1. Phylogeny  
   Serine/threonine‐protein kinase Chk2 is highly conserved across eukaryotes and is the mammalian homolog of the yeast checkpoint kinases Rad53 and Cds1. Its evolutionary conservation indicates that it emerged early in eukaryotic evolution and has been maintained as a critical component of the DNA damage response (DDR) network. Within the human kinome, Chk2 belongs to the CMGC group of kinases, which comprises cyclin‐dependent kinases (CDKs), mitogen-activated protein kinases (MAPKs), glycogen synthase kinases (GSKs), and CDK-like kinases. Phylogenetic studies show that orthologs of CHEK2 are present in all mammals, and more distantly related organisms such as yeast contain functionally analogous proteins (Rad53/Cds1), underscoring an evolutionary relationship that reflects conserved checkpoint mechanisms from yeast to man (buscemi2014chk2kinasein pages 3-4, nevanlinna2006thechek2gene pages 5-6).
2. Reaction Catalyzed  
   Chk2 catalyzes the transfer of the γ-phosphate from ATP to a serine or threonine residue in a protein substrate. In chemical terms, the reaction can be represented as:

  ATP + [protein]-OH → ADP + [protein]-O‑phosphate + H⁺

This reaction is central to its role as a signaling kinase and is critical for the phosphorylation of numerous substrates involved in cell cycle checkpoint arrest, DNA repair, and apoptosis (li2005regulationofchk2 pages 1-2).

1. Cofactor Requirements  
   The catalytic activity of Chk2, like most serine/threonine kinases, depends on the presence of divalent cations as cofactors. In particular, Mg²⁺ is required to coordinate ATP binding and facilitate the transfer of the phosphate group during kinase activity (seo2003determinationofsubstrate pages 1-2, li2005regulationofchk2 pages 1-2).
2. Substrate Specificity  
   Chk2 exhibits a distinct substrate specificity characterized by a consensus sequence that directs phosphorylation on serine/threonine residues. The protein preferentially phosphorylates substrates that contain the motif L-X-R-X-X-S/T, where “L” is leucine in the –5 position and “R” is arginine in the –3 position relative to the phosphoacceptor serine/threonine residue. This substrate preference is supported by studies showing that Chk2 phosphorylates key regulatory proteins involved in cell cycle progression and DNA repair functions, including CDC25 phosphatases, p53, BRCA1/2, and others (bell2007geneticandfunctional pages 6-7, buscemi2014chk2kinasein pages 15-16, kim2007identificationofnovel pages 1-2). Recent substrate specificity atlases using oriented peptide libraries further define the amino acid requirements surrounding the phosphorylated residue, underlining the importance of hydrophobic and basic residues in the positions upstream of the phosphoacceptor (buscemi2014chk2kinasein pages 13-14, seo2003determinationofsubstrate pages 1-2).
3. Structure  
   Chk2 is a 543–amino acid protein that is organized into three distinct functional domains. The N-terminal region comprises an SQ/TQ cluster domain (SCD) that contains multiple serine/threonine-glutamine motifs, which are targets for phosphorylation by upstream kinases such as ATM. Immediately following the SCD is the forkhead-associated (FHA) domain, a highly conserved phosphopeptide-binding module that is critical for mediating protein-protein interactions and for facilitating the dimerization of Chk2 upon activation. The C-terminal portion harbors the kinase domain, which is responsible for the catalytic activity of the enzyme. Within the kinase domain, key structural features include the activation loop—harboring threonine residues such as T383 and T387—and a conserved catalytic core that supports ATP binding and phosphate transfer. Crystallographic studies and structural models (including those derived from AlphaFold) highlight the overall bilobal architecture typical of protein kinases, with the smaller N-terminal lobe primarily binding ATP and the larger C-terminal lobe serving as the substrate recognition surface. Unique features include the intricate arrangement of the FHA domain that facilitates efficient dimerization as well as autophosphorylation events necessary for full kinase activity (buscemi2014chk2kinasein pages 3-4, stolarova2020chek2germlinevariants pages 7-9, wu2006characterizationofchek2 pages 4-6).
4. Regulation  
   The regulation of Chk2 is multifaceted and is predominantly controlled by phosphorylation events. In response to DNA double-strand breaks, ATM kinase phosphorylates Chk2 at threonine 68 (T68), an event that promotes homodimerization through interactions mediated by the FHA and SCD domains. This initial phosphorylation event is essential for subsequent autophosphorylations at residues such as T383, T387 in the activation loop and at serine 516, all of which are required for full activation of the kinase. In addition to phosphorylation, Chk2 regulation can involve ubiquitination—the process through which its stability is modulated—and dephosphorylation by specific phosphatases such as PP2A and WIP1, which act to return Chk2 to its basal inactive state once DNA repair is accomplished. Moreover, post-translational modifications may influence subcellular localization, as interactions with proteins such as karyopherin-alpha have been implicated in the nuclear import of Chk2. Such regulation ensures that Chk2 functions in a precise and timely manner to control cell cycle arrest, DNA repair, and apoptosis (li2005regulationofchk2 pages 1-2, wu2006characterizationofchek2 pages 3-4, stolarova2020chek2germlinevariants pages 30-31).
5. Function  
   Chk2 plays a central role in maintaining genomic integrity by acting as a key mediator of the DNA damage response. Upon activation by DNA double-strand breaks, Chk2 phosphorylates a variety of substrates to enforce cell cycle arrest at both the G1/S and G2/M checkpoints. For example, phosphorylation of the CDC25 family of phosphatases (including CDC25A, CDC25B, and CDC25C) leads to their inhibition, resulting in the accumulation of inhibitory phosphates on CDK-cyclin complexes and subsequently halting cell cycle progression. In addition, Chk2 phosphorylates the tumor suppressor p53—specifically at serine 20—which alleviates the inhibitory effect of MDM2 and facilitates the stabilization and activation of p53. Activated p53 then drives the transcription of target genes involved in DNA repair, cell cycle arrest, and apoptosis. Furthermore, Chk2 regulates the DNA repair machinery by phosphorylating proteins such as BRCA2, thereby enhancing the recruitment of RAD51 to sites of damage and promoting homologous recombination repair. Beyond its canonical DDR functions, Chk2 also plays roles in mitotic spindle assembly through phosphorylation of proteins like BRCA1 and has been implicated in the regulation of autophagy under conditions of oxidative stress via phosphorylation of E3 ubiquitin ligases such as TRIM32. Its activity has been linked to the transcriptional regulation of genes involved in DNA repair by modulating transcription factors including FOXM1. Collectively, Chk2 functions as a tumor suppressor by coordinating cell cycle checkpoint activation, DNA repair, and apoptosis in response to genomic stress (bell2007geneticandfunctional pages 6-7, buscemi2014chk2kinasein pages 7-8, smith2020dnadamagecheckpoint pages 1-2).
6. Other Comments  
   Several inhibitors targeting Chk2 have been developed, and these are of particular interest in cancer therapy. While some small-molecule inhibitors such as PV1019, VRX0466617, and bis-guanylhydrazone derivatives have been evaluated in preclinical settings, many of these compounds also affect related kinases like Chk1, complicating efforts to achieve absolute selectivity (lountos2011structuralcharacterizationof pages 10-10, stolz2011tumorsuppressorchk2 pages 3-5). Mutations in CHEK2, notably the 1100delC truncation and the I157T missense variant, have been robustly associated with an increased risk of several cancers, including breast, prostate, and colorectal carcinomas, as well as in familial cancer predisposition syndromes (bell2007geneticandfunctional pages 6-7, stolarova2020chek2germlinevariants pages 3-5, wu2006characterizationofchek2 pages 4-6). Owing to its crucial role in safeguarding genomic integrity, alterations in Chk2 function may contribute to chromosomal instability observed in tumors. Moreover, its involvement in modulating the balance between cell survival and apoptosis through the p53 pathway further underscores its significance as a tumor suppressor. In addition to its canonical functions in DDR, emerging evidence suggests that Chk2 may have roles in DNA damage-independent processes such as mitotic spindle assembly and autophagosome regulation under oxidative stress conditions (buscemi2014chk2kinasein pages 16-16, buscemi2014chk2kinasein pages 8-10). These findings render Chk2 a key target for tailoring therapeutic strategies, particularly those aimed at sensitizing tumor cells to DNA-damaging agents (chrisanthar2008chek2mutationsaffecting pages 9-10, mustofa2020rolesofchk2chek2 pages 1-4).
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