1. Phylogeny – Receptor‐interacting serine/threonine‐protein kinase 3 (RIPK3) is a member of the receptor‐interacting protein (RIP) kinase family, a specialized subgroup within the serine/threonine kinase superfamily that is broadly conserved among vertebrates. Orthologs of RIPK3 have been identified in diverse mammalian species, and comparative evolutionary analyses have demonstrated that RIPK3 shares essential catalytic features with other RIP kinases such as RIPK1 while simultaneously diverging to develop unique regulatory interaction motifs. In particular, RIPK3 possesses a characteristic RIP homotypic interaction motif (RHIM) in its C‐terminal region that mediates specific protein–protein interactions with RIPK1 and additional RHIM‐containing signaling proteins. This domain architecture and its conservation across species place RIPK3 within a core set of kinases that emerged early in eukaryotic evolution and are pivotal for innate immune responses and programmed cell death signaling events (fay2025evolutionaryandfunctional pages 1-4, lv2022comparativeandevolutionary pages 1-3). Furthermore, studies in plant and animal systems have underscored that although members of the RIP kinase family share a common catalytic structure, RIPK3 has acquired specialized functions related to necroptosis and inflammatory signaling that are not evident in kinases with simpler domain organizations (dardick2006plantandanimal pages 10-11, fay2025evolutionaryandfunctional pages 17-19).
2. Reaction Catalyzed – RIPK3 is a serine/threonine–protein kinase that catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on its substrate proteins. The catalytic reaction follows the canonical scheme:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction is fundamental to the kinase activity of RIPK3, enabling signal transduction that regulates downstream events in programmed cell death and inflammatory responses (wu2012investigationofreceptor pages 11-12, zu2021quantitativeanalysisof pages 11-11).
3. Cofactor Requirements – The kinase activity of RIPK3, like that of other serine/threonine–protein kinases, is strictly dependent on the availability of divalent metal ions. In particular, Mg²⁺ serves as an essential cofactor that facilitates the binding of ATP and the proper positioning required for efficient phosphoryl transfer. The requirement for Mg²⁺ is consistent with the structural demands of the active site, where it coordinates with both ATP and catalytic residues to promote a high‐efficiency phosphoryl transfer reaction (wu2012investigationofreceptor pages 11-12, zu2021quantitativeanalysisof pages 11-11).
4. Substrate Specificity – The substrate specificity of RIPK3 is defined by its ability to recognize and phosphorylate serine/threonine residues on critical target proteins within death and inflammatory signaling complexes. Experimentally, RIPK3 is known to phosphorylate the pseudokinase mixed lineage kinase domain–like protein (MLKL), thereby triggering its oligomerization and subsequent translocation to the plasma membrane to induce necroptosis. In addition, RIPK3 engages in reciprocal phosphorylation with RIPK1, which further modulates the assembly and function of the necrosome complex. Although a precise consensus substrate motif for RIPK3 has not been fully delineated, evidence derived from large-scale substrate specificity atlases suggests that its substrate recognition is influenced by the three-dimensional context provided by protein–protein interactions in RHIM-containing signaling complexes, rather than through a linear peptide consensus similar to that of other serine/threonine kinases (johnson2023anatlasof pages 10-11, licheva2022phosphoregulationofthe pages 15-16, martens2020inhibitorstargetingripk1ripk3 pages 4-6).
5. Structure – The three-dimensional organization of RIPK3 is characterized by a modular domain structure consisting of an N-terminal kinase domain and a C-terminal region that harbors the RIP homotypic interaction motif (RHIM). The N-terminal kinase domain adopts the typical bilobal fold seen in serine/threonine kinases: an N-lobe comprised predominantly of β-strands and a C-lobe that is largely α-helical, together forming the catalytic core. Key features of this domain include an activation loop containing the conserved DFG (Asp-Phe-Gly) motif, which is critical for the binding of ATP and coordination of Mg²⁺ ions, and a catalytic lysine residue that is required for phosphotransfer activity. Structural studies using crystallography and computational predictions such as those from AlphaFold illustrate that conformational changes in the activation loop and reorientation of the C-helix are central to the transition of RIPK3 between inactive and active states. The C-terminal RHIM domain is indispensable for mediating amyloid-like interactions with its partner proteins, most notably RIPK1. Such interactions facilitate the formation of the necrosome complex, an oligomeric assembly that is required for propagating necroptotic signaling (lopez2019functionalcharacterizationof pages 69-81, mace2021there’smoreto pages 32-39, johnson2023anatlasof pages 7-8).
6. Regulation – The regulatory mechanisms governing RIPK3 activity are multifaceted, involving both post-translational modifications and critical protein–protein interactions. A central regulatory event is the reciprocal auto- and trans-phosphorylation between RIPK3 and RIPK1, which enhances the catalytic activity of each kinase while promoting necrosome assembly. Phosphorylation events, especially those occurring within the activation loop of the kinase domain, are essential for transitioning RIPK3 into its fully active state capable of phosphorylating downstream substrates such as MLKL. Additionally, regulatory phosphorylation by upstream kinases, including PLK1 under specific cell cycle conditions, adds a layer of control ensuring that RIPK3’s activity is coordinated with cell cycle progression (du2021ripk1dephosphorylationand pages 14-14, martens2020inhibitorstargetingripk1ripk3 pages 2-4, mace2021there’smoreto pages 28-32). Beyond phosphorylation, RIPK3 interacts with metabolic enzymes such as GLUL, GLUD1, and PYGL, which may provide feedback regulation that integrates metabolic status with necroptotic and inflammatory signaling. Moreover, while its kinase activity is essential for necroptosis, kinase-inactive forms of RIPK3 are capable of influencing apoptotic signaling through interactions with RIPK1, FADD, and CASP8, demonstrating that non-catalytic scaffold functions also play a role in determining cell fate (martens2020inhibitorstargetingripk1ripk3 pages 4-6, mace2021there’smoreto pages 16-20, moriwaki2017theinflammatorysignal pages 13-16).
7. Function – Biologically, RIPK3 is a central signaling hub that orchestrates multiple cell death and inflammatory pathways. Its best-characterized function is in the activation of necroptosis. Upon stimulation by death-inducing signals such as those from the TNF-α family or by viral infection via ZBP1 sensing of double-stranded Z-RNA structures, RIPK3 becomes activated and phosphorylates MLKL. Phosphorylated MLKL oligomerizes and translocates to the plasma membrane where it disrupts membrane integrity, resulting in necrotic cell death characterized by calcium influx and leakage of cellular contents, thereby eliciting an inflammatory response. In addition to its classical role in necroptosis, RIPK3 may also modulate apoptotic signaling in a process that is independent of its catalytic activity on MLKL but instead requires the coordinated action of RIPK1, FADD, and CASP8. Notably, in certain cell types such as neurons infected with Zika virus, RIPK3 functions in a cell death–independent manner by cooperating with ZBP1 to initiate a transcriptional program that upregulates the enzyme ACOD1/IRG1; this leads to the production of itaconate, which inhibits succinate dehydrogenase and reprograms cellular metabolism to suppress viral replication. Additionally, RIPK3 can bind and enhance the activity of metabolic enzymes including GLUL, GLUD1, and PYGL, thereby potentially stimulating the tricarboxylic acid cycle and oxidative phosphorylation, with resultant increases in reactive oxygen species (ROS) production. These functions underscore the role of RIPK3 not only as a mediator of programmed cell death but also as a key regulator of antiviral responses and metabolic adaptations in the context of inflammatory stimuli (dardick2006plantandanimal pages 10-11, liu2021ripk3signalingand pages 1-2, zu2021quantitativeanalysisof pages 11-11).
8. Other Comments – Pharmacological studies have highlighted the therapeutic interest in modulating necroptotic signaling by targeting RIPK family kinases. Inhibitor studies, although more advanced for RIPK1, have underscored the potential of interfering with RIPK3’s kinase activity to ameliorate excessive inflammation and tissue injury in neurodegenerative and inflammatory diseases. Structural mutations that affect key motifs within the kinase domain, such as alterations in the DFG motif or disruptions in RHIM-mediated oligomerization, can profoundly alter RIPK3’s ability to propagate necroptosis and influence downstream metabolic and inflammatory responses. Dysregulation of RIPK3 signaling is implicated in a variety of pathological conditions, including acute tissue injuries, chronic inflammatory diseases, and viral infections, thereby positioning RIPK3 as a promising target for the development of novel therapeutic agents. The interplay between its catalytic activity and scaffold functions—where even kinase-dead mutants can modulate apoptosis through interaction with RIPK1, FADD, and CASP8—calls for a nuanced understanding of RIPK3 regulation in disease contexts (martens2020inhibitorstargetingripk1ripk3 pages 4-6, mace2021there’smoreto pages 16-20, moriwaki2017theinflammatorysignal pages 13-16).
9. References
10. alexa2015structuralassemblyof pages 6-6
11. dardick2006plantandanimal pages 10-11
12. du2021ripk1dephosphorylationand pages 14-14
13. eichner2024proteinproteininteractionsin pages 160-163
14. fay2025evolutionaryandfunctional pages 1-4
15. fay2025evolutionaryandfunctional pages 17-19
16. gupta2021plk1mediateds369phosphorylation pages 27-31
17. jha2025deeplearningcoupledproximity pages 24-26
18. jha2025deeplearningcoupledproximity pages 7-10
19. johnson2023anatlasof pages 10-11
20. johnson2023anatlasof pages 4-5
21. johnson2023anatlasof pages 7-8
22. licheva2022phosphoregulationofthe pages 15-16
23. liu2021ripk3signalingand pages 1-2
24. lopez2019functionalcharacterizationof pages 13-18
25. lopez2019functionalcharacterizationof pages 18-22
26. lopez2019functionalcharacterizationof pages 69-81
27. lopez2019peak3c19orf35pseudokinasea pages 1-2
28. lv2022comparativeandevolutionary pages 1-3
29. mace2021there’smoreto pages 1-5
30. mace2021there’smoreto pages 22-25
31. mace2021there’smoreto pages 32-39
32. mace2021there’smoreto pages 8-11
33. martens2020inhibitorstargetingripk1ripk3 pages 1-2
34. martens2020inhibitorstargetingripk1ripk3 pages 2-4
35. martens2020inhibitorstargetingripk1ripk3 pages 4-6
36. moriwaki2017theinflammatorysignal pages 13-16
37. wu2012investigationofreceptor pages 11-12
38. zhuang2020smallmoleculeinhibitorsof pages 29-32
39. zu2021quantitativeanalysisof pages 11-11

References

1. (alexa2015structuralassemblyof pages 6-6): Anita Alexa, Gergő Gógl, Gábor Glatz, Ágnes Garai, András Zeke, János Varga, Erika Dudás, Norbert Jeszenői, Andrea Bodor, Csaba Hetényi, and Attila Reményi. Structural assembly of the signaling competent erk2–rsk1 heterodimeric protein kinase complex. Proceedings of the National Academy of Sciences, 112:2711-2716, Feb 2015. URL: https://doi.org/10.1073/pnas.1417571112, doi:10.1073/pnas.1417571112. This article has 53 citations.
2. (dardick2006plantandanimal pages 10-11): Christopher Dardick and Pamela Ronald. Plant and animal pathogen recognition receptors signal through non-rd kinases. PLoS Pathogens, 2:e2, Jan 2006. URL: https://doi.org/10.1371/journal.ppat.0020002, doi:10.1371/journal.ppat.0020002. This article has 325 citations and is from a highest quality peer-reviewed journal.
3. (du2021ripk1dephosphorylationand pages 14-14): Jingchun Du, Yougui Xiang, Hua Liu, Shuzhen Liu, Ashwani Kumar, Chao Xing, and Zhigao Wang. Ripk1 dephosphorylation and kinase activation by ppp1r3g/pp1γ promote apoptosis and necroptosis. Nature Communications, Dec 2021. URL: https://doi.org/10.1038/s41467-021-27367-5, doi:10.1038/s41467-021-27367-5. This article has 27 citations and is from a highest quality peer-reviewed journal.
4. (eichner2024proteinproteininteractionsin pages 160-163): A Eichner. Protein-protein interactions in cell cycle proteins: an in silico investigation of two important players. Unknown journal, 2024.
5. (fay2025evolutionaryandfunctional pages 1-4): Elizabeth J. Fay, Kolya Isterabadi, Charles M. Rezanka, Jessica Le, and Matthew D. Daugherty. Evolutionary and functional analyses reveal a role for the rhim in tuning ripk3 activity across vertebrates. bioRxiv, Aug 2024. URL: https://doi.org/10.1101/2024.05.09.593370, doi:10.1101/2024.05.09.593370. This article has 1 citations.
6. (fay2025evolutionaryandfunctional pages 17-19): Elizabeth J. Fay, Kolya Isterabadi, Charles M. Rezanka, Jessica Le, and Matthew D. Daugherty. Evolutionary and functional analyses reveal a role for the rhim in tuning ripk3 activity across vertebrates. bioRxiv, Aug 2024. URL: https://doi.org/10.1101/2024.05.09.593370, doi:10.1101/2024.05.09.593370. This article has 1 citations.
7. (gupta2021plk1mediateds369phosphorylation pages 27-31): Kartik Gupta and Bo Liu. Plk1-mediated s369 phosphorylation of ripk3 during g2 and m phases enables its ripoptosome incorporation and activity. iScience, 24:102320, Apr 2021. URL: https://doi.org/10.1016/j.isci.2021.102320, doi:10.1016/j.isci.2021.102320. This article has 15 citations and is from a peer-reviewed journal.
8. (jha2025deeplearningcoupledproximity pages 24-26): Kanchan Jha, Daichi Shonai, Aditya Parekh, Akiyoshi Uezu, Tomoyuki Fujiyama, Hikari Yamamoto, Pooja Parameswaran, Masashi Yanagisawa, Rohit Singh, and Scott H. Soderling. Deep learning-coupled proximity proteomics to deconvolve kinase signaling in vivo. BioRxiv, Apr 2025. URL: https://doi.org/10.1101/2025.04.27.650849, doi:10.1101/2025.04.27.650849. This article has 0 citations.
9. (jha2025deeplearningcoupledproximity pages 7-10): Kanchan Jha, Daichi Shonai, Aditya Parekh, Akiyoshi Uezu, Tomoyuki Fujiyama, Hikari Yamamoto, Pooja Parameswaran, Masashi Yanagisawa, Rohit Singh, and Scott H. Soderling. Deep learning-coupled proximity proteomics to deconvolve kinase signaling in vivo. BioRxiv, Apr 2025. URL: https://doi.org/10.1101/2025.04.27.650849, doi:10.1101/2025.04.27.650849. This article has 0 citations.
10. (johnson2023anatlasof pages 10-11): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 422 citations and is from a highest quality peer-reviewed journal.
11. (johnson2023anatlasof pages 4-5): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 422 citations and is from a highest quality peer-reviewed journal.
12. (johnson2023anatlasof pages 7-8): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 422 citations and is from a highest quality peer-reviewed journal.
13. (licheva2022phosphoregulationofthe pages 15-16): Mariya Licheva, Babu Raman, Claudine Kraft, and Fulvio Reggiori. Phosphoregulation of the autophagy machinery by kinases and phosphatases. Autophagy, 18:104-123, May 2022. URL: https://doi.org/10.1080/15548627.2021.1909407, doi:10.1080/15548627.2021.1909407. This article has 67 citations and is from a domain leading peer-reviewed journal.
14. (liu2021ripk3signalingand pages 1-2): Shanhui Liu, Kanak Joshi, Mitchell F. Denning, and Jiwang Zhang. Ripk3 signaling and its role in the pathogenesis of cancers. Cellular and Molecular Life Sciences, 78:7199-7217, Oct 2021. URL: https://doi.org/10.1007/s00018-021-03947-y, doi:10.1007/s00018-021-03947-y. This article has 59 citations and is from a domain leading peer-reviewed journal.
15. (lopez2019functionalcharacterizationof pages 13-18): M Lopez. Functional characterization of peak3/c19orf35 pseudokinase and its role in regulation of crkii-dependent signaling. Unknown journal, 2019.
16. (lopez2019functionalcharacterizationof pages 18-22): M Lopez. Functional characterization of peak3/c19orf35 pseudokinase and its role in regulation of crkii-dependent signaling. Unknown journal, 2019.
17. (lopez2019functionalcharacterizationof pages 69-81): M Lopez. Functional characterization of peak3/c19orf35 pseudokinase and its role in regulation of crkii-dependent signaling. Unknown journal, 2019.
18. (lopez2019peak3c19orf35pseudokinasea pages 1-2): Mitchell L. Lopez, Megan Lo, Jennifer E. Kung, Małgorzata Dudkiewicz, Gwendolyn M. Jang, John Von Dollen, Jeffrey R. Johnson, Nevan J. Krogan, Krzysztof Pawłowski, and Natalia Jura. Peak3/c19orf35 pseudokinase, a new nfk3 kinase family member, inhibits crkii through dimerization. Proceedings of the National Academy of Sciences, 116:15495-15504, Jul 2019. URL: https://doi.org/10.1073/pnas.1906360116, doi:10.1073/pnas.1906360116. This article has 30 citations.
19. (lv2022comparativeandevolutionary pages 1-3): Shangge Lv, Yu Jiang, Yuzheng Li, Rui-hua Huang, Lingyu Peng, Zhaoyin Ma, Nan Lu, Xiaoying Lin, and Jie Yan. Comparative and evolutionary analysis of rip kinases in immune responses. Frontiers in Genetics, Oct 2022. URL: https://doi.org/10.3389/fgene.2022.796291, doi:10.3389/fgene.2022.796291. This article has 7 citations and is from a peer-reviewed journal.
20. (mace2021there’smoreto pages 1-5): Peter D. Mace and James M. Murphy. There’s more to death than life: noncatalytic functions in kinase and pseudokinase signaling. The Journal of Biological Chemistry, Apr 2021. URL: https://doi.org/10.1016/j.jbc.2021.100705, doi:10.1016/j.jbc.2021.100705. This article has 76 citations.
21. (mace2021there’smoreto pages 22-25): Peter D. Mace and James M. Murphy. There’s more to death than life: noncatalytic functions in kinase and pseudokinase signaling. The Journal of Biological Chemistry, Apr 2021. URL: https://doi.org/10.1016/j.jbc.2021.100705, doi:10.1016/j.jbc.2021.100705. This article has 76 citations.
22. (mace2021there’smoreto pages 32-39): Peter D. Mace and James M. Murphy. There’s more to death than life: noncatalytic functions in kinase and pseudokinase signaling. The Journal of Biological Chemistry, Apr 2021. URL: https://doi.org/10.1016/j.jbc.2021.100705, doi:10.1016/j.jbc.2021.100705. This article has 76 citations.
23. (mace2021there’smoreto pages 8-11): Peter D. Mace and James M. Murphy. There’s more to death than life: noncatalytic functions in kinase and pseudokinase signaling. The Journal of Biological Chemistry, Apr 2021. URL: https://doi.org/10.1016/j.jbc.2021.100705, doi:10.1016/j.jbc.2021.100705. This article has 76 citations.
24. (martens2020inhibitorstargetingripk1ripk3 pages 1-2): Sofie Martens, Sam Hofmans, Wim Declercq, Koen Augustyns, and Peter Vandenabeele. Inhibitors targeting ripk1/ripk3: old and new drugs. Trends in Pharmacological Sciences, 41:209-224, Mar 2020. URL: https://doi.org/10.1016/j.tips.2020.01.002, doi:10.1016/j.tips.2020.01.002. This article has 149 citations and is from a highest quality peer-reviewed journal.
25. (martens2020inhibitorstargetingripk1ripk3 pages 2-4): Sofie Martens, Sam Hofmans, Wim Declercq, Koen Augustyns, and Peter Vandenabeele. Inhibitors targeting ripk1/ripk3: old and new drugs. Trends in Pharmacological Sciences, 41:209-224, Mar 2020. URL: https://doi.org/10.1016/j.tips.2020.01.002, doi:10.1016/j.tips.2020.01.002. This article has 149 citations and is from a highest quality peer-reviewed journal.
26. (martens2020inhibitorstargetingripk1ripk3 pages 4-6): Sofie Martens, Sam Hofmans, Wim Declercq, Koen Augustyns, and Peter Vandenabeele. Inhibitors targeting ripk1/ripk3: old and new drugs. Trends in Pharmacological Sciences, 41:209-224, Mar 2020. URL: https://doi.org/10.1016/j.tips.2020.01.002, doi:10.1016/j.tips.2020.01.002. This article has 149 citations and is from a highest quality peer-reviewed journal.
27. (moriwaki2017theinflammatorysignal pages 13-16): K. Moriwaki and F.K.-M. Chan. The inflammatory signal adaptor ripk3: functions beyond necroptosis. International Review of Cell and Molecular Biology, pages 253-275, Jan 2017. URL: https://doi.org/10.1016/bs.ircmb.2016.08.007, doi:10.1016/bs.ircmb.2016.08.007. This article has 107 citations and is from a peer-reviewed journal.
28. (wu2012investigationofreceptor pages 11-12): Xiurong Wu, Lili Tian, Jie Li, Yingying Zhang, Victor Han, Yuanyue Li, Xiaozheng Xu, Hanjie Li, Xi Chen, Jinan Chen, Wenhai Jin, Yongming Xie, Jiahuai Han, and Chuan-Qi Zhong. Investigation of receptor interacting protein (rip3)-dependent protein phosphorylation by quantitative phosphoproteomics. Molecular & Cellular Proteomics, 11:1640-1651, Dec 2012. URL: https://doi.org/10.1074/mcp.m112.019091, doi:10.1074/mcp.m112.019091. This article has 85 citations.
29. (zhuang2020smallmoleculeinhibitorsof pages 29-32): Chunlin Zhuang and Fen‐er Chen. Small-molecule inhibitors of necroptosis: current status and perspectives. Journal of medicinal chemistry, Feb 2020. URL: https://doi.org/10.1021/acs.jmedchem.9b01317, doi:10.1021/acs.jmedchem.9b01317. This article has 86 citations and is from a highest quality peer-reviewed journal.
30. (zu2021quantitativeanalysisof pages 11-11): Rui Zu, Zhen Yu, Jing Zhao, Xiaojuan Lu, Wei Liang, Le Sun, Chenfang Si, Kezhou Zhu, Tian Zhang, Ganquan Li, Mengmeng Zhang, Yaoyang Zhang, Nan Liu, Junying Yuan, and Bing Shan. Quantitative analysis of phosphoproteome in necroptosis reveals a role of trim28 phosphorylation in promoting necroptosis-induced cytokine production. Cell Death & Disease, Oct 2021. URL: https://doi.org/10.1038/s41419-021-04290-7, doi:10.1038/s41419-021-04290-7. This article has 10 citations.