1. Phylogeny  
   Serine/threonine-protein kinase N3 (PKN3), also designated as PKN‐β or Protein-kinase C-related kinase 3, is a member of the PKN subfamily within the larger AGC kinase group. The AGC kinases constitute a major branch of the serine/threonine kinome and are evolutionarily conserved throughout eukaryotes. PKN3 exists alongside two other paralogous isoforms—PKN1 and PKN2—and these kinases share common catalytic domains as well as regulatory modules that are derived from a common ancestral gene. Comparative analyses based on sequence homology and conserved domain architecture indicate that the PKN family diverged early during the evolution of eukaryotic protein kinases, with orthologs in mammals displaying a conserved central kinase domain and variable N-terminal regulatory regions that mediate interactions with small GTPases and lipids (collazos2011siterecognitionand pages 1-3, browne2018achemoproteomicstrategy pages 18-19). Although complete phylogenetic trees with detailed branch lengths are not provided in the reviewed studies, available data clearly place PKN3 within an evolutionary core set of AGC kinases that includes protein kinase C (PKC), PDK1, and AKT. This grouping is consistent with the pioneering analyses by Manning et al., which traced key kinase families back to early eukaryotic evolution. In mammals, orthologs of PKN3 have been identified and are noted for their tissue-specific expression patterns that differ from those of PKN1 and PKN2. Importantly, PKN3 is distinguished from its family members by the presence of a reactive cysteine residue (C840) located proximal to its catalytic active site—a feature that is absent in PKN1 and PKN2 and that contributes to its unique inhibitor engagement profile (browne2018achemoproteomicstrategy pages 4-6, collazos2011siterecognitionand pages 7-9). In addition, the evolutionary history of PKN3 reflects gene duplication events followed by divergence that have enabled the specialization of cellular functions, particularly in signaling pathways that regulate cell adhesion, motility, and invasive behavior. The conserved catalytic domains of PKN isoforms demonstrate significant sequence similarity with other AGC kinases, affirming that PKN3 evolved from an ancestral kinase that already possessed the fundamental features necessary for ATP binding, substrate recognition, and phosphorylation, while also acquiring unique regulatory residues through evolutionary adaptation (collazos2011siterecognitionand pages 1-3). An appreciation of these evolutionary relationships emphasizes the functional significance of PKN3 among the serine/threonine kinases and supports the notion that its distinct regulatory mechanisms and substrate preferences have been preserved among vertebrates (browne2018achemoproteomicstrategy pages 18-19).
2. Reaction Catalyzed  
   PKN3 catalyzes the transfer of a phosphate group from adenosine triphosphate (ATP) to the hydroxyl group of serine and threonine residues in substrate proteins. The enzymatic reaction can be summarized as follows:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction is representative of the canonical activity of serine/threonine kinases and is crucial for the regulation of various cellular processes via reversible phosphorylation, as characterized in substrate assays for the PKN family (collazos2011siterecognitionand pages 3-5).
3. Cofactor Requirements  
   The catalytic efficiency of PKN3, like that of most protein kinases, is dependent on the presence of divalent metal ions. In vitro and cell-based kinase assays indicate that PKN3 requires magnesium ions (Mg²⁺) as a cofactor to facilitate ATP binding and proper catalysis of phosphoryl transfer reactions (browne2018achemoproteomicstrategy pages 7-9, collazos2011siterecognitionand pages 3-5).
4. Substrate Specificity  
   Substrate recognition by PKN3 is defined by a distinct amino acid sequence motif surrounding the phosphorylated serine or threonine residue. Biochemical assays and peptide array experiments have demonstrated that PKN3 shares substrate specificity features with its paralog PKN1, with both kinases exhibiting a marked preference for an arginine residue positioned at the –3 site relative to the target serine/threonine. Detailed peptide library experiments have led to the formulation of consensus motifs that distinguish the substrates of the PKN family. For instance, one study reports that the substrates of PKN3 conform to a motif described as “x-x-Arg-x-Ne/Z-Ser-Ho-x-x-x-Ne,” where “Ne/Z” indicates either neutral or acidic residues and “Ho” signifies hydrophobic residues adjacent to the phosphorylation site (collazos2011siterecognitionand pages 5-7). Such preferences ensure selective substrate engagement and contribute to the fidelity of downstream signaling cascades. In addition, substrate screens have revealed that the overall phosphorylation consensus is shaped not only by the presence of an arginine but also by the interplay of flanking amino acids that may adopt conformations conducive to binding within the catalytic cleft of PKN3. These biochemical studies further indicate that while there is substantial overlap in the substrates recognized by PKN1 and PKN3, subtle differences in motif preferences highlight the specialization of PKN3 in specific cellular contexts, particularly those related to carcinoma progression (collazos2011siterecognitionand pages 7-9, browne2018achemoproteomicstrategy pages 11-12). The substrate specificity of PKN3 thus contributes to its role in phosphorylating target proteins that are involved in cytoskeletal rearrangements, adhesion complexes, and other components of the cell migration machinery. Such precision in substrate selection is central to properly relaying signals downstream of PI3-kinase activation and Rho small GTPase engagement, pathways that are critical to cancer cell invasiveness (collazos2011siterecognitionand pages 5-7).
5. Structure  
   PKN3 is characterized by a modular architecture that is typical of eukaryotic serine/threonine kinases. Central to its function is the conserved catalytic kinase domain, which is organized into an N-terminal lobe and a larger C-terminal lobe, forming a bilobal structure that houses the ATP binding site and substrate recognition surfaces. The N-terminal lobe contains the glycine-rich loop, which plays a key role in ATP anchoring, and the critical C-helix, whose positioning influences the active conformation of the kinase. The C-terminal lobe, in turn, contains the activation loop that modulates substrate binding and catalytic turnover. A unique structural hallmark of PKN3 is the presence of a reactive cysteine residue at position 840 (C840) located adjacent to the ATP-binding pocket. This cysteine is the target of covalent inhibitors such as THZ1 and JZ128, and its modification has been shown to potently inhibit kinase activity. Notably, substitution of C840 with a serine residue (C840S mutation) abolishes covalent inhibitor binding, thereby underscoring the critical role of this residue in defining the unique inhibitor sensitivity profile of PKN3 (browne2018achemoproteomicstrategy pages 4-6, browne2018achemoproteomicstrategy pages 27-27).

In addition to the kinase domain, PKN3 likely harbors regulatory regions that modulate its spatial and temporal activity within the cell. Although the full-length three-dimensional structure of PKN3 has not been experimentally determined, the domain organization inferred from conserved sequence motifs and AlphaFold-based models suggests the existence of N-terminal regions that may mediate interactions with small GTPases—particularly those of the Rho family—and lipid-binding modules that could facilitate membrane association. Such domains add an additional layer of regulation by controlling subcellular localization and by participating in auto-inhibitory interactions. The overall structural paradigm of PKN3 aligns closely with that observed for other AGC kinases, where key structural elements such as the DFG motif, catalytic lysine, and activation loop contribute to a finely tuned balance between the inactive and active conformations of the kinase. Experimental insights derived from chemoproteomic approaches have further highlighted the importance of the active site configuration and the spatial arrangement of hydrophobic spines, which in canonical kinases stabilize the active conformation via non-contiguous hydrophobic residues spanning both lobes (browne2018achemoproteomicstrategy pages 7-9, rakette2012structuralanalysisof pages 4-6). Although direct crystallographic data for PKN3 are limited, comparisons with structurally characterized kinases have provided valuable insights into its potential three-dimensional organization, including the alignment and positioning of the C-helix and activation loop that are critical for catalytic function (collazos2011siterecognitionand pages 1-3). Overall, the structural attributes of PKN3—especially the disposition of C840 within the catalytic domain—are central to its differential inhibitor sensitivity and its role in modulating signaling pathways implicated in oncogenesis.

1. Regulation  
   Regulation of PKN3 activity is mediated by multiple factors that converge on its catalytic domain and regulatory regions. Key post-translational modifications such as phosphorylation play an essential role in modulating its kinase activity. PKN3 is activated downstream of PI3-kinase signaling and can form complexes with small GTPases, including RhoC, which facilitate conformational changes required for full activation. These interactions are further modulated by lipid cofactors, most notably arachidonic acid, which can enhance kinase activity in vitro by promoting alterations in the regulatory domains that control the catalytic core (collazos2011siterecognitionand pages 11-13, browne2018achemoproteomicstrategy pages 7-9). In addition, autophosphorylation events within the activation loop have been observed in chemoproteomic studies and are implicated in stabilizing the active conformation required for substrate phosphorylation (collazos2011siterecognitionand pages 15-18).

An important aspect of PKN3 regulation is the targeting of its active site by covalent inhibitors. Inhibitor compounds such as THZ1 and the more selective JZ128 have been shown to covalently modify the key cysteine residue at position 840. Binding studies, including competitive inhibitor assays and mutagenesis experiments, have demonstrated that the covalent attachment at C840 is both necessary and sufficient to inhibit kinase activity in vitro and in cellular contexts (browne2018achemoproteomicstrategy pages 4-6, browne2018achemoproteomicstrategy pages 27-27). These findings highlight the potential for allosteric and direct active-site regulation of PKN3 by small molecules. Moreover, regulatory mechanisms may also involve additional protein–protein interactions mediated by specific domains in the non-catalytic regions of the protein. For example, the interaction with Rho GTPases not only influences kinase activation but has also been implicated in controlling subcellular localization, which is essential for the spatial regulation of downstream signaling events (collazos2011siterecognitionand pages 1-3, browne2018achemoproteomicstrategy pages 7-9). Although the full spectrum of regulatory enzymes—such as phosphatases that may dephosphorylate PKN3—has not been completely elucidated, the known regulatory inputs from upstream kinases and binding partners underscore the multi-layered regulation of PKN3 activity that is critical for its function in modulating cell adhesion and migratory processes.

1. Function  
   PKN3 plays a pivotal role in mediating cellular processes that contribute to tumor aggressiveness and metastasis. Its involvement in regulating cellular invasiveness is particularly well documented in the context of malignant prostate cancer, where both genetic and proteomic studies have implicated PKN3 in promoting cell migration and tissue invasion. At the molecular level, PKN3 functions downstream of PI3-kinase signaling pathways and is activated in response to upstream cues such as Rho GTPase engagement. Once activated, PKN3 phosphorylates a range of substrates that are involved in the dynamic regulation of the actin cytoskeleton and adherens junctions. This control over cytoskeletal reorganization is critical for the processes of cell motility and invasion, which are hallmarks of metastatic cancer (browne2018achemoproteomicstrategy pages 18-19, collazos2011siterecognitionand pages 1-3).

In experimental systems, depletion or knockdown of PKN3 has been associated with reduced tumor growth and metastasis in models of prostate and pancreatic cancer. Functional assays, including wound healing experiments and cell migration studies, have demonstrated that inhibition of PKN3—either by genetic means or through the use of selective covalent inhibitors—leads to impaired cell motility, thereby confirming its role in the regulation of invasive behavior (browne2018achemoproteomicstrategy pages 23-27, collazos2011siterecognitionand pages 15-18). Moreover, phosphoproteomic analyses have identified several putative substrates of PKN3 that are enriched in proteins associated with cell junctions, such as components of the adherens junction complex. These substrates include proteins with roles in cytoskeletal rearrangement and signal transduction, which further buttress the function of PKN3 as a modulator of cell structural dynamics.

Furthermore, PKN3 is of particular interest in oncogenic signaling due to its selective expression patterns; while it is less studied than other kinases, its upregulation and functional activity have been strongly correlated with the progression of malignancy. The kinase’s contributions to processes such as angiogenesis and bone resorption, as well as its involvement in malignant prostate cell growth downstream of PI3-kinase, underscore its significance as a therapeutic target. Overall, the function of PKN3 is intricately linked to the control of signal transduction pathways that govern cell morphology, migration, and tissue invasion, all of which are central to the pathology of solid tumors and metastatic disease (browne2018achemoproteomicstrategy pages 11-12, collazos2011siterecognitionand pages 7-9).

1. Other Comments  
   Experimental efforts have led to the identification of selective inhibitors for PKN3 that operate via covalent modification of the critical C840 residue in the kinase domain. Notably, compounds such as JZ128 have been developed as first-in-class selective inhibitors that demonstrate an IC50 in the low nanomolar range in vitro, and their selectivity has been confirmed by differential binding in cell-based assays and via chemoproteomic profiling (browne2018achemoproteomicstrategy pages 4-6, browne2018achemoproteomicstrategy pages 27-27). In contrast, structural homologs within the PKN family—such as PKN1 and PKN2—lack an equivalently positioned cysteine residue and are therefore not susceptible to the same covalent inhibition strategy. This unique inhibitor sensitivity profile not only represents a valuable pharmacological tool but also underscores the potential of PKN3 as a therapeutic target in cancer.  
   PKN3 is closely associated with disease pathogenesis, particularly in the context of prostate cancer, where its activity correlates with enhanced tumor invasiveness and metastatic potential. Clinical investigations, including trials exploring liposomal siRNA-based therapeutics targeting PKN3, further highlight its relevance as a drug target in advanced solid tumors and metastatic pancreatic cancer (browne2018achemoproteomicstrategy pages 4-6, browne2018achemoproteomicstrategy pages 18-19). While the complete spectrum of PKN3 mutations in clinical specimens has not been fully delineated, mutational analysis of the kinase active site—especially the integrity of the C840 residue—has been crucial for understanding inhibitor binding and resistance mechanisms. As research continues to advance, additional selective inhibitors may be developed, and in-depth structural and biochemical characterization will further refine our understanding of PKN3’s role in oncogenic signaling networks.
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