1. Phylogeny  
   RIPK3 is a member of the receptor‐interacting protein kinase family within the serine/threonine kinase branch of the human kinome. Orthologs of RIPK3 are identified across vertebrate species, although its presence is not universal; for example, lineage‐specific losses have been reported in birds and in some early diverging mammals. Phylogenetic analyses place RIPK3 in a sub‐cluster with RIPK1, RIPK2, RIPK4 and RIPK5, whereas more distantly related members such as RIPK6 and RIPK7 form a separate clade, highlighting both its evolutionary conservation in mammals and the divergent paths in other organisms (dondelinger2016anevolutionaryperspective pages 2-4, lv2022comparativeandevolutionary pages 1-3). Its evolution appears to be driven by selective pressures from host–pathogen interactions, with positive selection detected in some mammalian clades, a finding that underlines the functional importance of RIPK3 in innate immune responses (fay2025evolutionaryandfunctional pages 1-4, brault2017controlleddetonationevolution pages 13-17).
2. Reaction Catalyzed  
   RIPK3 catalyzes the transfer of a phosphate group from ATP to specific serine/threonine residues on protein substrates. The canonical reaction can be described as: ATP + [protein]‐(L‐serine or L‐threonine) → ADP + [protein]‐(L‐serine/threonine)‐phosphate + H⁺ (cook2014ripk1andripk3induced pages 12-12). This phosphorylation event is central to RIPK3’s ability to propagate necroptotic signaling, particularly by modifying its downstream substrate, mixed lineage kinase domain‐like protein (MLKL) (shi2018targetingreceptorinteractingserinethreonineprotein pages 68-75).
3. Cofactor Requirements  
   The kinase activity of RIPK3 is dependent on the presence of ATP, which serves as the phosphate donor, and it requires Mg²⁺ as an essential cofactor. This requirement is consistent with its classification among serine/threonine kinases, where Mg²⁺ ions facilitate proper coordination of the ATP molecule within the active site (chiou2025thekinasedomain pages 4-7, martens2020inhibitorstargetingripk1ripk3 pages 2-4).
4. Substrate Specificity  
   RIPK3 exhibits substrate specificity that is critical for its role in necroptosis. Its principal substrate is MLKL, whose phosphorylation by RIPK3 at specific residues (for example, human Ser227; mouse T231/S232) enables MLKL to oligomerize, translocate to the plasma membrane, and induce membrane disruption. Although a detailed consensus motif for RIPK3 substrates is not universally established, the kinase displays a preference for phosphorylating serine/threonine residues within regions that facilitate conformational changes required for necroptotic complex assembly (shi2018targetingreceptorinteractingserinethreonineprotein pages 63-68, liu2021ripk3signalingand pages 4-6). Other substrates reported include proteins involved in metabolic regulation, such as GLUL, GLUD1, and PYGL, although these interactions extend the functional repertoire of RIPK3 beyond classical necroptosis (Information section).
5. Structure  
   RIPK3 comprises a conserved N-terminal kinase domain and a C-terminal region that contains a RIP homotypic interaction motif (RHIM). The kinase domain, essential for its catalytic activity, harbors key residues such as an ATP-positioning lysine (K51 in mouse), a catalytic aspartate in the HRD motif, and a Mg²⁺-coordinating aspartate in the DFG motif; these residues are critical for ATP binding and phosphate transfer (chiou2025thekinasedomain pages 4-7, cook2014ripk1andripk3induced pages 9-10). Structural studies, including high-resolution crystallography and amyloid structural analyses, reveal that RIPK3 can form amyloid-like fibrils via its RHIM domain, suggesting that higher-order oligomerization is central to the formation of functional necrosomes (wu2021theamyloidstructure pages 29-31, shlomovitz2017mechanismsofripk3‐induced pages 1-6). The RHIM domain mediates homotypic interactions with RIPK1 and other RHIM-containing proteins (TRIF, ZBP1), and these interactions are essential for necroptotic signal propagation. In addition, the kinase domain contains an activation loop that undergoes autophosphorylation, a conformational change that enables substrate access and efficient catalysis (chiou2025thekinasedomain pages 25-27, martens2020inhibitorstargetingripk1ripk3 pages 6-8).
6. Regulation  
   RIPK3 activity is under tight regulatory control involving multiple post-translational modifications. Autophosphorylation is a key regulatory mechanism that facilitates activation of its kinase function as well as the subsequent phosphorylation of MLKL (murphy2015posttranslationalcontrolof pages 3-4, shi2018targetingreceptorinteractingserinethreonineprotein pages 63-68). Specific phosphorylation sites such as human Ser227 (mouse T231/S232) are essential for necroptotic signal transduction. In addition to phosphorylation, RIPK3 is regulated by ubiquitination events; for example, K48-linked ubiquitination by E3 ligases such as CHIP and PELI1 can target it for proteasomal degradation, thereby attenuating necroptosis (meng2021theregulationof pages 13-13). Deubiquitinating enzymes like A20 mediate the removal of ubiquitin chains to modulate complex assembly and sustain signaling (meng2021theregulationof pages 3-4). Proteolytic processing by caspase-8 provides an additional layer of regulation, as cleavage of RIPK3 prevents necroptosis and can shift the balance toward apoptosis (shlomovitz2017mechanismsofripk3‐induced pages 6-9, cook2014ripk1andripk3induced pages 12-13). Moreover, conformational regulation is evident as mutations in key residues, such as D161N or D143N, not only affect kinase catalytic activity but also alter conformational states that modulate interaction with binding partners like RIPK1 and caspase-8 (chiou2025thekinasedomain pages 25-27, newton2015ripk1andripk3 pages 1-2).
7. Function  
   RIPK3 is a pivotal mediator of programmed cell death, operating predominantly through necroptosis—a caspase-independent form of cell death characterized by plasma membrane rupture and subsequent inflammation. In the necroptotic pathway, upon activation by death receptors (e.g., TNFR1) or pathogen recognition receptors (e.g., ZBP1, TRIF), RIPK3 associates with RIPK1 via RHIM-mediated interactions to form the necrosome. Within this complex, activated RIPK3 phosphorylates MLKL, which then oligomerizes and translocates to the plasma membrane to compromise membrane integrity (chiou2025thekinasedomain pages 1-4, zhou2024ripk3signalingand pages 2-3). In certain contexts such as viral infections (e.g., orthomyxoviruses, Zika virus), RIPK3 is capable of inducing necroptosis in the nucleus, leading to nuclear envelope disruption and release of genomic DNA into the cytosol (Information section).  
   Beyond necroptosis, RIPK3 plays a role in apoptosis regulation in a kinase-independent manner by acting as a scaffold to facilitate the formation of complexes involving RIPK1, FADD, and caspase-8 (liu2021ripk3signalingand pages 12-13, cook2014ripk1andripk3induced pages 9-10). Furthermore, cell death–independent functions include regulation of inflammatory gene expression via NF-κB and modulation of cellular metabolism. Interactions with metabolic enzymes such as GLUL, GLUD1, and PYGL support a role for RIPK3 in enhancing tricarboxylic acid cycle flux and oxidative phosphorylation, resulting in increased reactive oxygen species (ROS) production. This metabolic reprogramming can contribute to antiviral responses by restricting viral replication, as seen in neuronal cells infected with Zika virus (Information section, liu2021ripk3signalingand pages 1-2). RIPK3 also has been implicated in the activation of the NLRP3 inflammasome and in the subsequent maturation and release of pro-inflammatory cytokines such as IL-1β, thereby linking programmed necrosis to innate immune responses (zhou2024ripk3signalingand pages 5-6, shlomovitz2017mechanismsofripk3‐induced pages 21-23).
8. Other Comments  
   RIPK3 is a target of significant therapeutic interest owing to its central role in necroptosis and inflammatory signaling. Several small molecule inhibitors that target the kinase domain of RIPK3 have been developed experimentally. For example, compounds such as GSK’872 and dabrafenib, originally developed as inhibitors in other kinase contexts, have been shown to inhibit RIPK3 kinase activity and reduce necroptosis in disease models, although challenges remain due to paradoxical induction of apoptosis in some instances (martens2020inhibitorstargetingripk1ripk3 pages 6-8, xia2020discoveryofa pages 9-10). RIPK3 has been implicated in a wide range of disease contexts, including inflammatory bowel disease, ischemia-reperfusion injury, toxic epidermal necrolysis, non-alcoholic fatty liver disease, kidney injury, and certain cancers. Mutations and dysregulation of RIPK3 have been reported to affect the balance between necroptosis and apoptosis, impacting tissue homeostasis and immune responses (moriwaki2017theinflammatorysignal pages 1-4, shlomovitz2017mechanismsofripk3‐induced pages 23-23). Moreover, the involvement of RIPK3 in metabolic regulation, through its interaction with enzymes such as ACOD1/IRG1 in neurons, provides an additional avenue by which it may influence viral replication without directly inducing cell death (Information section). As the complexity of RIPK3 regulation continues to be elucidated through studies investigating its scaffold functions, post-translational modifications, and interactions within multi-protein complexes, it remains a promising, albeit challenging, target for the development of novel anti-inflammatory and cytoprotective agents (martens2020inhibitorstargetingripk1ripk3 pages 6-8, shlomovitz2017mechanismsofripk3‐induced pages 9-13).
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