1. Phylogeny  
   Receptor‐interacting serine/threonine‐protein kinase 2 (RIPK2) belongs to the RIP kinase family, a subgroup within the tyrosine‐kinase‐like (TKL) superfamily of protein kinases. RIPK2 is evolutionarily conserved across vertebrates, and its orthologs can be found in all mammalian species, indicating that its functions in immune regulation have been maintained during evolution (nikhar2021…ofpyridoa pages 1-3). Within the kinase complement, RIPK2 is phylogenetically distinct from related kinases such as RIPK1 and RIPK3, which display specialized roles in cell death pathways, while RIPK2 is uniquely adapted as a signaling adaptor in innate immunity (zare2022theroleof pages 32-35, topal2021ripk2nodsto pages 1-6). Moreover, this kinase is part of an evolutionarily ancient set of signaling molecules that emerged early during the evolution of eukaryotes; similar to many kinases described by Manning and colleagues, RIPK2 is a core component in cellular signal transduction that has diversified in higher organisms (nikhar2021…ofpyridoa pages 26-28).
2. Reaction Catalyzed  
   RIPK2 catalyzes a classical protein phosphorylation reaction using ATP as a phosphate donor. In this reaction, ATP and a protein substrate containing serine, threonine and, under certain conditions, tyrosine residues are converted to ADP and a phosphorylated protein with the release of a proton. This reaction can be summarized as:  
     ATP + [protein]–(L-serine/threonine/tyrosine) → ADP + [protein]–phosphate + H⁺ (misehe2024designsynthesisand pages 44-49, nikhar2021…ofpyridoa pages 1-3).
3. Cofactor Requirements  
   The catalytic activity of RIPK2, like most kinases, is dependent on the presence of divalent metal ions. In particular, magnesium ions (Mg²⁺) are essential for its enzymatic function as they coordinate ATP binding within the active site of the kinase domain (misehe2024designsynthesisand pages 44-49, shen2025currentadvanceson pages 1-2).
4. Substrate Specificity  
   RIPK2 has been characterized as a dual‐specificity protein kinase with a predominant activity toward serine/threonine phosphorylation and emerging evidence for tyrosine phosphorylation in appropriate contexts. Kinome-wide profiling studies have provided detailed insight into substrate motifs for serine/threonine kinases, and RIPK2 appears to display substrate preferences similar to those reported in Johnson et al. (2023) for the serine/threonine kinome. Additionally, recent data support that RIPK2 can phosphorylate tyrosine residues on specific substrates, as highlighted by Yaron-Barir et al. (2024), which further reinforces the dual-specificity nature of this enzyme (nikhar2021…ofpyrido pages 26-28, pham2023recentadvancesin pages 17-18, you2023ripk2apromising pages 13-14). The consensus motif for its serine/threonine activity has not been fully delineated; however, the intrinsic substrate specificity studies indicate that the kinase core engages substrates through a typical linear motif that is optimally phosphorylated when presented in an appropriate structural context (pham2023recentadvancesin pages 1-3, zare2022theroleof pages 35-39).
5. Structure  
   RIPK2 is composed of an N-terminal kinase domain and a C-terminal caspase activation and recruitment domain (CARD). The kinase domain is responsible for its catalytic activity and displays the conserved bilobal structure characteristic of protein kinases with a smaller N-lobe dominated by a glycine-rich loop and a larger C-lobe that contains the activation segment, including key motifs such as the DFG motif, the HRD motif in the catalytic loop, and a conserved catalytic lysine that coordinates ATP binding (misehe2024designsynthesisand pages 44-49, pellegrini2017structuresofthe pages 1-2). Crystal structures and AlphaFold models of RIPK2 reveal that its kinase domain not only adopts a canonical fold but also is capable of dimerization – a feature critical for its autophosphorylation and subsequent activation. Dimerization occurs in a side-by-side arrangement where the N-lobe of one monomer interfaces with the C-lobe of its partner, stabilizing the activation loop and facilitating trans-autophosphorylation events; these structural arrangements are well supported by data from crystallographic studies and are consistent with the general principles of kinase activation (nikhar2021…ofpyridoa pages 1-3, lethier2023structureshowsthat pages 1-2). In contrast, the CARD domain at the C-terminus mediates homotypic interactions with other CARD-containing proteins such as NOD1 and NOD2. This domain is essential for the assembly of higher order signaling complexes or “RIPosomes” that function as scaffolds for downstream signal propagation (shen2025currentadvanceson pages 2-3, zare2022theroleofa pages 32-35). Unique structural features include the disposition of the activation loop, which includes key autophosphorylation sites such as serine 176 and tyrosine 474, residues that have been implicated in conformational shifts necessary for full enzymatic activity and optimal scaffolding function (nikhar2021…ofpyridoa pages 26-28, zare2022theroleof pages 35-39). Additionally, structural studies have identified a hydrophobic pocket near residue K209 in the kinase domain, which has been shown to serve as a regulatory interface for interacting with the BIR2 domain of the E3 ubiquitin ligase XIAP (heim2020aregulatoryinterface pages 7-9, nachbur2015aripk2inhibitor pages 1-2).
6. Regulation  
   The activity and function of RIPK2 are tightly controlled by multiple post-translational modifications (PTMs) that affect its catalytic activity, protein–protein interactions, stability, and subcellular localization. Autophosphorylation is a key initial regulatory event; RIPK2 autophosphorylates within its activation loop – notably at serine 176 – which is critical for its full activation, and phosphorylation at tyrosine 474 is essential for maximal NOD2 signaling via NF-κB activation (pellegrini2017structuresofthe pages 1-2, zare2022theroleof pages 35-39). In addition to autophosphorylation, RIPK2 is subject to extensive Lys63-linked polyubiquitination, mediated primarily by E3 ubiquitin ligases such as XIAP, BIRC2, and BIRC3. Later, the linear (Met1-linked) polyubiquitination by the LUBAC complex further modulates downstream signaling by recruiting adaptor molecules including IKBKG/NEMO and facilitating the activation of TAK1, ultimately leading to NF-κB activation (misehe2024designsynthesisand pages 44-49, topal2021ripk2nodsto pages 11-15). Specific lysine residues, such as K209, play a regulatory role in this modification process. Although ubiquitination at K209 has been proposed, mutagenesis studies have revealed that alteration of K209 (e.g. K209R) results in impaired ubiquitination and diminished NF-κB activation, likely due to disruption of a structural regulatory interface rather than solely a loss of a ubiquitin acceptor site (heim2020aregulatoryinterface pages 7-9, nachbur2015aripk2inhibitor pages 1-2). Moreover, residues in the hydrophobic pocket around K209 and I212 have been shown to affect the binding of XIAP, which is essential for propagating the ubiquitin signal; mutation of I212 to a polar residue (I212D) abolishes ubiquitination and downstream signaling, whereas a more conservative mutation such as I212A can enhance activity (heim2020aregulatoryinterface pages 7-9, zare2022theroleofa pages 35-39). Collectively, these PTMs operate in a coordinated manner: phosphorylation activates and structurally primes the kinase while ubiquitination, primarily of the Lys63-linked type, converts RIPK2 into a robust scaffolding platform that recruits and organizes downstream signaling effectors (shen2025currentadvanceson pages 12-13, ellwanger2019xiapcontrolsripk2 pages 13-14).
7. Function  
   RIPK2 plays a central role in connecting innate immune detection with the activation of inflammatory signaling pathways. Functionally, it is recruited by the cytoplasmic pattern recognition receptors NOD1 and NOD2 upon binding bacterial peptidoglycan components, such as muramyl dipeptide. This recruitment occurs via homotypic CARD-CARD interactions, which lead to the assembly of higher order signaling complexes known as RIPosomes. Once activated, RIPK2 autophosphorylates and undergoes specific ubiquitination events, thereby serving as a platform for recruiting the TAK1 kinase complex, IKK complex, and other downstream signaling molecules necessary for NF-κB and MAPK pathway activation (nikhar2021…ofpyridoa pages 1-3, topal2021ripk2nodsto pages 1-6, shen2025currentadvanceson pages 12-13). Additionally, RIPK2 contributes to adaptive immunity by participating in T-cell receptor (TCR) signaling and by promoting the phosphorylation of key adaptor proteins such as BCL10, further linking innate and adaptive immune responses (nikhar2021…ofpyridoa pages 1-3, shen2025currentadvanceson pages 13-13). Expression of RIPK2 is broad, with significant levels found in immune cells, including macrophages, dendritic cells, and lymphocytes, as well as in epithelial cells lining mucosal surfaces, where it plays a critical role in the host defense against pathogens (you2023ripk2apromising pages 1-2, topal2021ripk2nodsto pages 6-11). Moreover, RIPK2 signaling is implicated in the regulation of autophagy, a process that further modulates immune responses and maintains cellular homeostasis. This multifaceted role in immune signaling has also linked RIPK2 to various pathological conditions, including chronic inflammatory diseases (such as inflammatory bowel disease, rheumatoid arthritis, and psoriasis) and certain forms of cancer, where aberrant activation of NF-κB and inflammatory cytokine production contribute to tumor progression (zare2022theroleofb pages 62-65, you2023ripk2apromising pages 6-7).
8. Other Comments  
   RIPK2 has attracted considerable attention as a drug target due to its central role in the regulation of both innate and adaptive immune responses. Numerous small molecule inhibitors have been developed to interfere with its kinase and scaffolding functions; examples include ATP-competitive compounds such as WEHI-345, ponatinib, and more selective inhibitors like GSK583. Some of these inhibitors function by directly occupying the ATP-binding site, while others exert their effects by allosterically disrupting the interaction between RIPK2 and XIAP, thereby suppressing the polyubiquitination necessary for downstream NF-κB activation (hrdinka2018smallmoleculeinhibitors pages 1-2, you2023ripk2apromising pages 6-7). In addition, proteolysis-targeting chimera (PROTAC) strategies have been explored to induce selective degradation of RIPK2 as an alternative therapeutic strategy (you2023ripk2apromising pages 6-7). Disease associations for RIPK2 extend to several inflammatory and autoimmune disorders, including Crohn’s disease, Blau syndrome, and multiple sclerosis, and emerging studies also suggest a role in certain cancers, such as inflammatory breast cancer, where overexpression or amplification of RIPK2 correlates with poor prognosis (zare2022theroleofb pages 62-65, you2023ripk2apromising pages 1-2). Notable mutations and alterations in the regulatory domains, particularly those affecting key residues such as K209 and I212 in the kinase domain, have been shown to disrupt XIAP binding and impair normal immune signaling. These findings underscore the potential impact of genetic variations in RIPK2 on disease susceptibility and progression (heim2020aregulatoryinterface pages 7-9, nachbur2015aripk2inhibitor pages 1-2).
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