1. Phylogeny  
   TLK2 is a highly conserved serine/threonine protein kinase that, together with its close paralog TLK1, belongs to the Tousled‐like kinase (TLK) family. Mammalian TLK2 is evolutionarily related to the original Tousled (TSL) kinase first discovered in Arabidopsis thaliana, and its orthologs have been identified in diverse eukaryotic species including invertebrates such as Drosophila melanogaster and Caenorhabditis elegans, as well as in mammals and plants (bayona2018roleofthe pages 40-42, segurabayona2019thetousledlikekinases pages 1-3). TLK2 and TLK1 share high sequence similarity in their catalytic domains—up to 94% identity—and overall about 80–84% identity, indicating that they arise from a relatively recent gene duplication event in vertebrate evolution and are part of an evolutionarily ancient set of cell cycle regulators required for chromatin dynamics (buron2014theroleof pages 43-48, segurabayona2019thetousledlikekinases pages 3-4). Furthermore, the typical domain architecture, featuring coiled-coil motifs and a conserved kinase domain, emphasizes that TLK2 is embedded within a core network of eukaryotic kinases that regulate DNA replication and repair, with evolutionary relationships that trace back to the last eukaryotic common ancestor (LECA) (buron2014theroleof pages 127-131).
2. Reaction Catalyzed  
   TLK2 catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on target proteins. In this canonical kinase reaction, ATP and the substrate protein (usually one with serine/threonine acceptor sites) are converted into ADP and a phosphorylated protein along with the release of a proton (asquith2024discoveryandoptimization pages 1-3). The best‐characterized substrates for TLK2 are the histone chaperones ASF1A and ASF1B, which upon phosphorylation become stabilized and better able to facilitate nucleosome assembly during DNA replication and repair processes (asquith2024discoveryandoptimization pages 1-3, bayona2018roleofthe pages 42-52).
3. Cofactor Requirements  
   The catalytic activity of TLK2 is dependent on the presence of ATP as the phosphate donor, and like many protein kinases, it requires divalent metal ions—most notably Mg2+—to coordinate ATP binding and facilitate the phosphoryl transfer reaction (mortuza2018molecularbasisof pages 1-2). Although specific studies detailing additional cofactors or regulatory molecules are limited in the provided context, the typical kinase mechanism implies a requirement for Mg2+ and related ions to maintain the structure of the ATP binding pocket and stabilize the transition state during catalysis (bayona2018roleofthe pages 92-94).
4. Substrate Specificity  
   TLK2 exhibits substrate specificity primarily for serine/threonine residues in proteins associated with chromatin structure. Its best‐characterized substrates are the histone chaperones ASF1A and ASF1B, which play pivotal roles in histone deposition during DNA replication. Phosphorylation of ASF1 by TLK2 prevents its degradation via the proteasome and thereby enhances chromatin assembly (asquith2024discoveryandoptimization pages 1-3). Although the exact consensus sequence for TLK2 phosphorylation is not strictly rigid, experimental data from peptide array studies suggest that TLK2 may prefer substrates featuring acidic residues adjacent to the phosphorylation site, though this appears to be a component of a complex substrate recognition pattern that may also involve higher order structural determinants (buron2014theroleof pages 127-131, bayona2018roleofthe pages 96-101). Additional substrates proposed include proteins involved in DNA damage response such as RAD9 and potentially other proteins that contribute to replication fork stability. However, the strongest supporting evidence points to ASF1 as the canonical substrate whose phosphorylation is functionally linked to chromatin assembly and genome stability (segurabayona2019thetousledlikekinases pages 4-5, mortuza2018molecularbasisof pages 10-11).
5. Structure  
   TLK2 displays a modular architecture consisting of an N-terminal regulatory region and a C-terminal catalytic kinase domain. The N-terminal portion is largely disordered but harbors multiple coiled-coil motifs, which are critical for dimerization and oligomerization. These coiled-coil domains, especially the first coiled-coil (CC1), not only facilitate homo- and hetero-dimer formation with TLK1 but also contribute to the regulation of substrate access (bayona2018roleofthe pages 40-42, segurabayona2019thetousledlikekinases pages 3-4). The catalytic domain exhibits the conserved bilobal fold common to protein kinases, with an ATP binding pocket that shows key interactions with inhibitors such as oxindole derivatives; structural studies using X-ray crystallography have resolved the TLK2 kinase domain bound to ATP analogs, revealing distinct features such as deviations in the P-loop motif and extended activation segments (mortuza2018molecularbasisof pages 9-9, asquith2024discoveryandoptimization pages 9-11). Unique among many kinases is that while TLK2 undergoes autophosphorylation at multiple serine/threonine sites within both the N- and C-lobes, certain phosphorylation events—such as on S617, S686, and T695—are crucial for modulating its catalytic efficiency and structural stability; mutations at these residues have been shown to either inhibit or stabilize its activity (mortuza2018molecularbasisof pages 92-94, bayona2018roleofthe pages 90-92). In addition, TLK2 contains a C-terminal tail that may serve as a negative regulator of enzymatic activity in a fashion reminiscent of its paralog TLK1 (buron2014theroleof pages 122-127).
6. Regulation  
   The regulation of TLK2 is multifaceted, involving autophosphorylation, dimerization, and response to DNA damage. Autophosphorylation plays a central role in TLK2 activation, with distinct cis- and trans-phosphorylation events occurring within dimeric complexes that sequentially enhance its catalytic competence. For instance, phosphorylation on residues such as S617 immediately following the conserved DFG motif can inhibit activity when mimicked by negative charge, suggesting a finely tuned regulatory mechanism (mortuza2018molecularbasisof pages 90-92, bayona2018roleofthe pages 94-96). In addition to autophosphorylation, TLK2 is subject to regulation by checkpoint kinases; phosphorylation by CHK1 has been implicated in regulating the activity of TLK1, and similar mechanisms are proposed for TLK2 in the context of DNA damage responses (bayona2018roleofthe pages 178-188, segurabayona2019thetousledlikekinases pages 5-7). The oligomerization mediated by the N-terminal coiled-coil domains is also critical for its activation, as TLK2 functions optimally in a dimeric state that favors inter-molecular phosphorylation (mortuza2018molecularbasisof pages 11-13, bayona2018roleofthe pages 78-85). Furthermore, interactions with other proteins—such as the dynein light chain LC8 (DYNLL1)—can modulate its assembly and possibly fine-tune its substrate recognition; however, LC8 is not a direct phosphorylation substrate but rather serves as a multimerization hub (bayona2018roleofthe pages 160-167, segurabayona2019thetousledlikekinases pages 4-5). Regulation by environmental cues is evident in TLK2’s response to DNA damage and replication stress, where its activity is rapidly attenuated to allow proper checkpoint activation and repair, thereby linking its kinase function to the broader cellular DNA damage response (asquith2024discoveryandoptimization pages 45-46, segurabayona2019thetousledlikekinases pages 5-7).
7. Function  
   TLK2 plays a pivotal role in maintaining genome and epigenome stability through its regulation of chromatin dynamics during DNA replication, repair, transcription, and chromosome segregation. The kinase phosphorylates the histone chaperones ASF1A and ASF1B, thereby stabilizing these proteins and promoting efficient chromatin assembly by ensuring a sufficient supply of histones H3 and H4 during DNA replication (asquith2024discoveryandoptimization pages 1-3, bayona2018roleofthe pages 40-42). In doing so, TLK2 prevents the accumulation of single-stranded DNA, replication fork stalling, and subsequent DNA damage (mortuza2018molecularbasisof pages 3-4). Additionally, TLK2 has been implicated in the resolution of the G2/M checkpoint following DNA damage, with its activity ensuring timely recovery and progression into mitosis; this function has critical implications for genome integrity and cell cycle fidelity (segurabayona2019thetousledlikekinases pages 5-7, bayona2018roleofthe pages 178-188). Beyond its classical role in chromatin assembly, TLK2 is reported to act as a negative regulator of amino acid starvation-induced autophagy, thus linking its kinase activity to metabolic homeostasis (asquith2024discoveryandoptimization pages 1-3). The expression of TLK2 is ubiquitous, and although it predominantly localizes in the nucleus, splice variants and regulated nuclear export have been noted especially in differentiated neuronal cells, suggesting additional roles in cell differentiation and neuronal function (nuhusoso2025neuronaldifferentiationenhances pages 1-4). Furthermore, genetic studies have associated TLK2 mutations or altered expression with neurodevelopmental disorders—including autosomal dominant intellectual disability—and with oncogenic processes, particularly in cancers where TLK2 gene amplification correlates with poor clinical outcomes (OpenTargets Search: -TLK2, segurabayona2019thetousledlikekinases pages 9-10).
8. Other Comments  
   TLK2 is emerging as an attractive therapeutic target owing to its central role in genome stability and its implication in disease contexts ranging from cancer to neurodevelopmental disorders. Recent medicinal chemistry efforts, for example through the use of quantitative structure-activity relationships (QSAR), have led to the development of narrow spectrum inhibitors that target the ATP-binding pocket of TLK2, with oxindole-based compounds demonstrating potent activity and selectivity (asquith2024discoveryandoptimization pages 9-11, bayona2018roleofthe pages 54-56). These inhibitors take advantage of key interactions within the kinase domain—specifically with hinge-region residues—and have been characterized using kinome-wide selectivity profiling to confirm their narrow spectrum of inhibition (asquith2024discoveryandoptimization pages 9-11). In addition, TLK2’s regulation by protein–protein interactions (for example, its association with LC8 and its heteromerization with TLK1) and post-translational modifications provides multiple potential strategies for modulation. Genetic evidence also supports the involvement of TLK2 in conditions such as autosomal dominant intellectual disability and neurodevelopmental syndromes, making it a focal point for both basic research and drug discovery programs (OpenTargets Search: -TLK2, buron2014theroleof pages 131-138). Despite these advances, challenges remain, including the need for more-specific inhibitors and a deeper understanding of TLK2’s substrate recognition and regulation in different cellular contexts. Ongoing research continues to integrate structural biology, chemical genetics, and cell biology approaches to develop effective therapeutic strategies that target TLK2 (mortuza2018molecularbasisof pages 13-13, segurabayona2019thetousledlikekinases pages 12-13).
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