathassociatedproteinkinase pages 1-2, tur2017restorationofdap pages 1-3). Its grouping within the DAPK family reflects common ancestral origins and ties its functional roles in programmed cell death to an evolutionary core set of kinase‐mediated signaling pathways (levystrumpf1998deathassociatedproteins pages 2-3).
2. Reaction Catalyzed  
   DAPK3 catalyzes the phosphorylation of protein substrates by transferring a phosphate group from ATP onto serine or threonine residues within those substrates. The chemical reaction catalyzed can be described by the equation: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (elbadawy2018novelfunctionsof pages 10-12, carlson2013fluorescencelinkedenzyme pages 8-10).
3. Cofactor Requirements  
   The kinase activity of DAPK3 requires ATP binding in a reaction that is dependent on the presence of divalent metal ions, most notably Mg²⁺. In contrast to other DAPK family members that are activated by Ca²⁺/calmodulin binding, DAPK3 lacks the Ca²⁺/calmodulin regulatory domain and is not dependent on Ca²⁺/calmodulin for its catalytic activity (elbadawy2018novelfunctionsof pages 1-3, farag2019death‐associatedproteinkinase pages 4-6).
4. Substrate Specificity  
   DAPK3 exhibits specificity for serine/threonine residues in protein substrates involved in cytoskeletal regulation and apoptosis. In smooth muscle cells, DAPK3 phosphorylates myosin regulatory light chains, such as MYL12B and MYL9, thereby directly regulating contractile function; in addition, it phosphorylates PPP1R12A (the regulatory subunit of smooth muscle myosin phosphatase) to inhibit phosphatase activity and enhance muscle contraction (elbadawy2018novelfunctionsof pages 14-15, farag2019death‐associatedproteinkinase pages 28-30). In non‐muscle cells, phosphorylation of myosin light chains by DAPK3 contributes to reorganization of the actin cytoskeleton and condensation of actin stress fibers. Furthermore, DAPK3 can phosphorylate substrates such as histone H3 and the pro‐apoptotic protein Par4, linking its substrate specificity to both transcriptional regulation and apoptotic signaling (shani2004deathassociatedproteinkinase pages 13-14, scheidtmann2007dlkzipkinasea pages 4-5).
5. Structure  
   The human DAPK3 protein comprises 454 amino acids and is organized into a distinct domain architecture. At the N-terminus, DAPK3 contains a catalytic kinase domain, approximately 275 amino acids in length, that adopts the canonical bilobal structure typical of serine/threonine kinases; this domain includes key structural features such as the N-terminal lobe with β-sheets, a C-terminal lobe rich in α-helices, an activation loop that harbors critical autophosphorylation sites (e.g., Thr180, Thr225, and Thr265), and a C-helix essential for catalytic activity (scheidtmann2007dlkzipkinasea pages 2-4, temmerman2013structuralandfunctional pages 6-7). Unlike DAPK1 and DAPK2, DAPK3 does not possess a Ca²⁺/calmodulin-binding domain; instead, its C-terminal region contains a nuclear localization signal and a leucine zipper motif, which mediate dimerization or oligomerization and contribute to the regulation of subcellular localization and enzymatic activity (tur2017restorationofdap pages 3-5, shani2004deathassociatedproteinkinase pages 1-2). This unique regulatory region distinguishes DAPK3 within the DAPK family and has been implicated in the modulation of apoptosis and cytoskeletal dynamics. The 3D structure, as inferred from experimentally derived models and structural analyses, reveals a hydrophobic spine and an active site cleft lined by conserved catalytic residues, features that are critical for its ATP-dependent phosphotransferase activity (scheidtmann2007dlkzipkinasea pages 5-7, temmerman2013structuralandfunctional pages 6-7).
6. Regulation  
   Regulation of DAPK3 activity is primarily mediated via phosphorylation events and oligomerization dynamics. Autophosphorylation at key residues—including Thr180, Thr225, and Thr265—is essential for attaining full catalytic activity (farag2019death‐associatedproteinkinase pages 30-31, temmerman2013structuralandfunctional pages 6-7). In addition, DAPK3 is phosphorylated by upstream kinases such as DAPK1; this phosphorylation event forms a kinase hierarchy that facilitates the activation of DAPK3 and enhances its pro-apoptotic signaling (shani2004deathassociatedproteinkinase pages 11-13). The phosphorylation state also influences subcellular localization; for instance, phosphorylated forms of DAPK3 tend to localize preferentially within the cytoplasm, where they mediate membrane blebbing and actin cytoskeleton reorganization, while dephosphorylated forms may accumulate in the nucleus (tur2017restorationofdap pages 3-5, shani2004deathassociatedproteinkinase pages 14-16). Moreover, cancer-associated loss-of-function mutations, which have been reported in DAPK3, impair its catalytic function and exert dominant-negative effects when co-expressed with the wild-type protein (brognard2011cancerassociatedlossoffunctionmutations pages 1-2, brognard2011cancerassociatedlossoffunctionmutations pages 5-7). These post-translational modifications and mutational events modulate DAPK3 activity, altering its role in apoptosis and cytoskeletal organization.
7. Function  
   DAPK3 serves multiple cellular roles that span the regulation of apoptosis, autophagy, transcription, translation, and actin cytoskeleton reorganization. Functionally, DAPK3 is a serine/threonine kinase that participates in both type I (caspase-dependent) and type II (caspase-independent) cell death pathways. In smooth muscle cells, DAPK3 contributes to the regulation of muscle contraction by phosphorylating myosin regulatory light chains (MYL12B and MYL9) directly, or indirectly by phosphorylating PPP1R12A to inhibit smooth muscle myosin phosphatase (SMPP1M), thereby enhancing Ca²⁺ sensitivity and maintaining a contractile state (elbadawy2018novelfunctionsof pages 14-15, farag2019death‐associatedproteinkinase pages 28-30). In non-muscle cells, DAPK3-mediated phosphorylation of myosin leads to condensation of actin stress fibers and reorganization of the actin cytoskeleton, contributing to changes in cell morphology (elbadawy2018novelfunctionsof pages 3-5, tur2017restorationofdap pages 3-5). Beyond its role in cytoskeletal regulation, DAPK3 is implicated in the control of transcription and translation by phosphorylating nuclear targets, and it functions in starvation-induced autophagy, which is critical under conditions of nutrient deprivation (elbadawy2018novelfunctionsof pages 1-3, farag2019death‐associatedproteinkinase pages 26-28). Through these diverse functions, DAPK3 acts as a key regulator of cell fate, integrating signals from apoptosis and autophagy pathways to control cellular responses under varying physiological conditions.
8. Other Comments  
   Selective inhibitors for DAPK3 have been identified using structure-based virtual screening approaches; for example, HS38 is a potent, ATP-competitive inhibitor that demonstrates high affinity for DAPK3 and related family members, and its development has provided insights into inhibitor design strategies targeting the ATP-binding site (carlson2013fluorescencelinkedenzyme pages 3-4, carlson2013fluorescencelinkedenzyme pages 8-10). Furthermore, cancer-associated mutations in DAPK3, such as T112M, D161N, and P216S, have been reported; these mutations result in loss-of-function and are associated with impaired pro-apoptotic signaling, providing a mechanistic link to tumorigenesis (brognard2011cancerassociatedlossoffunctionmutations pages 1-2, brognard2011cancerassociatedlossoffunctionmutations pages 5-7). In addition to its established role in apoptosis and autophagy, DAPK3 has been implicated in the regulation of vascular smooth muscle tone and inflammatory responses, suggesting potential applications in the treatment of cardiovascular and neurodegenerative diseases (farag2019death‐associatedproteinkinase pages 19-22, elbadawy2018novelfunctionsof pages 12-14). Currently, the development of more selective and potent inhibitors for DAPK3 remains an area of active research, with ongoing efforts to better profile its inhibitor spectrum using chemoproteomic strategies (carlson2013fluorescencelinkedenzyme pages 3-4). Overall, DAPK3’s multifaceted roles in regulating cytoskeletal dynamics, apoptosis, and autophagy, combined with its unique structural features, underscore its importance as a signaling node and a potential therapeutic target.
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