## 1. Phylogeny

RIPK1 is a conserved serine/threonine kinase that belongs to the receptor‐interacting protein (RIP) kinase family and is present in all mammalian species, with orthologs identified in vertebrates and lower eukaryotes (dara2018thereceptorinteracting pages 1-3). RIPK1 clusters phylogenetically with other members of the RIP kinases such as RIPK2, RIPK3, and RIPK4, reflecting an evolutionary relationship characterized by conserved kinase domains and distinct interaction modules (torre2021theroleof pages 17-18). It is part of the broader serine/threonine kinase group that functions as key regulators in programmed cell death, inflammation and stress response pathways, a feature that can be traced back to early eukaryotic ancestors (annibaldi2018checkpointsintnfinduced pages 3-6).

## 2. Reaction Catalyzed

RIPK1 catalyzes the transfer of a phosphate group from ATP to target serine or threonine residues on substrate proteins, thereby converting ATP into ADP and generating a phosphorylated protein product (xie2013structuralbasisof pages 1-2).

## 3. Cofactor Requirements

The kinase activity of RIPK1 depends on the presence of divalent metal ions, with magnesium (Mg²⁺) serving as a critical cofactor that facilitates ATP binding and proper enzymatic function (mifflin2020receptorinteractingproteinkinase pages 1-2).

## 4. Substrate Specificity

Although the complete consensus substrate motif for RIPK1 has not been fully delineated, its well‐characterized substrates include itself via autophosphorylation and the kinase RIPK3, which is phosphorylated to propagate necroptosis signaling; additionally, RIPK1 phosphorylates DAB2IP at Ser-728 in a TNF-α-dependent manner, triggering downstream MAP3K5–JNK apoptotic cascades (annibaldi2018checkpointsintnfinduced pages 6-7, chen2022advancesinripk1 pages 1-2). The substrate specificity reflects a preference for serine/threonine residues within targets that engage in death and inflammation signaling pathways (li2023targetingripk1kinase pages 2-4).

## 5. Structure

RIPK1 is a 671–amino acid protein that is organized into three main domains: an N-terminal kinase domain, a centrally located intermediate domain, and a C-terminal death domain (DD) (dara2018thereceptorinteracting pages 1-3, siregar2024theroleof pages 4-6). The N-terminal kinase domain contains characteristic motifs, including a catalytic loop with conserved residues such as the lysine (Lys45) that is essential for ATP binding and catalysis, and it adopts an inactive DLG-out conformation when bound by inhibitors such as necrostatins (chen2022advancesinripk1 pages 2-3, xie2013structuralbasisof pages 1-2). The intermediate domain harbors a RIP homotypic interaction motif (RHIM) that enables homo- or hetero-oligomerization with RIPK3 and other RHIM-containing proteins, and it also contains critical ubiquitination sites such as Lys377 that regulate its signaling (annibaldi2018checkpointsintnfinduced pages 2-3, dara2018thereceptorinteracting pages 1-3). The C-terminal death domain mediates interactions with adaptor proteins like FADD, TRADD, and TNFR1, facilitating recruitment into various signaling complexes (dara2018thereceptorinteracting pages 1-3, torre2021theroleof pages 3-5). Overall, the modular structure of RIPK1 supports its dual functions as an enzyme and as a scaffold in assembling complex signaling platforms (chen2022advancesinripk1 pages 3-4).

## 6. Regulation

RIPK1 is intricately regulated by multiple post-translational modifications that control its switch between pro-survival and pro-death functions. Ubiquitination by E3 ligases such as cIAP1/2 and the LUBAC complex (including HOIP, HOIL-1L, and SHARPIN) attaches K63-linked and linear (M1-linked) ubiquitin chains to RIPK1 (annibaldi2018checkpointsintnfinduced pages 2-3, witt2017diverseubiquitinlinkages pages 5-6) and serves to tether RIPK1 within complex-I for NF-κB activation. Conversely, deubiquitinases like CYLD, A20, and Cezanne remove these ubiquitin chains, thereby facilitating RIPK1 dissociation from complex-I and promoting formation of death-inducing complex-II (annibaldi2018checkpointsintnfinduced pages 9-11, ju2022theresurrectionof pages 4-5). In addition to ubiquitination, RIPK1 is phosphorylated at several key serine residues such as Ser166, Ser161, and residues near S320/S335 (or S321/S336 in mice) by kinases including IKK1/2, MK2, TAK1, and TBK1; these phosphorylation events suppress its kinase activity and inhibit assembly of pro-death signaling complexes (annibaldi2018checkpointsintnfinduced pages 7-9, ju2022theresurrectionof pages 5-6). Furthermore, caspase-8–mediated cleavage of RIPK1 at Asp324 inactivates its kinase activity and prevents overactivation of necroptosis (dara2018thereceptorinteracting pages 3-4, mifflin2020receptorinteractingproteinkinase pages 3-4). The coordinated interplay between ubiquitination, phosphorylation, and proteolytic processing establishes an early cell death checkpoint that finely tunes the functional outcome of TNF receptor signaling (annibaldi2018checkpointsintnfinduced pages 7-9, ju2022theresurrectionof pages 4-5).

## 7. Function

RIPK1 is a central signaling hub that integrates signals from death receptors such as TNFR1 to determine cell fate by balancing pro-survival and programmed cell death pathways. Through its kinase-independent scaffold function, RIPK1 facilitates NF-κB activation and the transcriptional upregulation of anti-apoptotic and inflammatory genes such as cFLIP, Bcl2, and IL-6, thereby promoting cell survival in response to TNF stimulation (annibaldi2018checkpointsintnfinduced pages 2-3, dara2018thereceptorinteracting pages 1-3). Conversely, when its ubiquitination is removed and its kinase activity becomes engaged, RIPK1 promotes assembly of complex-IIa (comprising FADD, caspase-8, and cFLIP) to induce apoptosis, or complex-IIb (the necrosome formed with RIPK3 and MLKL) to trigger necroptosis (annibaldi2018checkpointsintnfinduced pages 6-7, feoktistova2015programmednecrosisand pages 1-4). RIPK1 also phosphorylates RIPK3 in a reciprocal autophosphorylation process that is essential for necrosome formation (annibaldi2018checkpointsintnfinduced pages 7-9, li2023targetingripk1kinase pages 1-2). In addition, by phosphorylating DAB2IP at Ser-728, RIPK1 participates in activating the MAP3K5-JNK apoptotic cascade, linking it to additional cell death and stress response pathways (Information section). These diverse functions illustrate how RIPK1 modulates both inflammatory signaling and cell death decisions during embryogenesis, tissue homeostasis, and in response to pathological stimuli such as infection and DNA damage (annibaldi2018checkpointsintnfinduced pages 7-9, mifflin2020receptorinteractingproteinkinase pages 4-5).

## 8. Other Comments

RIPK1 is the focus of extensive pharmacological research owing to its dual role in regulating cell death and inflammatory responses; potent inhibitors such as Necrostatin-1 (Nec-1) and GSK2982772 have been developed and are under clinical investigation for inflammatory diseases including psoriasis, rheumatoid arthritis, and inflammatory bowel disease (annibaldi2018checkpointsintnfinduced pages 7-9, martens2020inhibitorstargetingripk1ripk3 pages 10-12). Mutations or dysregulation of RIPK1 activity have been implicated in various pathological conditions, ranging from cancer and autoimmune disorders to neurodegeneration and ischemia-reperfusion injury, highlighting its potential as a therapeutic target (annibaldi2018checkpointsintnfinduced pages 9-11, ju2022theresurrectionof pages 1-2). Notable among its regulatory mechanisms, the balance between ubiquitination and phosphorylation acts as a critical checkpoint that determines whether RIPK1 promotes NF-κB–mediated survival or triggers cell death via apoptosis or necroptosis, thereby influencing cellular sensitivity to TNF and other inflammatory stimuli (ju2022theresurrectionof pages 4-5, li2023targetingripk1kinase pages 2-4).

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