1. Phylogeny  
   CHK1, encoded by the CHEK1 gene (UniProt O14757), is a serine/threonine protein kinase that is highly conserved throughout eukaryotes, being found in model organisms such as Schizosaccharomyces pombe as well as in all higher vertebrates, which underscores its fundamental role in cell cycle checkpoint control and DNA damage response (caparelli2013regulatorymotifsin pages 1-2, nyberg2002towardmaintainingthe pages 3-5). Extensive kinome studies based on the seminal work by Manning and colleagues have classified CHK1 within the CMGC subgroup of the eukaryotic protein kinase superfamily—a group that also includes cyclin-dependent kinases (CDKs), mitogen-activated protein kinases (MAPKs), glycogen synthase kinase 3 (GSK3) and CDC-like kinases (CLKs)—indicating that CHK1 emerged early during eukaryotic evolution as a central regulator of genome integrity (hunter2015theeukaryoticprotein pages 3-6, mcneely2014chekagainrevisiting pages 1-2). Phylogenetic analyses that integrate sequence and limited structural data have revealed that, despite its conserved catalytic core, CHK1 diverges from typical CAMK family members and is often placed in a distinct clade, reflecting both its ancient origin and its unique regulatory adaptation to DNA repair and replication checkpoint functions (scheeff2005structuralevolutionof pages 17-17, caparelli2013regulatorymotifsin pages 2-3). This evolutionary divergence is further highlighted by the distinct regulatory modules present in its C-terminal region, which are maintained across diverse species and functionally underpin its ability to respond to replication stress and DNA damage (caparelli2013regulatorymotifsin pages 1-2). Collectively, these observations place CHK1 among highly conserved checkpoint kinases that form an evolutionary core set of proteins crucial for cell cycle regulation and stress adaptation in all eukaryotes (mcneely2014chekagainrevisiting pages 2-3).
2. Reaction Catalyzed  
   CHK1 catalyzes the phosphorylation reaction in which the terminal γ-phosphate from ATP is transferred to the hydroxyl group of serine or threonine residues in substrate proteins, thereby generating ADP, a phosphorylated substrate, and a proton (fabbro2015tenthingsyou pages 1-2). This fundamental reaction underpins the enzyme’s role in modifying key regulators involved in checkpoint signaling and DNA repair processes, and the chemical reaction can be formally represented as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (foote2015druggingatrprogress pages 17-18).
3. Cofactor Requirements  
   The catalytic activity of CHK1 is dependent on the presence of divalent cations, with Mg²⁺ being the primary cofactor required to stabilize the ATP molecule in the active site and facilitate the phosphoryl transfer reaction (fabbro2015tenthingsyou pages 1-2, foote2015druggingatrprogress pages 17-18).
4. Substrate Specificity  
   CHK1 exhibits substrate specificity that is largely determined by the presence of a consensus motif in its target proteins. This enzyme preferentially phosphorylates substrates that contain the motif [R-X-X-S/T], where an arginine residue situated three amino acids upstream of the target serine or threonine is critical for efficient recognition and phosphorylation (caparelli2013regulatorymotifsin pages 1-2, johnson2023anatlasof pages 7-7). The presence of this consensus sequence enables CHK1 to select among a variety of substrates that are pivotal in enforcing cell cycle checkpoints and initiating DNA repair mechanisms (johnson2023anatlasof pages 7-7).
5. Structure  
   The three-dimensional structure of CHK1 is characterized by a well-conserved N-terminal kinase domain that adopts a bilobal architecture typical of serine/threonine kinases. The smaller N-terminal lobe, predominantly composed of β-sheets, forms an ATP-binding pocket, while the larger C-terminal lobe, mainly consisting of α-helices, contains the catalytic residues and supports substrate binding (caparelli2013regulatorymotifsin pages 1-2). In addition to the kinase domain, CHK1 possesses a less structured C-terminal regulatory region that includes a kinase-associated 1 (KA1) domain and a unique C-terminal extension (CTE); the KA1 domain, which adopts a βαββββα fold, has been implicated in autoinhibition and in mediating interactions with checkpoint mediator proteins such as Crb2/53BP1, while the adjacent CTE is essential for maintaining proper protein conformation and activation (caparelli2013regulatorymotifsin pages 2-3, caparelli2013regulatorymotifsin pages 5-6). Crystal structures of the catalytic domain have been resolved at high resolution (approximately 1.7 Å), providing detailed insights into key catalytic features such as the activation loop, the hydrophobic spine, and the positioning of the conserved C-helix, all of which are critical for ATP binding and phosphotransfer activity (caparelli2013regulatorymotifsin pages 6-7, attwood2021trendsinkinase pages 13-14). The modular organization of CHK1, with its discrete catalytic and regulatory domains, enables tight intramolecular regulation and allows it to integrate signals from upstream kinases and damage sensors effectively (caparelli2013regulatorymotifsin pages 3-5).
6. Regulation  
   CHK1 activity is primarily regulated by phosphorylation events that serve to modulate its conformation and catalytic function. In response to DNA damage or replication stress, the ATR kinase phosphorylates CHK1 at serine 345 located within its C-terminal regulatory domain; this critical modification relieves the autoinhibitory influence imposed by the KA1 domain and permits CHK1 to adopt an active conformation (caparelli2013regulatorymotifsin pages 3-5, mcneely2014chekagainrevisiting pages 3-4). Additionally, CHK1 activation is further modulated by interactions with mediator proteins such as Claspin and Crb2/53BP1, which facilitate its recruitment to sites of DNA damage and enhance its phosphorylation efficiency, thereby ensuring a robust checkpoint response (caparelli2013regulatorymotifsin pages 5-6, mcneely2014chekagainrevisiting pages 7-7). Dephosphorylation events mediated by specific protein phosphatases, including PP1, contribute to checkpoint recovery by returning CHK1 to its inactive state once the DNA damage has been resolved (mcneely2014chekagainrevisiting pages 7-8). This dynamic regulation, involving both activating and inactivating post-translational modifications, enables CHK1 to respond rapidly to genotoxic stress while preventing unwarranted cell cycle delays during normal cell proliferation (mcneely2014chekagainrevisiting pages 8-9).
7. Function  
   CHK1 plays a central role in maintaining genomic integrity by orchestrating checkpoint-mediated cell cycle arrest and activating DNA repair processes in the presence of DNA damage or incomplete DNA replication. Upon activation, CHK1 phosphorylates key substrates, including the CDC25 family of phosphatases and the Wee1 kinase, thereby inhibiting the activation of cyclin-dependent kinases and delaying progression through the S and G2/M phases of the cell cycle (caparelli2013regulatorymotifsin pages 1-2, mcneely2014chekagainrevisiting pages 2-3). By enforcing a temporal delay in cell cycle progression, CHK1 provides cells with the necessary window to repair damaged DNA, stabilize stalled replication forks, and prevent the onset of mitotic catastrophe (foote2015druggingatrprogress pages 17-18, nyberg2002towardmaintainingthe pages 3-5). Additionally, CHK1 exerts its function not only under conditions of induced genotoxic stress but also during unperturbed cell cycles, where it contributes to fine-tuning cell cycle progression and ensuring proper DNA replication (caparelli2013regulatorymotifsin pages 1-2, mcneely2014chekagainrevisiting pages 7-8). The concerted action of CHK1 with other checkpoint proteins, such as ATR and CHK2, establishes a highly adaptive signaling network that protects cells from the accumulation of mutations and genomic instability (johnson2023anatlasof pages 7-7). In tumor cells, which often exhibit heightened replication stress and compromised p53 signaling, CHK1 becomes critically important for cell survival, thereby rendering it a valuable target for anticancer therapies (mcneely2014chekagainrevisiting pages 9-10).
8. Other Comments  
   CHK1 has emerged as a prominent target in cancer therapy due to its essential role in regulating the DNA damage response and maintaining replication fork stability. Several small-molecule inhibitors directed against CHK1, including SCH900776, AZD7762, PF-00477736, and LY2606368, have been developed and evaluated in preclinical and clinical settings, particularly in combination with DNA-damaging agents, to exploit the synthetic lethality of tumor cells that rely heavily on CHK1 signaling (mcneely2014chekagainrevisiting pages 5-7, mcneely2014chekagainrevisiting pages 9-10). Inhibition of CHK1 disrupts the proper checkpoint control, leading to replication catastrophe and apoptotic cell death in cancer cells, especially in those with defective p53 function (foote2015druggingatrprogress pages 1-2, mcneely2014chekagainrevisiting pages 7-8). Moreover, genetic mutations and dysregulation of CHK1 have been implicated in various cancer types, consistent with its vital role in preserving genomic stability (caparelli2013regulatorymotifsin pages 1-2, nyberg2002towardmaintainingthe pages 27-29). Ongoing research continues to refine the specificity and efficacy of CHK1 inhibitors, and studies examining the combinatorial use of CHK1 inhibitors with other agents, such as WEE1 kinase inhibitors, underscore the therapeutic potential of targeting the DNA damage checkpoint machinery in oncology (mcneely2014chekagainrevisiting pages 7-8, knippschild2014theck1family pages 1-2).
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