1. Phylogeny  
   Receptor-interacting serine/threonine-protein kinase 3 (RIPK3), also known as RIP3, is a member of the receptor‐interacting protein kinase family, which is characterized by a conserved N‐terminal kinase domain and a key C‐terminal RIP homotypic interaction motif (RHIM) shared with several other RIP kinases such as RIPK1. (dara2018thereceptorinteracting pages 1-3) Orthologs of RIPK3 have been identified in all mammalian species and its homologs are present in vertebrates, indicating an ancient origin that can be traced back to early eukaryotic evolution. (newton2015ripk1andripk3 pages 1-2) In the context of the human kinome, RIPK3 is classified as a serine/threonine kinase that occupies a distinct branch within the necroptotic signaling network, a classification that is consistent with the evolutionary trends described for kinases in studies of the protein kinase complement of the human genome. (dara2018thereceptorinteracting pages 1-3, newton2015ripk1andripk3 pages 1-2) Its close evolutionary relationship with RIPK1 is testified by the presence of the RHIM domain in both proteins, underscoring a co‐evolution that enables the formation of necrosome complexes. (dara2018thereceptorinteracting pages 1-3)
2. Reaction Catalyzed  
   RIPK3 catalyzes the phosphorylation reaction in which the γ‐phosphate from adenosine triphosphate (ATP) is transferred to specific serine or threonine residues on substrate proteins. (dara2018thereceptorinteracting pages 3-4) In its canonical activity, RIPK3 phosphorylates the pseudokinase mixed lineage kinase domain‐like protein (MLKL), resulting in the conversion of ATP to adenosine diphosphate (ADP) alongside the generation of a phosphorylated substrate and a proton; that is, ATP + [protein]–(Ser/Thr) → ADP + [protein]–(Ser/Thr)-phosphate + H⁺. (liu2021ripk3signalingand pages 1-2)
3. Cofactor Requirements  
   Like many serine/threonine protein kinases, RIPK3 requires divalent metal ions such as Mg²⁺ as a cofactor to coordinate ATP binding and ensure proper catalytic activity. (dara2018thereceptorinteracting pages 3-4)
4. Substrate Specificity  
   RIPK3 exhibits substrate specificity for the phosphorylation of downstream effectors that are central to the execution of necroptosis. (he2009receptorinteractingprotein pages 7-8) Its best‐characterized substrate is MLKL, for which RIPK3 phosphorylates key residues in the activation loop—most notably Thr357 and Ser358 in humans—to trigger a conformational change that exposes the lipid‐binding domain and initiates oligomerization. (liu2021ripk3signalingand pages 4-6) In addition, RIPK3 is known to undergo reciprocal auto‐phosphorylation with RIPK1, with this mutual modification further fine‐tuning the necroptotic response. (dara2018thereceptorinteracting pages 3-4) Although the consensus sequence motif for RIPK3 has not been as explicitly defined as that for classical serine/threonine kinases, recent substrate specificity studies on the human serine/threonine kinome, such as those by Johnson et al. (2023), provide a framework for understanding the general amino acid preferences in kinase substrates; however, for RIPK3, its substrate recognition is largely dictated by the formation of necrosome complexes and the structural context of its substrates. (liu2021ripk3signalingand pages 1-2, Johnson2023)
5. Structure  
   RIPK3 is a protein of approximately 518 amino acids in humans and contains two major structural domains: an N‐terminal serine/threonine kinase domain and a C‐terminal RHIM domain. (dara2018thereceptorinteracting pages 1-3, shi2018targetingreceptorinteractingserinethreonineprotein pages 63-68) The kinase domain is responsible for binding ATP and catalyzing the phosphorylation reaction and includes the canonical motifs found in the kinase superfamily, such as the glycine-rich loop, the catalytic loop with the conserved DFG motif, the activation loop (T-loop), and the C-helix; these structural elements together form the catalytic core essential for enzymatic activity. (shi2018targetingreceptorinteractingserinethreonineprotein pages 63-68, newton2015ripk1andripk3 pages 6-7) The C-terminal RHIM domain, on the other hand, mediates homotypic protein–protein interactions with other RHIM-containing proteins such as RIPK1, TRIF, and ZBP1, which is a critical prerequisite for necrosome assembly and subsequent activation of necroptosis. (dara2018thereceptorinteracting pages 1-3, zhou2024ripk3signalingand pages 1-2) Structural modeling and experimental analyses, including crystallographic studies and predictions from AlphaFold, have confirmed that the kinase domain adopts a typical bilobal architecture with the N-lobe primarily involved in ATP binding and the larger C-lobe providing the substrate-binding region. (shi2018targetingreceptorinteractingserinethreonineprotein pages 68-75)
6. Regulation  
   RIPK3 is regulated by multiple post-translational modifications that influence its catalytic activity and the type of cell death pathway executed. (dara2018thereceptorinteracting pages 16-18) Autophosphorylation of the kinase domain, including key residues such as serine 227 (in humans) and corresponding residues in the mouse ortholog, is critical for its activation and its ability to phosphorylate downstream effectors like MLKL. (shi2018targetingreceptorinteractingserinethreonineprotein pages 63-68, dara2018thereceptorinteracting pages 3-4) Additional phosphorylation events, such as those at serine 164 and threonine 165, have been shown to serve as molecular switches that modulate the balance between necroptotic and apoptotic signaling; phosphorylated mutants mimicking these modifications promote the formation of complexes with RIPK1, FADD, and caspase-8, thereby inducing apoptosis. (li2021aphosphorylationof pages 5-6, li2021aphosphorylationof pages 6-7) Moreover, reciprocal phosphorylation between RIPK3 and RIPK1, as well as regulation by deubiquitinating enzymes, further refines its activity and the stability of the necrosome complex. (he2009receptorinteractingprotein pages 7-8, newton2015ripk1andripk3 pages 7-7) Regulatory interactions with caspase-8 are also important, as caspase-8-mediated cleavage of RIPK3 acts as a negative feedback mechanism to limit necroptotic signaling. (dara2018thereceptorinteracting pages 16-18)
7. Function  
   RIPK3 is a multifunctional kinase that plays a central role in mediating regulated cell death and inflammatory responses. (dara2018thereceptorinteracting pages 1-3) Its best-established function is as an activator of necroptosis: upon activation by death receptor stimulation (such as TNF-α) or by viral sensors like ZBP1, RIPK3 forms a necrosome complex with RIPK1 and subsequently phosphorylates MLKL. (dara2018thereceptorinteracting pages 3-4, liu2021ripk3signalingand pages 1-2) Phosphorylated MLKL undergoes oligomerization and translocates to the plasma membrane, where it disrupts membrane integrity by forming pores, leading to calcium influx, cell swelling, and ultimately, necrotic cell death. (dara2018thereceptorinteracting pages 3-4, liu2021ripk3signalingand pages 4-6) In addition to driving necroptosis, RIPK3 is involved in apoptosis signaling in certain cellular contexts; when its kinase activity is compromised or its regulatory phosphorylation pattern is altered, RIPK3 can recruit RIPK1, FADD, and caspase-8 to initiate apoptosis independent of MLKL. (mandal2014rip3inducesapoptosis pages 15-15, li2021aphosphorylationof pages 6-7) Beyond cell death, RIPK3 influences inflammatory signaling by promoting the transcriptional upregulation of cytokines and chemokines through NF-κB and MAPK pathways, and it can indirectly restrict viral replication via cell death-independent mechanisms, as evidenced in studies involving Zika virus infection in neurons. (dara2018thereceptorinteracting pages 9-11, ying2021theroleof pages 3-4) Furthermore, RIPK3 has been shown to bind and enhance the enzymatic activities of metabolic enzymes such as GLUL, GLUD1, and PYGL, which may lead to increased tricarboxylic acid cycle activity and heightened reactive oxygen species production, linking its activity to cellular metabolic reprogramming. (Information section, liu2021ripk3signalingand pages 1-2)
8. Other Comments  
   RIPK3 is an attractive therapeutic target due to its pivotal role in necroptosis and inflammatory diseases. (chung2022highreceptorinteractingserinethreonineprotein pages 1-2) Experimental inhibitors, including necrostatins that primarily target RIPK1 and indirectly modulate RIPK3 activity, have shown promising effects in preclinical models of ischemia-reperfusion injury, liver disease, and neurodegeneration. (fayaz2016novelripk3inhibitors pages 1-1, martens2020inhibitorstargetingripk1ripk3 pages 2-4) Moreover, structure-based drug design efforts have led to the identification of novel RIPK3 inhibitors that offer neuroprotection in post-ischemic settings. (fayaz2016novelripk3inhibitors pages 1-1, martens2020inhibitorstargetingripk1ripk3 pages 4-6) Mutations affecting RIPK3 activity, such as kinase‐dead variants (e.g., D161N), have been associated with embryonic lethality in murine models, underscoring the essential nature of its kinase function in development. (dara2018thereceptorinteracting pages 22-27, newton2015ripk1andripk3 pages 7-7) Additionally, aberrant or elevated expression of RIPK3 has been linked to pathological conditions including chronic liver diseases, certain cancers, and cardiovascular disorders, further emphasizing the role of RIPK3 in disease progression and the potential benefits of its pharmacological inhibition. (dara2018thereceptorinteracting pages 9-11, deroo2020theroleof pages 11-13) The dual functionality of RIPK3 in mediating both necroptosis and apoptosis—as well as its involvement in metabolic reprogramming—necessitates careful consideration in the development of selective inhibitors. (liu2021ripk3signalingand pages 12-13, silke2015thediverserole pages 1-2)
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