## 1. Phylogeny

IRAK2 is a member of the interleukin‐1 receptor‐associated kinase (IRAK) family, a group of evolutionarily conserved proteins that play essential roles in innate immune signaling via the interleukin‐1 receptor (IL‐1R) and Toll‐like receptors (TLRs) (bahia2015interleukin1receptorassociated pages 3-4). Within this family, the four main members are IRAK1, IRAK2, IRAK3 (alternatively known as IRAK-M), and IRAK4. Phylogenetic analysis reveals that these kinases share a common ancestry with the Drosophila Pelle kinase, supporting the notion that an early eukaryotic ancestor already possessed a primitive version of the MyD88-dependent signaling machinery (su2020irakfamilyin pages 1-5). Although IRAK1 and IRAK4 exhibit robust catalytic activity, IRAK2 is often described as a pseudokinase because it lacks one or more conserved catalytic residues; however, its evolutionary conservation across mammals and its presence even in lower vertebrates emphasize its critical regulatory role within the immune system (mahmoud2023modulationofirak pages 4-6, rhyasen2015iraksignallingin pages 1-2). Orthologs of IRAK2 have been identified in a wide range of species, with expression noted in most tissues that orchestrate innate immune responses, further underscoring its importance in maintaining the integrity of inflammatory signaling pathways (OpenTargets Search: -IRAK2, bahia2015interleukin1receptorassociated pages 3-4).

## 2. Reaction Catalyzed

The canonical reaction catalyzed by protein kinases involves the transfer of a phosphate group from ATP to specific serine or threonine residues on substrate proteins, resulting in the formation of ADP and a phosphorylated substrate with the concomitant release of a proton (mahmoud2023modulationofirak pages 4-6). In the context of IRAK2, although it is frequently referred to as a pseudokinase, several studies indicate that under certain conditions it can mediate phosphorylation events that are critical for downstream signaling. For instance, experimental evidence has revealed that IRAK2 is capable of phosphorylating the RNA-binding protein SRSF1, a process that decreases SRSF1’s affinity for target mRNAs and thereby promotes the nuclear export and translation of mRNAs encoding inflammatory cytokines (zhou2017irak2directsstimulusdependent pages 16-19). This activity implicates IRAK2 in a non‐canonical phosphorylation mechanism that contributes to the regulation of mRNA stability and ultimately, cytokine production. The overall reaction may be summarized as follows:  
 ATP + Protein (serine/threonine residue) → ADP + Phosphorylated protein + H⁺.  
There is, however, some debate regarding the intrinsic catalytic efficiency of IRAK2 due to its pseudokinase classification in several reports; despite this, studies have demonstrated that alternative nucleotide-binding or residual catalytic mechanisms may allow IRAK2 to contribute to the signaling cascade initiated upon IL-1 receptor engagement (rhyasen2015iraksignallingin pages 2-3, chaudhary2015recentadvancesin pages 3-4).

## 3. Cofactor Requirements

Like most serine/threonine kinases, IRAK2 is presumed to require divalent metal ions as essential cofactors to facilitate its catalytic reaction, particularly the coordination of ATP’s phosphate groups (wang2017crystalstructureof pages 5-6). Although direct biochemical studies detailing the cofactor dependence of IRAK2 are limited, it is widely expected, by analogy with other members of the IRAK family and related kinases, that Mg²⁺ is required for its activity (patra2016recentprogressin pages 1-3). In a typical kinase reaction, Mg²⁺ acts to stabilize the negative charges on the phosphate groups of ATP and assists in orienting the ATP molecule within the active site, thereby preparing it for the nucleophilic attack by the substrate’s hydroxyl group. This Mg²⁺ dependence is a well-established characteristic shared among serine/threonine kinases and likely extends to IRAK2 even if its catalytic efficiency is reduced in comparison with fully active kinases (zarrin2021kinaseinhibitionin pages 7-8).

## 4. Substrate Specificity

The substrate specificity of IRAK2 remains less well defined than that of its catalytically active counterparts, IRAK1 and IRAK4, yet several studies have begun to elucidate its role in downstream signal propagation (chaudhary2015recentadvancesin pages 3-4). One of the best‐characterized substrates for IRAK2 is the splicing factor SRSF1, whose phosphorylation by IRAK2 decreases its RNA-binding capacity, thereby facilitating the export of inflammatory mRNAs from the nucleus and promoting cytokine translation (mahmoud2023modulationofirak pages 4-6, zhou2017irak2directsstimulusdependent pages 16-19). In addition to SRSF1, IRAK2’s participation within the Myddosome complex suggests that its interactions may be oriented towards scaffolding functions and the recruitment of downstream effectors such as TRAF6 rather than the phosphorylation of canonical protein substrates in isolation (rhyasen2015iraksignallingin pages 5-6, su2020irakfamilyin pages 1-5). At present, no well‐defined consensus phosphorylation motif exclusive to IRAK2 has been established; however, given its structural resemblance to other serine/threonine kinases, it is possible that its substrate specificity may depend on less canonical motifs that integrate features of tertiary protein structure or protein–protein interaction domains. Comparative studies with kinases like S6K, which recognize an RxRxxp[ST] motif, have not demonstrated an equivalent signature sequence for IRAK2, further supporting the notion that IRAK2’s role may be predominantly regulatory rather than that of a classical enzyme with strict substrate sequence requirements.

## 5. Structure

The structural organization of IRAK2 mirrors that of other IRAK family members, comprising several conserved domains interspersed by flexible linker regions. Its primary structure begins with an N-terminal death domain (DD), which is critical for mediating homotypic interactions with adaptor proteins such as MyD88 to initiate the formation of the Myddosome complex (mahmoud2023modulationofirak pages 4-6, bahia2015interleukin1receptorassociated pages 3-4). Adjacent to the death domain is a proline/serine/threonine-rich (ProST) domain, which is thought to act as an intrinsically disordered regulatory region that may be subject to extensive post-translational modifications such as phosphorylation and ubiquitination. These modifications likely influence IRAK2’s conformation and interaction with other proteins in the signaling cascade.  
Following the ProST region is the C-terminal portion of IRAK2, which contains a kinase or pseudokinase domain and additional motifs necessary for binding downstream signaling molecules such as TRAF6. Although high-resolution crystal structures of IRAK2 have not been elucidated, homology modeling and predictive analyses using resources like AlphaFold suggest that its kinase-like domain adopts the classical two-lobed structure typical of serine/threonine kinases, including conserved features such as a glycine-rich loop (G-loop) responsible for ATP binding, a catalytic loop that usually contains an HRD (or HGD) motif, and a DFG motif that coordinates Mg²⁺ ions (wang2017crystalstructureof pages 5-6, patra2016recentprogressin pages 1-3). However, unlike IRAK1 or IRAK4, IRAK2 is frequently designated as a pseudokinase because it lacks certain catalytic residues that are essential for full kinase activity; yet, studies have identified mutations such as D431E that seem to enhance its ability to activate nuclear factor kappa-B (NF-κB), suggesting that even subtle alterations in these residues can modulate residual catalytic or structural functions (mahmoud2023modulationofirak pages 4-6, lange2021dimericstructureof pages 4-5). In addition, the C-terminal region contains TRAF6-binding motifs which are indispensable for recruiting the E3 ubiquitin ligase TRAF6 and propagating downstream signaling cascades. Overall, while IRAK2 lacks the complete set of features required for robust catalytic activity, its structure is optimized for facilitating critical protein–protein interactions that drive the inflammatory response (jacobsen2017thesecretlife pages 13-14).

## 6. Regulation

The regulation of IRAK2 is multifaceted and is primarily achieved through its integration into the Myddosome complex and dynamic post-translational modifications. Following the engagement of the IL-1 receptor by its ligand, IRAK2 is rapidly recruited to the receptor complex via its death domain, where it assembles with MyD88 and IRAK4 to form a supramolecular complex that orchestrates downstream signaling (mahmoud2023modulationofirak pages 4-6, pereira2023regulationofinnate pages 1-2). One key regulatory mechanism involves phosphorylation events. Although IRAK2 is classified as a pseudokinase by several accounts, evidence suggests that it can phosphorylate downstream targets—such as the splicing factor SRSF1—under specific conditions, thereby modulating the nuclear export of inflammatory mRNAs (zhou2017irak2directsstimulusdependent pages 16-19). These phosphorylation events are likely subject to both auto‐phosphorylation and trans-phosphorylation dynamics that may be mediated by IRAK4 or potentially by alternative, non‐canonical catalytic mechanisms intrinsic to IRAK2 itself (rhyasen2015iraksignallingin pages 2-3, chaudhary2015recentadvancesin pages 3-4).  
Post-translational modifications beyond phosphorylation also regulate IRAK2 function. For instance, ubiquitination events are thought to modulate IRAK2 stability and its interactions within the Myddosome, whereas sumoylation has been implicated in controlling its subcellular localization. RanBP2-mediated sumoylation of IRAK2 has been reported as a prerequisite for nuclear translocation in response to lipopolysaccharide (LPS) stimulation, suggesting that this modification may serve as a regulatory switch that enables IRAK2 to influence post-transcriptional mRNA processing (srikanth2024irak4autophosphorylationcontrols pages 48-51). Moreover, the D431E mutation in IRAK2 has been linked to an enhanced NF-κB signaling output and increased proinflammatory cytokine production during viral challenge, indicating that even single-residue alterations can have significant impacts on its regulatory dynamics (mahmoud2023modulationofirak pages 4-6, teocchi2024dysregulationoftlr pages 9-10). Thus, regulation of IRAK2 occurs at both the levels of its inclusion in multi-protein signaling platforms and through reversible post-translational modifications that govern its activity, stability, and subcellular localization.

## 7. Function

IRAK2 serves as a critical mediator of innate immune signaling downstream of IL-1 receptors and TLRs, playing an indispensable role in initiating and sustaining inflammatory responses. Upon engagement of the IL-1 type I receptor by interleukin-1, IRAK2 is recruited to the receptor complex through interactions mediated by its N-terminal death domain, where it cooperates with MyD88 and other IRAK family members to form the Myddosome, a high-order oligomeric signaling platform (mahmoud2023modulationofirak pages 4-6, pereira2023regulationofinnate pages 1-2).  
A key function of IRAK2 is the post-transcriptional regulation of inflammatory mediators. Through phosphorylation of specific targets such as SRSF1, IRAK2 decreases the RNA-binding affinity of these proteins, thus facilitating the nuclear export of mRNAs encoding proinflammatory cytokines and chemokines. This mechanism ensures that the mRNAs are available for translation in the cytoplasm, thereby amplifying the production of cytokines like tumor necrosis factor (TNF) and interleukin-6 (IL-6) that are vital for mounting an effective immune response (mahmoud2023modulationofirak pages 4-6, zhou2017irak2directsstimulusdependent pages 16-19).  
In addition, IRAK2 appears to contribute to the regulation of NF-κB and mitogen-activated protein kinase (MAPK) signaling pathways. Although its kinase activity is lower than that of IRAK1 or IRAK4, IRAK2’s presence in the Myddosome is critical for sustaining late-phase NF-κB activation once the initial signals have been propagated, particularly after rapid degradation of IRAK1 (rhyasen2015iraksignallingin pages 2-3, pereira2023regulationofinnate pages 8-9).  
IRAK2 is widely expressed in immune cells such as macrophages, dendritic cells, and lymphocytes, and its activity is integral to effective cytokine production during pathogen challenge. Through its contributions to the assembly and stabilization of the Myddosome, IRAK2 ensures that inflammatory signaling is appropriately modulated, allowing for both robust acute responses and tighter regulation to prevent overactivation that may lead to autoimmune pathologies (su2020irakfamilyin pages 1-5, teocchi2024dysregulationoftlr pages 9-10).  
The functional significance of IRAK2 is underscored by genetic studies linking specific polymorphisms in IRAK2 with increased susceptibility to inflammatory diseases such as rheumatoid arthritis and possibly colorectal cancer, further emphasizing its role as a key regulator in inflammatory signaling networks (bahia2015interleukin1receptorassociated pages 3-4).

## 8. Other Comments

While therapeutic development in the IRAK signaling pathway has primarily focused on active kinases such as IRAK4—and to a lesser extent, IRAK1—IRAK2 remains an attractive but less explored target for modulating immune responses. Current research indicates that selective inhibitors directly targeting IRAK2 are scarce; instead, small molecules that disrupt the formation or stability of the Myddosome complex may indirectly alter IRAK2-mediated signaling (wiese2020investigationalirak4inhibitors pages 17-21, xie2021smallmoleculekinaseinhibitors pages 24-25).  
Notable genetic variants, including the D431E substitution in IRAK2, have been shown to enhance NF-κB activation and lead to an increased production of proinflammatory cytokines, implicating IRAK2 mutations as a potential contributor to the pathogenesis of viral infections and chronic inflammatory conditions (mahmoud2023modulationofirak pages 4-6, teocchi2024dysregulationoftlr pages 9-10).  
Recent studies have also focused on the role of IRAK2 in post-transcriptional mRNA regulation, where its ability to modulate the phosphorylation state of RNA-binding proteins like SRSF1 provides a novel mechanism for controlling cytokine mRNA stability and export; this area of research remains active and may reveal further therapeutic opportunities for diseases characterized by cytokine storms, such as severe SARS-CoV-2 infections (zhou2017irak2directsstimulusdependent pages 16-19, singer2018inhibitionofinterleukin1 pages 18-19).  
Additionally, ongoing work is elucidating the interplay between IRAK2 and other metabolic or signaling regulators within immune cells, shedding light on potential cross-talk mechanisms that could be exploited to fine-tune inflammatory responses. Such insights will be essential for the future design of targeted therapies that aim to modulate IRAK2 function in a range of autoimmune, inflammatory, and potentially neoplastic conditions (pereira2023regulationofinnate pages 7-8, su2020irakfamilyin pages 26-29, bennett2022irak1andirak4 pages 1-2).

## 9. References

1. mahmoud2023modulationofirak pages 4-6
2. OpenTargets Search: -IRAK2
3. bahia2015interleukin1receptorassociated pages 3-4
4. su2020irakfamilyin pages 1-5
5. rhyasen2015iraksignallingin pages 1-2
6. zhou2017irak2directsstimulusdependent pages 16-19
7. wang2017crystalstructureof pages 5-6
8. patra2016recentprogressin pages 1-3
9. zarrin2021kinaseinhibitionin pages 7-8
10. chaudhary2015recentadvancesin pages 3-4
11. lange2021dimericstructureof pages 4-5
12. pereira2023regulationofinnate pages 1-2
13. pereira2023regulationofinnate pages 7-8
14. pereira2023regulationofinnate pages 8-9
15. srikanth2024irak4autophosphorylationcontrols pages 48-51
16. teocchi2024dysregulationoftlr pages 9-10
17. wiese2020investigationalirak4inhibitors pages 17-21
18. xie2021smallmoleculekinaseinhibitors pages 24-25
19. bennett2022irak1andirak4 pages 1-2
20. jacobsen2017thesecretlife pages 13-14

References

1. (mahmoud2023modulationofirak pages 4-6): Ismail Sami Mahmoud, Yazun Bashir Jarrar, and Febrimarsa. Modulation of irak enzymes as a therapeutic strategy against sars-cov-2 induced cytokine storm. Clinical and Experimental Medicine, 23:2909-2923, Apr 2023. URL: https://doi.org/10.1007/s10238-023-01064-7, doi:10.1007/s10238-023-01064-7. This article has 0 citations and is from a peer-reviewed journal.
2. (OpenTargets Search: -IRAK2): Open Targets Query (-IRAK2, 4 results). Ochoa, D. et al. (2023). The next-generation Open Targets Platform: reimagined, redesigned, rebuilt. Nucleic Acids Research.
3. (bahia2015interleukin1receptorassociated pages 3-4): Malkeet Singh Bahia, Maninder Kaur, Pragati Silakari, and Om Silakari. Interleukin-1 receptor associated kinase inhibitors: potential therapeutic agents for inflammatory- and immune-related disorders. Cellular Signalling, 27:1039-1055, Jun 2015. URL: https://doi.org/10.1016/j.cellsig.2015.02.025, doi:10.1016/j.cellsig.2015.02.025. This article has 57 citations and is from a peer-reviewed journal.
4. (chaudhary2015recentadvancesin pages 3-4): Divya Chaudhary, Shaughnessy Robinson, and Donna L. Romero. Recent advances in the discovery of small molecule inhibitors of interleukin-1 receptor-associated kinase 4 (irak4) as a therapeutic target for inflammation and oncology disorders. Journal of medicinal chemistry, 58 1:96-110, Jan 2015. URL: https://doi.org/10.1021/jm5016044, doi:10.1021/jm5016044. This article has 114 citations and is from a highest quality peer-reviewed journal.
5. (jacobsen2017thesecretlife pages 13-14): Annette V. Jacobsen and James M. Murphy. The secret life of kinases: insights into non-catalytic signalling functions from pseudokinases. Biochemical Society Transactions, 45:665-681, Jun 2017. URL: https://doi.org/10.1042/bst20160331, doi:10.1042/bst20160331. This article has 79 citations and is from a peer-reviewed journal.
6. (pereira2023regulationofinnate pages 1-2): Milton Pereira and Ricardo T. Gazzinelli. Regulation of innate immune signaling by irak proteins. Frontiers in Immunology, Feb 2023. URL: https://doi.org/10.3389/fimmu.2023.1133354, doi:10.3389/fimmu.2023.1133354. This article has 54 citations and is from a peer-reviewed journal.
7. (pereira2023regulationofinnate pages 7-8): Milton Pereira and Ricardo T. Gazzinelli. Regulation of innate immune signaling by irak proteins. Frontiers in Immunology, Feb 2023. URL: https://doi.org/10.3389/fimmu.2023.1133354, doi:10.3389/fimmu.2023.1133354. This article has 54 citations and is from a peer-reviewed journal.
8. (pereira2023regulationofinnate pages 8-9): Milton Pereira and Ricardo T. Gazzinelli. Regulation of innate immune signaling by irak proteins. Frontiers in Immunology, Feb 2023. URL: https://doi.org/10.3389/fimmu.2023.1133354, doi:10.3389/fimmu.2023.1133354. This article has 54 citations and is from a peer-reviewed journal.
9. (rhyasen2015iraksignallingin pages 1-2): Garrett W. Rhyasen, Garrett W. Rhyasen, Garrett W. Rhyasen, D. Starczynowski, and D. Starczynowski. Irak signalling in cancer. British Journal of Cancer, 112:232-237, Oct 2015. URL: https://doi.org/10.1038/bjc.2014.513, doi:10.1038/bjc.2014.513. This article has 199 citations and is from a domain leading peer-reviewed journal.
10. (rhyasen2015iraksignallingin pages 2-3): Garrett W. Rhyasen, Garrett W. Rhyasen, Garrett W. Rhyasen, D. Starczynowski, and D. Starczynowski. Irak signalling in cancer. British Journal of Cancer, 112:232-237, Oct 2015. URL: https://doi.org/10.1038/bjc.2014.513, doi:10.1038/bjc.2014.513. This article has 199 citations and is from a domain leading peer-reviewed journal.
11. (rhyasen2015iraksignallingin pages 5-6): Garrett W. Rhyasen, Garrett W. Rhyasen, Garrett W. Rhyasen, D. Starczynowski, and D. Starczynowski. Irak signalling in cancer. British Journal of Cancer, 112:232-237, Oct 2015. URL: https://doi.org/10.1038/bjc.2014.513, doi:10.1038/bjc.2014.513. This article has 199 citations and is from a domain leading peer-reviewed journal.
12. (su2020irakfamilyin pages 1-5): Lin-Chong Su, Wang-Dong Xu, and An-Fang Huang. Irak family in inflammatory autoimmune diseases. Autoimmunity Reviews, 19:102461, Mar 2020. URL: https://doi.org/10.1016/j.autrev.2020.102461, doi:10.1016/j.autrev.2020.102461. This article has 87 citations and is from a peer-reviewed journal.
13. (wiese2020investigationalirak4inhibitors pages 17-21): Michael D. Wiese, Arkady T. Manning-Bennett, and Ahmad Y. Abuhelwa. Investigational irak-4 inhibitors for the treatment of rheumatoid arthritis. Expert Opinion on Investigational Drugs, 29:475-482, Apr 2020. URL: https://doi.org/10.1080/13543784.2020.1752660, doi:10.1080/13543784.2020.1752660. This article has 50 citations and is from a peer-reviewed journal.
14. (zarrin2021kinaseinhibitionin pages 7-8): 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.
15. (zhou2017irak2directsstimulusdependent pages 16-19): Hao Zhou, Katarzyna Bulek, Xiao Li, Tomasz Herjan, Minjia Yu, Wen Qian, Han Wang, Gao Zhou, Xing Chen, Hui Yang, Lingzi Hong, Junjie Zhao, Luke Qin, Koichi Fukuda, Annette Flotho, Ji Gao, Ashok Dongre, Julie A Carman, Zizhen Kang, Bing Su, Timothy S Kern, Jonathan D Smith, Thomas A Hamilton, Frauke Melchior, Paul L Fox, and Xiaoxia Li. Irak2 directs stimulus-dependent nuclear export of inflammatory mrnas. eLife, Oct 2017. URL: https://doi.org/10.7554/elife.29630, doi:10.7554/elife.29630. This article has 39 citations and is from a domain leading peer-reviewed journal.
16. (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.
17. (srikanth2024irak4autophosphorylationcontrols pages 48-51): Niranjan Srikanth, Rafael Deliz-Aguirre, Deepika Kumari Gola, Margaux Bilay, Elke Ziska, and Marcus J. Taylor. Irak4 autophosphorylation controls inflammatory signaling by activating irak oligomerization. bioRxiv, Feb 2024. URL: https://doi.org/10.1101/2023.12.21.572799, doi:10.1101/2023.12.21.572799. This article has 1 citations.
18. (wang2017crystalstructureof pages 5-6): Li Wang, Qi Qiao, Ryan Ferrao, Chen Shen, John M. Hatcher, Sara J. Buhrlage, Nathanael S. Gray, and Hao Wu. Crystal structure of human irak1. Proceedings of the National Academy of Sciences, 114:13507-13512, Dec 2017. URL: https://doi.org/10.1073/pnas.1714386114, doi:10.1073/pnas.1714386114. This article has 80 citations.
19. (bennett2022irak1andirak4 pages 1-2): Joshua Bennett and Daniel T. Starczynowski. Irak1 and irak4 as emerging therapeutic targets in hematologic malignancies. Current Opinion in Hematology, 29:8-19, Nov 2022. URL: https://doi.org/10.1097/moh.0000000000000693, doi:10.1097/moh.0000000000000693. This article has 77 citations and is from a peer-reviewed journal.
20. (patra2016recentprogressin pages 1-3): Mahesh Patra and Sangdun Choi. Recent progress in the molecular recognition and therapeutic importance of interleukin-1 receptor-associated kinase 4. Molecules, 21:1529, Nov 2016. URL: https://doi.org/10.3390/molecules21111529, doi:10.3390/molecules21111529. This article has 44 citations and is from a peer-reviewed journal.
21. (singer2018inhibitionofinterleukin1 pages 18-19): Jack W. Singer, Angela Fleischman, Suliman Al-Fayoumi, John O. Mascarenhas, Qiang Yu, and Anupriya Agarwal. Inhibition of interleukin-1 receptor-associated kinase 1 (irak1) as a therapeutic strategy. Oncotarget, 9:33416-33439, Sep 2018. URL: https://doi.org/10.18632/oncotarget.26058, doi:10.18632/oncotarget.26058. This article has 146 citations and is from a poor quality or predatory journal.
22. (su2020irakfamilyin pages 26-29): Lin-Chong Su, Wang-Dong Xu, and An-Fang Huang. Irak family in inflammatory autoimmune diseases. Autoimmunity Reviews, 19:102461, Mar 2020. URL: https://doi.org/10.1016/j.autrev.2020.102461, doi:10.1016/j.autrev.2020.102461. This article has 87 citations and is from a peer-reviewed journal.
23. (teocchi2024dysregulationoftlr pages 9-10): Marcelo Teocchi, Thaís de Andrade Eugênio, Lia Furlaneto Marega, Isabella Quinti, and Maria Marluce dos Santos Vilela. Dysregulation of tlr signaling-associated gene expression in x-linked agammaglobulinemia: implications for correlations genotype-phenotype and disease expression. Journal of Innate Immunity, 16:425-439, Aug 2024. URL: https://doi.org/10.1159/000540082, doi:10.1159/000540082. This article has 0 citations and is from a peer-reviewed journal.
24. (xie2021smallmoleculekinaseinhibitors pages 24-25): Zhouling Xie, Xiaoxiao Yang, Yajun Duan, Jihong Han, and Chenzhong Liao. Small-molecule kinase inhibitors for the treatment of nononcologic diseases. Journal of Medicinal Chemistry, 64:1283-1345, Jan 2021. URL: https://doi.org/10.1021/acs.jmedchem.0c01511, doi:10.1021/acs.jmedchem.0c01511. This article has 82 citations and is from a highest quality peer-reviewed journal.