1. Phylogeny  
   IRAK3 is a member of the interleukin-1 receptor-associated kinase (IRAK) family, which also comprises the catalytically active IRAK1, IRAK2, and IRAK4. Although it shares the common domain architecture of an N-terminal death domain (DD), a central kinase (or pseudokinase) domain, and a C-terminal region, IRAK3 has diverged in function from its paralogs by losing key catalytic residues. Phylogenetic analyses show that IRAK3 is broadly conserved across vertebrates, underscoring its ancient role in regulating innate immunity. Within the kinome, IRAK3 is grouped with the Pelle family kinases and is considered a regulatory or pseudokinase that emerged early during evolution of TLR/IL-1 receptor signaling complexes. Its orthologs can be identified in species ranging from mammals to other vertebrates, placing it in an evolutionarily conserved subgroup of IRAK proteins that have adapted specialized roles in modulating immune responses (degorce2020discoveryofproteolysistargeting pages 1-2, lange2021dimericstructureof pages 1-3).
2. Reaction Catalyzed  
   Unlike conventional protein kinases that catalyze ATP-dependent phosphorylation of protein substrates, IRAK3 functions primarily as a regulatory scaffold rather than as an active enzyme. It lacks the full complement of catalytic residues found in active kinases and therefore does not efficiently mediate phosphoryl-transfer to a target substrate. Instead, IRAK3 modulates signaling downstream of immune receptors by inhibiting the phosphorylation and subsequent dissociation of IRAK1 and IRAK4 from the Toll-like receptor (TLR) signaling complex. In effect, its “reaction” is one of interference or stabilization of protein–protein interactions within the Myddosome, rather than the transfer of a phosphate group from ATP to serine/threonine residues (degorce2020discoveryofproteolysistargeting pages 1-2, zhou2024il1receptorassociatedkinase pages 1-2).
3. Cofactor Requirements  
   Although IRAK3 is classified as a pseudokinase with minimal catalytic activity, its structured kinase-like domain still retains an ATP-binding pocket. Structural studies indicate that IRAK3 binds ATP with very low affinity relative to active kinases; for example, its nucleotide interaction is demonstrably weak, with an estimated dissociation constant (Kd) that suggests little to no occupancy under physiological conditions. Despite having a conserved ATP-binding site element, IRAK3 does not require the metal ion cofactors (such as Mg²⁺) in the conventional sense for catalysis. Rather, any bound ATP is likely involved in maintaining structural conformation or allosteric regulation rather than driving a phosphoryl-transfer reaction (lange2021dimericstructureof pages 6-7).
4. Substrate Specificity  
   IRAK3 does not exhibit typical kinase substrate specificity since it lacks robust catalytic phosphorylation activity. Instead, its regulatory role is mediated by specific interactions with other proteins within the TLR/IL-1 receptor signaling complex. Notably, IRAK3 is reported to inhibit the dissociation of IRAK1 and IRAK4 from the receptor complex, possibly through binding interfaces that recognize these kinases or associated adaptor proteins such as MyD88. Consequently, rather than phosphorylating a consensus motif (for example, RxRxx[pST] as in many serine/threonine kinases), IRAK3 functions by sequestering signaling molecules thereby modulating the downstream activation of NF-κB and related transcription factors (degorce2020discoveryofproteolysistargeting pages 1-2, gurkan2024theirakmdeath pages 1-2).
5. Structure  
   IRAK3 is composed of multiple functional regions. At its N-terminus, a death domain mediates protein–protein interactions necessary for assembly of the Myddosome. The central region, normally defined as a kinase domain, has evolved into a pseudokinase domain that adopts a closed, pseudoactive conformation reminiscent of active kinases. Structural studies, notably crystallography at 2.9 Å resolution, reveal that IRAK3 forms a unique head-to-head homodimer mediated by interactions centered on its αC-helices and further stabilized by disulfide bridges and conserved hydrophobic contacts (e.g., involving residues such as L210 and E214). Its kinase domain harbors key modifications: canonical motifs are altered (for instance, the HRD motif is replaced by a ‘CGS’ sequence and the DFG motif is substituted by DFA), and the G-loop is notably stabilized by a conserved hydrophobic residue (F177) that forms a rigid network with nearby secondary structure elements. The overall three-dimensional structure preserves the typical bilobal kinase fold, yet the ATP-binding pocket is largely accessible to inhibitors such as staurosporine rather than ATP under physiological conditions (degorce2020discoveryofproteolysistargeting pages 1-2, lange2021dimericstructureof pages 3-4, lange2021dimericstructureof pages 6-7).
6. Regulation  
   IRAK3 regulates downstream immune responses primarily by functioning as a negative modulator of TLR and IL-1 receptor signaling. Its regulatory mechanisms are as much structural as they are post-translational. Dimerization via its pseudokinase domain, which occurs through a head-to-head interface and involves key residues that are sensitive to redox conditions (e.g., the formation of disulfide bridges), is central to its activity. This dimerization may allosterically modulate interactions with IRAK4 or other signaling components, thereby inhibiting the phosphorylation events that are necessary for full activation of pro-inflammatory pathways. Additionally, studies have suggested that IRAK3 might stabilize the receptor complex by preventing the disassembly or excessive phosphorylation of IRAK1 and IRAK4, thereby attenuating NF-κB activation. Although specific post-translational modifications (such as phosphorylation on regulatory sites) have not been fully characterized for IRAK3, its interactions and dimerization status are clearly critical for its inhibitory function (lange2021dimericstructureof pages 7-8, lange2021dimericstructureof pages 11-12, gurkan2024theirakmdeath pages 15-16).
7. Function  
   IRAK3 is predominantly expressed in leukocytes such as monocytes, macrophages, and neutrophils, where it plays an essential role in modulating innate immune responses. Its primary function is to act as a negative regulator downstream of TLR and IL-1 receptor engagement, thereby dampening the production of pro-inflammatory cytokines. By inhibiting the phosphorylation and subsequent dissociation of IRAK1 and IRAK4 from the receptor complex, IRAK3 stabilizes the Myddosome and prevents overactivation of NF-κB-dependent transcription. This role is critical in preventing hyperinflammatory states and autoimmune manifestations. In addition, under certain pathological conditions—such as IL-33-induced lung inflammation and in specific cancer contexts including colorectal cancer and lung adenocarcinoma—IRAK3 has been implicated in modulating cytokine expression profiles (increasing IL6, CSF3, CXCL2, and CCL5 mRNAs) and influencing immune cell infiltration, thereby affecting both inflammatory and tumor microenvironment dynamics (degorce2020discoveryofproteolysistargeting pages 1-2, zhou2024il1receptorassociatedkinase pages 1-2).
8. Other Comments  
   Although lacking classical catalytic activity, IRAK3 is a promising therapeutic target due to its central role in regulating immune receptor signaling. Recent studies have exploited its unique ATP-binding pocket—despite its weak affinity for ATP—to develop proteolysis-targeting chimeras (PROTACs) that selectively induce its degradation, thereby modulating the inflammatory response. Disease associations include its involvement in immune dysregulation observed in conditions such as persistent asthma, certain cancers (e.g., colorectal cancer and lung adenocarcinoma), and neuroinflammatory conditions. Notable mutations, some of which cluster on conserved surfaces involved in dimerization and interaction with IRAK4, have been linked to altered function and may serve as biomarkers or therapeutic targets. Current areas of active research focus on elucidating the precise structural mechanisms of its allosteric regulation and on developing small molecules or biological agents to modulate its stability and interactions, with the goal of fine-tuning immune responses in pathological conditions (degorce2020discoveryofproteolysistargeting pages 1-2, lange2021dimericstructureof pages 9-10, gurkan2024theirakmdeath pages 1-2).
9. References  
   degorce2020discoveryofproteolysistargeting pages 1-2; gurkan2024theirakmdeath pages 1-2; gurkan2024theirakmdeath pages 15-16; lange2021dimericstructureof pages 1-3; lange2021dimericstructureof pages 10-11; lange2021dimericstructureof pages 11-12; lange2021dimericstructureof pages 3-4; lange2021dimericstructureof pages 4-5; lange2021dimericstructureof pages 6-7; lange2021dimericstructureof pages 7-8; lange2021dimericstructureof pages 9-10; wang2025interleukin1receptorassociatedkinase3 pages 17-19; wang2025interleukin1receptorassociatedkinase3 pages 19-21; wang2025interleukin1receptorassociatedkinase3 pages 21-22; zhou2024il1receptorassociatedkinase pages 1-2; zhou2024il1receptorassociatedkinase pages 17-19; zhou2024il1receptorassociatedkinase pages 12-13; zhou2024il1receptorassociatedkinase pages 16-17; zhou2024il1receptorassociatedkinase pages 22-23; lange2021dimericstructureof pages 12-13; lange2021dimericstructureof pages 13-13; lange2021dimericstructureof pages 13-14; lange2021dimericstructureof pages 14-16; lange2021dimericstructureof pages 16-17; lange2021dimericstructureof pages 17-19; lange2021dimericstructureof pages 5-6; lange2021dimericstructureof pages 8-9; zarrin2021kinaseinhibitionin pages 22-22; zhou2024il1receptorassociatedkinase pages 11-12.

References

1. (degorce2020discoveryofproteolysistargeting pages 1-2): Sébastien L. Degorce, Omid Tavana, Erica Banks, Claire Crafter, Lakshmaiah Gingipalli, David Kouvchinov, Yumeng Mao, Fiona Pachl, Anisha Solanki, Viia Valge-Archer, Bin Yang, and Scott D. Edmondson. Discovery of proteolysis-targeting chimera molecules that selectively degrade the irak3 pseudokinase. Journal of Medicinal Chemistry, 63:10460-10473, Aug 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c01125, doi:10.1021/acs.jmedchem.0c01125. This article has 53 citations and is from a highest quality peer-reviewed journal.
2. (gurkan2024theirakmdeath pages 1-2): Berke Gürkan, Hessel Poelman, Liza Pereverzeva, Danielle Kruijswijk, Alex F. de Vos, Anouk G. Groenen, Edgar E. Nollet, Kanin Wichapong, Esther Lutgens, Tom van der Poll, Jiangfeng Du, W. Joost Wiersinga, Gerry A. F. Nicolaes, and Cornelis van ‘t Veer. The irak-m death domain: a tale of three surfaces. Frontiers in Molecular Biosciences, Jan 2024. URL: https://doi.org/10.3389/fmolb.2023.1265455, doi:10.3389/fmolb.2023.1265455. This article has 1 citations and is from a peer-reviewed journal.
3. (gurkan2024theirakmdeath pages 15-16): Berke Gürkan, Hessel Poelman, Liza Pereverzeva, Danielle Kruijswijk, Alex F. de Vos, Anouk G. Groenen, Edgar E. Nollet, Kanin Wichapong, Esther Lutgens, Tom van der Poll, Jiangfeng Du, W. Joost Wiersinga, Gerry A. F. Nicolaes, and Cornelis van ‘t Veer. The irak-m death domain: a tale of three surfaces. Frontiers in Molecular Biosciences, Jan 2024. URL: https://doi.org/10.3389/fmolb.2023.1265455, doi:10.3389/fmolb.2023.1265455. This article has 1 citations and is from a peer-reviewed journal.
4. (lange2021dimericstructureof pages 1-3): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
5. (lange2021dimericstructureof pages 10-11): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
6. (lange2021dimericstructureof pages 11-12): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
7. (lange2021dimericstructureof pages 3-4): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
8. (lange2021dimericstructureof pages 4-5): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
9. (lange2021dimericstructureof pages 6-7): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
10. (lange2021dimericstructureof pages 7-8): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
11. (lange2021dimericstructureof pages 9-10): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
12. (wang2025interleukin1receptorassociatedkinase3 pages 17-19): Jun Wang, Yulong Li, Chunyu Tan, Jinlian Shao, Weitai Tang, Quan Kong, Wenqianjun Sheng, Zhiquan Ding, Feng Li, Jifeng Piao, Dingyi Lv, Libin Hu, Qinghua Wang, and Xiaodan Jiang. Interleukin-1 receptor-associated kinase-3 aggravates neuroinflammatory injury after intracerebral hemorrhage via activation nf-κb/il-17a pathway in mice. Journal of Inflammation Research, Volume 18:1167-1189, Jan 2025. URL: https://doi.org/10.2147/jir.s494611, doi:10.2147/jir.s494611. This article has 0 citations and is from a peer-reviewed journal.
13. (wang2025interleukin1receptorassociatedkinase3 pages 19-21): Jun Wang, Yulong Li, Chunyu Tan, Jinlian Shao, Weitai Tang, Quan Kong, Wenqianjun Sheng, Zhiquan Ding, Feng Li, Jifeng Piao, Dingyi Lv, Libin Hu, Qinghua Wang, and Xiaodan Jiang. Interleukin-1 receptor-associated kinase-3 aggravates neuroinflammatory injury after intracerebral hemorrhage via activation nf-κb/il-17a pathway in mice. Journal of Inflammation Research, Volume 18:1167-1189, Jan 2025. URL: https://doi.org/10.2147/jir.s494611, doi:10.2147/jir.s494611. This article has 0 citations and is from a peer-reviewed journal.
14. (wang2025interleukin1receptorassociatedkinase3 pages 21-22): Jun Wang, Yulong Li, Chunyu Tan, Jinlian Shao, Weitai Tang, Quan Kong, Wenqianjun Sheng, Zhiquan Ding, Feng Li, Jifeng Piao, Dingyi Lv, Libin Hu, Qinghua Wang, and Xiaodan Jiang. Interleukin-1 receptor-associated kinase-3 aggravates neuroinflammatory injury after intracerebral hemorrhage via activation nf-κb/il-17a pathway in mice. Journal of Inflammation Research, Volume 18:1167-1189, Jan 2025. URL: https://doi.org/10.2147/jir.s494611, doi:10.2147/jir.s494611. This article has 0 citations and is from a peer-reviewed journal.
15. (zhou2024il1receptorassociatedkinase pages 1-2): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.
16. (zhou2024il1receptorassociatedkinase pages 17-19): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.
17. (lange2021dimericstructureof pages 12-13): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
18. (lange2021dimericstructureof pages 13-13): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
19. (lange2021dimericstructureof pages 13-14): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
20. (lange2021dimericstructureof pages 14-16): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
21. (lange2021dimericstructureof pages 16-17): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
22. (lange2021dimericstructureof pages 17-19): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
23. (lange2021dimericstructureof pages 5-6): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
24. (lange2021dimericstructureof pages 8-9): Sven M. Lange, Marina I. Nelen, Philip Cohen, and Yogesh Kulathu. Dimeric structure of the pseudokinase irak3 suggests an allosteric mechanism for negative regulation. Structure, 29:238-251.e4, Mar 2021. URL: https://doi.org/10.1016/j.str.2020.11.004, doi:10.1016/j.str.2020.11.004. This article has 33 citations and is from a domain leading peer-reviewed journal.
25. (zarrin2021kinaseinhibitionin pages 22-22): Ali A. Zarrin, Katherine Bao, Patrick Lupardus, and Domagoj Vucic. Kinase inhibition in autoimmunity and inflammation. Nature Reviews Drug Discovery, 20:39-63, Oct 2021. URL: https://doi.org/10.1038/s41573-020-0082-8, doi:10.1038/s41573-020-0082-8. This article has 384 citations and is from a highest quality peer-reviewed journal.
26. (zhou2024il1receptorassociatedkinase pages 12-13): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.
27. (zhou2024il1receptorassociatedkinase pages 16-17): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.
28. (zhou2024il1receptorassociatedkinase pages 22-23): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.
29. (zhou2024il1receptorassociatedkinase pages 11-12): Yang Zhou, Wei Rao, Zhao Li, Wei Guo, Fei Shao, Zhen Zhang, Hao Zhang, Tiejun Liu, Zitong Li, F. Tan, Qi Xue, Shugeng Gao, and Jie He. Il-1 receptor-associated kinase 3 (irak3) in lung adenocarcinoma predicts prognosis and immunotherapy resistance: involvement of multiple inflammation-related pathways. Translational Lung Cancer Research, 13:2139-2161, Jan 2024. URL: https://doi.org/10.21037/tlcr-24-391, doi:10.21037/tlcr-24-391. This article has 1 citations and is from a peer-reviewed journal.