1. Phylogeny  
   RIPK3 is a member of the receptor‐interacting protein kinase (RIPK) family, which groups within the serine/threonine kinases and is evolutionarily related to other RIPK family members such as RIPK1, RIPK2, and RIPK4. The phylogenetic analyses reveal that RIPK3 and its close relatives share a conserved kinase domain and a distinctive RIP homotypic interaction motif (RHIM) that is essential for protein–protein interactions integral to necroptosis signaling (lv2022comparativeandevolutionary pages 1-3, moriwaki2013rip3amolecular pages 1-3). Within the kinome classification, RIPK3 is placed in the tyrosine kinase-like (TKL) group even though its enzymatic activity is exclusively serine/threonine kinase in nature (cuny2021ripkproteinkinase pages 6-8). Orthologs of RIPK3 have been identified in diverse vertebrate species, although certain lineages such as birds and some mammals exhibit loss of the RIPK3 gene, which suggests that adaptive evolutionary pressures – potentially related to metabolic regulation and resistance to ischemia–reperfusion injury – have stimulated divergence in RIP kinase signaling pathways (lv2022comparativeandevolutionary pages 18-20, dondelinger2016anevolutionaryperspective pages 5-7). This conservation among species highlights the critical role of RIPK3 in innate immune responses and programmed cell death pathways.
2. Reaction Catalyzed  
   RIPK3 catalyzes the transfer of the γ-phosphate from ATP to specific serine and threonine residues on substrate proteins, thereby phosphorylating them. The canonical reaction can be summarized as: ATP + [protein]–(Ser/Thr) → ADP + [protein]–(pSer/pThr) + H⁺ (murphy2015posttranslationalcontrolof pages 3-4). The primary physiological substrate of RIPK3 is the pseudokinase MLKL, whose phosphorylation by RIPK3 triggers its oligomerization and translocation to the plasma membrane, ultimately executing necroptosis by disrupting membrane integrity (moriwaki2013rip3amolecular pages 3-5, newton2015ripk1andripk3 pages 1-2). RIPK3 also engages in reciprocal auto- and trans-phosphorylation events with RIPK1, a process that is integral to the assembly and activation of the necrosome complex (cuny2021ripkproteinkinase pages 1-2, cook2014ripk1andripk3induced pages 12-13).
3. Cofactor Requirements  
   The catalytic activity of RIPK3, like most kinases, is dependent on the presence of divalent metal ions that act as cofactors; most notably, Mg²⁺ is required for proper ATP coordination and for facilitating phosphate transfer during the phosphorylation reaction (moriwaki2017theinflammatorysignal pages 7-10, newton2015ripk1andripk3 pages 1-2). Although explicit mention of Mn²⁺ or other metal ions is less common in the context of RIPK3 regulation, the structural and mechanistic parallels with other serine/threonine kinases support the necessity for divalent cations in its enzymatic function (gardner2023from(tool)benchto pages 1-2). Additionally, RIPK3’s kinase domain exhibits typical catalytic motifs that coordinate ATP binding and catalysis, suggesting that its regulation is intricately dependent on the availability and binding of ATP in a metal‐ion assisted manner (moriwaki2013rip3amolecular pages 3-5).
4. Substrate Specificity  
   RIPK3 displays substrate specificity primarily for proteins involved in programmed necrosis. Its most well-characterized substrate is MLKL; phosphorylation of MLKL by RIPK3 at critical serine/threonine residues (e.g., human S227 and murine T231/S232) is necessary for MLKL activation and subsequent necroptotic execution (zhou2024ripk3signalingand pages 1-2, moriwaki2013rip3amolecular pages 3-5). In addition, RIPK3 engages in reciprocal phosphorylation with RIPK1, suggesting that it may recognize specific structural features or motifs that facilitate interaction via the RHIM domain (newton2015ripk1andripk3 pages 1-2, cuny2021ripkproteinkinase pages 2-3). Although no clear consensus phosphorylation motif akin to the RxRxxp[ST] motif found in other serine/threonine kinases has been fully defined for RIPK3, its substrate recognition appears to be strongly influenced by protein–protein interactions mediated by its regulatory RHIM domain and by complex formation within the necrosome (raju2018kinasedomaindimerization pages 1-2, shlomovitz2017mechanismsofripk3‐induced pages 1-6).
5. Structure  
   RIPK3 comprises an N-terminal serine/threonine kinase domain that harbors all the canonical motifs, including the ATP-binding P loop, the catalytic lysine (e.g., Lys50 in human RIPK3), and the conserved DFG, HRD motifs essential for catalysis (moriwaki2013rip3amolecular pages 3-5, cuny2021ripkproteinkinase pages 6-8). C-terminal to the kinase domain, RIPK3 contains a RIP homotypic interaction motif (RHIM) that is crucial for mediating interactions with other RHIM-containing proteins such as RIPK1, TRIF, and ZBP1; these interactions are vital for necrosome assembly and signaling (moriwaki2017theinflammatorysignal pages 22-23, shlomovitz2017mechanismsofripk3‐induced pages 6-9). Structural studies, including crystallography and AlphaFold models, indicate that the kinase domain adopts a typical bilobal structure with an N-lobe rich in β-sheets and a C-lobe predominantly composed of α-helices, forming the catalytic cleft in which ATP binds (cuny2021ripkproteinkinase pages 6-8). Unique structural features of RIPK3 include its small, hydrophilic gatekeeper residue that influences inhibitor access to its kinase domain and allows for some atypical conformational dynamics during activation (cuny2021ripkproteinkinase pages 6-8). The RHIM region itself, although predicted to be largely unstructured in isolation, forms amyloid-like fibrils upon interaction with partner proteins, providing a scaffold for kinase activation (shlomovitz2017mechanismsofripk3‐induced pages 9-13).
6. Regulation  
   RIPK3 activity is subject to multiple layers of regulation that include both its catalytic activity and its scaffolding function. One key regulatory mechanism is auto- and trans-phosphorylation, wherein RIPK3 phosphorylates itself as well as RIPK1 within the necrosome complex; these phosphorylation events are required for the proper propagation of necroptotic signaling (cuny2021ripkproteinkinase pages 1-2, newton2015ripk1andripk3 pages 1-2). In addition, several post-translational modifications control RIPK3 function. Phosphorylation at specific residues, such as those within its activation loop (e.g., S227 in human RIPK3), is critical for its kinase activity and for the recruitment of downstream effectors such as MLKL (zhou2024ripk3signalingand pages 1-2, moriwaki2013rip3amolecular pages 3-5). Ubiquitination also plays a role in modulating RIPK3 stability and activity; ubiquitin ligases can target RIPK3 for proteasomal or lysosomal degradation, thereby acting as a negative regulatory mechanism (cuny2021ripkproteinkinase pages 6-8, murphy2015posttranslationalcontrolof pages 3-4). Furthermore, proteolytic cleavage by caspase-8 can inactivate RIPK3 and divert cell death away from necroptosis toward apoptosis, illustrating a kinase-independent function in cell death regulation (moriwaki2017theinflammatorysignal pages 10-13, shlomovitz2017mechanismsofripk3‐induced pages 6-9). During cell cycle progression, RIPK3 is phosphorylated at serine 369 by PLK1, a modification that stabilizes RIPK3 and promotes its incorporation into higher-order death-inducing complexes known as the ripoptosome, thereby linking cell cycle cues to cell death pathways (gupta2021plk1mediateds369phosphorylation pages 1-2, gupta2021plk1mediateds369phosphorylation pages 27-31). These regulatory events are critical for ensuring that RIPK3 activity is tightly controlled to prevent unwarranted cell death and inflammation.
7. Function  
   RIPK3 is best known for its central role in the execution of necroptosis, a form of programmed necrosis that is characterized by rapid plasma membrane rupture and the induction of inflammation due to the release of damage-associated molecular patterns (DAMPs) (moriwaki2013rip3amolecular pages 3-5, newton2015ripk1andripk3 pages 1-2). In the canonical necroptotic pathway, RIPK3, upon activation by death receptor ligands such as TNF-α or by pattern recognition receptors via ZBP1, phosphorylates MLKL; activated MLKL oligomerizes and translocates to the plasma membrane to disturb membrane integrity, resulting in cell lysis and the subsequent inflammatory response (cuny2021ripkproteinkinase pages 6-8, zhou2024ripk3signalingand pages 1-2). Beyond necroptosis, RIPK3 has been implicated in apoptosis under certain conditions; for example, kinase-dead mutants of RIPK3 can trigger an alternative form of cell death in a RIPK1-, FADD-, and caspase-8-dependent manner, highlighting its role as a bifunctional mediator of cell death (moriwaki2017theinflammatorysignal pages 1-4, shlomovitz2017mechanismsofripk3‐induced pages 1-6). RIPK3 also contributes to antiviral responses independent of its cell death functions; in neuronal cells infected with Zika virus, RIPK3 helps establish a metabolic program that upregulates ACOD1/IRG1 and leads to the production of itaconate, a metabolite that inhibits succinate dehydrogenase and restricts viral replication (Information section, by similarity). In addition, RIPK3 modulates cellular metabolism through interactions with metabolic enzymes such as GLUL, GLUD1, and PYGL, thereby potentially influencing the tricarboxylic acid cycle, oxidative phosphorylation, and reactive oxygen species (ROS) production (Information section, PubMed:19498109). Tissue-specific expression patterns indicate that RIPK3 is widely expressed in immune cells, where it regulates inflammatory cytokine production and inflammasome activation; in some contexts, RIPK3-dependent signaling contributes to the production of interferons and other cytokines that are crucial for innate immune responses (yang2023dichotomousrolesof pages 1-3, shlomovitz2017mechanismsofripk3‐induced pages 21-23). Collectively, the functions of RIPK3 encompass both cell death pathways – necroptosis and apoptosis – and cell survival/adaptive responses such as metabolic reprogramming and inflammatory signaling, positioning it as a key integrator of stress and immune signals.
8. Other Comments  
   Multiple experimental inhibitors targeting the necroptotic pathway focus indirectly on RIPK3 by inhibiting upstream kinases such as RIPK1; however, several small molecule inhibitors that directly modulate RIPK3 activity are under investigation (martens2020inhibitorstargetingripk1ripk3 pages 2-4, martens2020inhibitorstargetingripk1ripk3 pages 4-6). Dysregulation of RIPK3 activity has been associated with a variety of pathological states including inflammatory diseases, ischemia–reperfusion injury, neurodegenerative conditions, and even certain cancers, given its role in both cell death and non-cell death signaling pathways (cuny2021ripkproteinkinase pages 6-8, morgan2022rolesofripk3 pages 1-2). Notable mutations that affect the kinase domain, such as kinase-dead mutants (e.g., D161N), have revealed that catalytic inactivity can sometimes drive alternative, gain-of-function apoptotic signaling, demonstrating a complex interplay between different modes of programmed cell death (moriwaki2017theinflammatorysignal pages 10-13, shlomovitz2017mechanismsofripk3‐induced pages 6-9). Current areas of active research include elucidating the detailed structural dynamics of RIPK3 during necrosome formation, the impact of various post-translational modifications on its function, and the development of therapeutically effective inhibitors that can selectively block its necroptotic function without triggering compensatory cell death pathways (gupta2021plk1mediateds369phosphorylation pages 27-31, urwylerrosselet2023functionsofthe pages 12-13). Additionally, RIPK3 has been recognized for its role in executing cell death in distinct subcellular compartments such as the nucleus, particularly in response to viral infections that induce Z-RNA formation, thereby expanding its functional repertoire beyond the cytoplasmic necrosome (Information section, by similarity; zhou2024ripk3signalingand pages 3-5). The complex regulation of RIPK3, involving autophosphorylation, interactions with RIPK1 and other adaptors via the RHIM domain, and modulation by ubiquitination and proteolysis, continues to be an intensive focus given its therapeutic potential for modulating inflammatory and neurodegenerative diseases (cuny2021ripkproteinkinase pages 2-3, murphy2015posttranslationalcontrolof pages 3-4).
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