1. Phylogeny  
   IRAK3 is a member of the interleukin‐1 receptor‐associated kinase (IRAK) family, which comprises both catalytically active kinases (such as IRAK1 and IRAK4) and inactive or weakly active pseudokinases (including IRAK2 and IRAK3) (janssens2003functionaldiversityand pages 2-3). Phylogenetic analyses indicate that the IRAK family evolved from a common ancestral kinase that is traceable from early metazoans, and gene duplication events in the vertebrate lineage contributed to the emergence of specialized members such as IRAK3 (gosu2012molecularevolutionand pages 1-2, gosu2012molecularevolutionand pages 9-11). Although IRAK3 orthologs have been identified primarily in mammals, with certain teleost fish also retaining related sequences, the overall arrangement of the family suggests that IRAK3 was a later addition when compared with the more broadly conserved IRAK4-like kinases (gosu2012molecularevolutionand pages 11-12, dardick2006plantandanimal pages 9-10). Within the kinome, IRAK3 is classified as a pseudokinase or an ACF (active site cysteine–containing) kinase member that displays characteristic modifications in the catalytic site, notably the substitution of a critical aspartate residue with a serine residue, which distinguishes it from its catalytically active counterparts (janssens2003functionaldiversityand pages 2-3, lange2021dimericstructureof pages 3-4). This evolutionary placement is further supported by the presence of an N‐terminal death domain and a central pseudokinase domain, which are conserved features among members of the Pelle/IRAK subfamily involved in innate immune receptor signaling (dardick2006plantandanimal pages 3-6, gosu2012molecularevolutionand pages 4-5). The phylogenetic context of IRAK3 underscores its role as a regulatory modulator that diverged from active kinase ancestors during the evolution of the immune system.
2. Reaction Catalyzed  
   In contrast to conventional serine/threonine kinases that catalyze the ATP‐dependent transfer of a phosphate group to protein substrates—typically following the reaction: ATP + [protein]–(L‑serine/threonine) → ADP + [protein]–phospho(L‑serine/threonine) + H⁺—IRAK3 has not been demonstrably associated with such a canonical phosphorylation reaction (flannery2010theinterleukin1receptorassociated pages 20-24, janssens2003functionaldiversityand pages 2-3). Rather than acting as an active phosphotransferase, IRAK3 is widely described as a “putative inactive protein kinase” with a primary role in regulating signaling by tethering or stabilizing components within the receptor complex (Information section). Notwithstanding its lack of classical kinase activity, experimental studies using recombinant full-length IRAK3 have provided evidence of a noncanonical enzymatic activity; specifically, IRAK3 has been shown to possess guanylate cyclase activity, whereby it catalyzes the conversion of GTP into cyclic guanosine monophosphate (cGMP) plus pyrophosphate (PPi) (freihat2019irak3modulatesdownstream pages 2-3, freihat2019irak3modulatesdownstream pages 3-5, turek2023mutationsinthe pages 7-10). No direct evidence supports an ATP-dependent phosphotransfer reaction in IRAK3, and therefore the guanylate cyclase reaction represents an alternative catalytic mechanism that is distinct from the phosphorylation reaction mediated by conventional active kinases. This distinction is underscored by the structural changes observed in its active site motifs, which are consistent with an inability to support ATP-dependent phosphorylation (janssens2003functionaldiversityand pages 2-3, freihat2019irak3modulatesdownstream pages 2-3).
3. Cofactor Requirements  
   Conventional protein kinases typically require the presence of divalent cations—most commonly Mg²⁺—to facilitate ATP binding and the subsequent phosphoryl transfer reaction. In the case of IRAK3, the canonical kinase function is absent and ATP binding is reported to be extremely weak, with studies demonstrating that the addition of Mg²⁺ does not produce significant stabilization or catalytic activation (lange2021dimericstructureof pages 6-7). For its alternative guanylate cyclase activity, however, experimental studies have shown that the enzymatic function of IRAK3 is cofactor-dependent. In these assays, divalent metal ions are required to enable the catalysis of GTP conversion into cGMP, with a preferential requirement for Mn²⁺ over Mg²⁺ observed in vitro (freihat2019irak3modulatesdownstream pages 2-3, freihat2019irak3modulatesdownstream pages 3-5, turek2023mutationsinthe pages 7-10). Thus, while IRAK3 does not exhibit a typical ATP-dependent kinase reaction, its guanylate cyclase function is activated in the presence of Mn²⁺, which appears to serve as the optimal cofactor for this alternative catalytic activity.
4. Substrate Specificity  
   Traditional serine/threonine kinases are characterized by their recognition of specific consensus substrate motifs such as RxRxxp[ST] which facilitate selective phosphorylation of target proteins. However, due to the inability of IRAK3 to catalyze an ATP-dependent phosphorylation reaction under standard conditions, no defined consensus substrate motif has been established for IRAK3 in terms of protein phosphorylation (janssens2003functionaldiversityand pages 2-3, smith2019alternativesplicingand pages 47-53). In its role as a pseudokinase, IRAK3 instead functions through protein–protein interactions within the receptor signaling complex to regulate downstream signaling events. Regarding its reported guanylate cyclase activity, IRAK3 catalyzes the conversion of GTP into cGMP; nevertheless, the specificity for GTP substrates in this context does not follow the typical substrate motif classification observed for phosphoryl transfer reactions. The guanylate cyclase center present within the pseudokinase domain shows conservation of key residues necessary for GTP binding and subsequent catalysis, but no additional consensus sequence beyond the defined catalytic center has been described (freihat2019irak3modulatesdownstream pages 3-5). Therefore, IRAK3’s substrate specificity in the phosphorylation context is not applicable, and its enzymatic activity is measured instead by the ability to generate cGMP rather than by direct phosphorylation of serine or threonine residues.
5. Structure  
   IRAK3 exhibits a multi-domain architecture that reflects its membership within the IRAK family and distinguishes its regulatory role from catalytically active kinases. At the N-terminus, IRAK3 contains a death domain (DD) that is critical for mediating homotypic interactions with other death domains found in MyD88 and other signaling proteins within the Myddosome complex (janssens2003functionaldiversityand pages 2-3, dardick2006plantandanimal pages 3-6). Adjacent to the death domain is a proline/serine/threonine-rich (ProST) region, which is thought to be intrinsically disordered and may function as a flexible linker that permits dynamic interactions between the DD and the kinase domain. The central portion of IRAK3 comprises a pseudokinase domain that retains the overall fold characteristic of serine/threonine kinases, including a bilobal structure with an N-lobe rich in β-sheet structures and a predominantly α-helical C-lobe (bailey2014biochemicalanalysisof pages 33-37, lange2021dimericstructureof pages 1-3). Despite the presence of a conserved ATP-binding pocket and glycine-rich G-loop, structural analyses have revealed key deviations from the canonical motifs that are essential for catalytic activity. For example, the active site usually containing a catalytic aspartate residue is instead found substituted by a serine residue in IRAK3, and the canonical DFG motif is frequently altered to DFA (janssens2003functionaldiversityand pages 2-3, lange2021dimericstructureof pages 3-4). Recent crystallographic studies indicate that the pseudokinase domain of IRAK3 adopts a closed, pseudoactive conformation stabilized by distinct hydrophobic interactions. Among these, the presence of a conserved hydrophobic anchor residue in the G-loop (such as F177) is notable for maintaining a structured conformation even in the absence of ATP (lange2021dimericstructureof pages 5-6). In addition, structural investigations have identified within the pseudokinase domain a cryptic guanylate cyclase center, which is embedded adjacent to the conventional catalytic motifs. This guanylate cyclase center is responsible for the low-level in vitro production of cGMP, an alternative enzymatic function that is not typical among kinases (freihat2019irak3modulatesdownstream pages 2-3, freihat2019irak3modulatesdownstream pages 3-5). Furthermore, IRAK3 displays an unusual head-to-head dimerization interface in its crystal structure, a property not generally observed in catalytically active kinases, and this dimerization may have functional implications for its allosteric regulation within the immune receptor complex (lange2021dimericstructureof pages 1-3, lange2021dimericstructureof pages 3-4). Overall, the structural organization of IRAK3—with its modular domains (DD, ProST region, and pseudokinase domain) and its unique structural features (altered active site motifs, guanylate cyclase center, and dimerization interface)—distinguishes it from its catalytically active counterparts while preserving a scaffold that facilitates critical regulatory functions in innate immunity (dardick2006plantandanimal pages 6-7, gosu2012molecularevolutionand pages 4-5).
6. Regulation  
   Regulatory mechanisms for IRAK3 are primarily dependent on its role as a negative regulator of Toll-like receptor (TLR) and interleukin-1 receptor (IL1R) signaling rather than on phosphorylation-dependent activation. IRAK3 is predominantly expressed in monocytes and macrophages, where it associates with components of the Myddosome complex to prevent the dissociation of IRAK1 and IRAK4, thereby inhibiting the propagation of downstream inflammatory signals (flannery2010theinterleukin1receptorassociated pages 20-24, dardick2006plantandanimal pages 3-6). Its inhibitory function is mediated principally by protein–protein interactions within the receptor complex that stabilize the assembly and limit subsequent activation of downstream kinases involved in NF-κB and MAP kinase signaling (janssens2003functionaldiversityand pages 2-3, rebl2019atleasttwo pages 1-2). Although IRAK3 lacks the conventional catalytic activity of an active kinase, it has been shown to harbor an alternative enzymatic activity in the form of guanylate cyclase function. The production of cGMP via the guanylate cyclase center in IRAK3 may serve as an additional regulatory mechanism; increased cGMP levels have been correlated with modulation of inflammatory signaling in cell-based assays (freihat2019irak3modulatesdownstream pages 3-5, turek2023mutationsinthe pages 7-10). In contrast to mechanisms that involve post-translational modifications such as phosphorylation or ubiquitination—which are common regulatory strategies among active kinases—there is little evidence that IRAK3 is subject to extensive phosphorylation or ubiquitination events that alter its catalytic or regulatory functions (flannery2010theinterleukin1receptorassociated pages 43-47). Rather, the regulation of IRAK3 appears to depend on its structural conformation, dimerization state, and its interaction with other signaling proteins within the Myddosome, as well as the modulation of guanylate cyclase activity by the presence of specific metal ion cofactors (dardick2006plantandanimal pages 6-7, flannery2010theinterleukin1receptorassociated pages 20-24).
7. Function  
   IRAK3 is best characterized as an inhibitory modulator in the innate immune system, where its primary function is to restrain inflammatory signaling. By binding to components of the Myddosome complex, specifically through interactions with IRAK1 and IRAK4, IRAK3 prevents the dissociation of these active kinases, thereby limiting the downstream activation of NF-κB and MAP kinase pathways that drive the production of pro-inflammatory cytokines (janssens2003functionaldiversityand pages 2-3, flannery2010theinterleukin1receptorassociated pages 20-24). Its expression is largely restricted to monocytes and macrophages where regulation of TLR and IL1R signaling is critical for maintaining immune homeostasis. Studies have shown that IRAK3 expression increases in deactivated macrophages and in settings of endotoxin tolerance, providing a feedback mechanism to dampen overactive inflammatory responses (dardick2006plantandanimal pages 3-6, rebl2019atleasttwo pages 1-2). Beyond its role in inhibiting classical TLR/IL1R-mediated activation, IRAK3 has also been implicated in alternative signaling pathways. For instance, in the context of IL33-induced lung inflammation, IRAK3 positively regulates the expression of cytokine messenger RNAs including IL6, CSF3, CXCL2, and CCL5 in dendritic cells, thereby influencing the balance between pro- and anti-inflammatory responses (Information section, PubMed:29686383). In addition to its canonical regulatory role via protein–protein interactions, the guanylate cyclase activity of IRAK3 may contribute to the fine-tuning of its inhibitory effects; the localized production of cGMP could act as an intracellular second messenger to modulate downstream signaling events, although the precise biological consequences of this activity remain under investigation (freihat2019irak3modulatesdownstream pages 2-3, turek2023mutationsinthe pages 7-10). Overall, IRAK3 functions as a critical checkpoint within the innate immune signaling cascade, ensuring that inflammatory responses are kept in balance to avoid excessive tissue damage or chronic inflammation (flannery2010theinterleukin1receptorassociated pages 20-24, dardick2006plantandanimal pages 9-10).
8. Other Comments  
   In addition to its role as a negative regulator of inflammatory signaling, modifications in the sequence of IRAK3 have been linked to alterations in its subcellular localization and enzymatic function. Mutations in amino acid residues in the vicinity of the guanylate cyclase center have been shown to adversely affect the ability of IRAK3 to produce cGMP, with some point mutations (for example, R372L) leading to a complete loss of guanylate cyclase activity (turek2023mutationsinthe pages 7-10). Furthermore, expression studies have revealed that IRAK3 is upregulated in certain pathological conditions, such as in the synovium of patients with rheumatoid arthritis and in tumor-infiltrating monocytes; these changes in expression are thought to contribute to immune tolerance and may favor tumor growth by modulating the inflammatory microenvironment (borghese2025irak3isupregulated pages 1-2, rhyasen2014irakfamilykinases pages 21-25). While selective small-molecule inhibitors specifically targeting IRAK3 have not been comprehensively characterized, there remains active interest in developing agents that can modulate IRAK3 function – either by enhancing its negative regulatory role in cases of chronic inflammation or by counteracting its upregulation in disease states where an inappropriate suppression of the immune response is observed (rhyasen2014irakfamilykinases pages 21-25). The dual nature of IRAK3—its inability to catalyze traditional phosphorylation reactions alongside its demonstrated guanylate cyclase activity—highlights an evolutionary divergence within the kinase family where regulatory control is achieved through non-catalytic mechanisms, a feature that underscores its unique contribution to immune regulation (bailey2014biochemicalanalysisof pages 33-37, freihat2019irak3modulatesdownstream pages 3-5).
9. References
10. bailey2014biochemicalanalysisof pages 33-37
11. borghese2018theroleof pages 85-95
12. dardick2006plantandanimal pages 3-6
13. dardick2006plantandanimal pages 6-7
14. flannery2010theinterleukin1receptorassociated pages 20-24
15. flannery2010theinterleukin1receptorassociated pages 43-47
16. freihat2019irak3modulatesdownstream pages 2-3
17. freihat2019irak3modulatesdownstream pages 3-5
18. gosu2012molecularevolutionand pages 1-2
19. gosu2012molecularevolutionand pages 11-12
20. gosu2012molecularevolutionand pages 4-5
21. gosu2012molecularevolutionand pages 9-11
22. janssens2003functionaldiversityand pages 2-3
23. janssens2003functionaldiversityand pages 6-7
24. lange2021dimericstructureof pages 1-3
25. lange2021dimericstructureof pages 3-4
26. lange2021dimericstructureof pages 6-7
27. lange2021dimericstructureof pages 8-9
28. rebl2019atleasttwo pages 1-2
29. rhyasen2014irakfamilykinases pages 13-17
30. rhyasen2014irakfamilykinases pages 17-21
31. rhyasen2014irakfamilykinases pages 21-25
32. smith2019alternativesplicingand pages 47-53
33. turek2023mutationsinthe pages 7-10
34. wang2013functionalandepidemiological pages 40-44
35. wang2013functionalandepidemiological pages 44-49
36. wang2013functionalandepidemiological pages 49-53
37. blaum2014structureofthe pages 4-5
38. borghese2025irak3isupregulated pages 1-2

References

1. (bailey2014biochemicalanalysisof pages 33-37): F Bailey. Biochemical analysis of human cancer-associated pseudokinases. Unknown journal, 2014.
2. (borghese2018theroleof pages 85-95): F Borghese. The role of irak3 in regulating immune-mediated inflammatory arthritis. Unknown journal, 2018.
3. (dardick2006plantandanimal pages 3-6): 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 323 citations and is from a highest quality peer-reviewed journal.
4. (dardick2006plantandanimal pages 6-7): 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 323 citations and is from a highest quality peer-reviewed journal.
5. (freihat2019irak3modulatesdownstream pages 2-3): L. A. Freihat, J. I. Wheeler, A. Wong, I. Turek, D. T. Manallack, and H. R. Irving. Irak3 modulates downstream innate immune signalling through its guanylate cyclase activity. Scientific Reports, Oct 2019. URL: https://doi.org/10.1038/s41598-019-51913-3, doi:10.1038/s41598-019-51913-3. This article has 55 citations and is from a poor quality or predatory journal.
6. (freihat2019irak3modulatesdownstream pages 3-5): L. A. Freihat, J. I. Wheeler, A. Wong, I. Turek, D. T. Manallack, and H. R. Irving. Irak3 modulates downstream innate immune signalling through its guanylate cyclase activity. Scientific Reports, Oct 2019. URL: https://doi.org/10.1038/s41598-019-51913-3, doi:10.1038/s41598-019-51913-3. This article has 55 citations and is from a poor quality or predatory journal.
7. (gosu2012molecularevolutionand pages 1-2): Vijayakumar Gosu, Shaherin Basith, Prasannavenkatesh Durai, and Sangdun Choi. Molecular evolution and structural features of irak family members. PLoS ONE, 7:e49771, Nov 2012. URL: https://doi.org/10.1371/journal.pone.0049771, doi:10.1371/journal.pone.0049771. This article has 64 citations and is from a peer-reviewed journal.
8. (gosu2012molecularevolutionand pages 11-12): Vijayakumar Gosu, Shaherin Basith, Prasannavenkatesh Durai, and Sangdun Choi. Molecular evolution and structural features of irak family members. PLoS ONE, 7:e49771, Nov 2012. URL: https://doi.org/10.1371/journal.pone.0049771, doi:10.1371/journal.pone.0049771. This article has 64 citations and is from a peer-reviewed journal.
9. (gosu2012molecularevolutionand pages 4-5): Vijayakumar Gosu, Shaherin Basith, Prasannavenkatesh Durai, and Sangdun Choi. Molecular evolution and structural features of irak family members. PLoS ONE, 7:e49771, Nov 2012. URL: https://doi.org/10.1371/journal.pone.0049771, doi:10.1371/journal.pone.0049771. This article has 64 citations and is from a peer-reviewed journal.
10. (gosu2012molecularevolutionand pages 9-11): Vijayakumar Gosu, Shaherin Basith, Prasannavenkatesh Durai, and Sangdun Choi. Molecular evolution and structural features of irak family members. PLoS ONE, 7:e49771, Nov 2012. URL: https://doi.org/10.1371/journal.pone.0049771, doi:10.1371/journal.pone.0049771. This article has 64 citations and is from a peer-reviewed journal.
11. (janssens2003functionaldiversityand pages 2-3): Sophie Janssens and Rudi Beyaert. Functional diversity and regulation of different interleukin-1 receptor-associated kinase (irak) family members. Molecular Cell, 11:293-302, Feb 2003. URL: https://doi.org/10.1016/s1097-2765(03)00053-4, doi:10.1016/s1097-2765(03)00053-4. This article has 756 citations and is from a highest quality peer-reviewed journal.
12. (janssens2003functionaldiversityand pages 6-7): Sophie Janssens and Rudi Beyaert. Functional diversity and regulation of different interleukin-1 receptor-associated kinase (irak) family members. Molecular Cell, 11:293-302, Feb 2003. URL: https://doi.org/10.1016/s1097-2765(03)00053-4, doi:10.1016/s1097-2765(03)00053-4. This article has 756 citations and is from a highest quality peer-reviewed journal.
13. (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.
14. (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.
15. (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.
16. (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.
17. (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.
18. (rebl2019atleasttwo pages 1-2): Alexander Rebl, Henrike Rebl, Marieke Verleih, Stephanie Haupt, Judith M. Köbis, Tom Goldammer, and Hans-Martin Seyfert. At least two genes encode many variants of irak3 in rainbow trout, but neither the full-length factor nor its variants interfere directly with the tlr-mediated stimulation of inflammation. Frontiers in Immunology, Sep 2019. URL: https://doi.org/10.3389/fimmu.2019.02246, doi:10.3389/fimmu.2019.02246. This article has 16 citations and is from a peer-reviewed journal.
19. (rhyasen2014irakfamilykinases pages 13-17): GW Rhyasen. Irak family kinases as therapeutic targets for myelodysplastic syndrome and acute myeloid leukemia. Unknown journal, 2014.
20. (rhyasen2014irakfamilykinases pages 17-21): GW Rhyasen. Irak family kinases as therapeutic targets for myelodysplastic syndrome and acute myeloid leukemia. Unknown journal, 2014.
21. (rhyasen2014irakfamilykinases pages 21-25): GW Rhyasen. Irak family kinases as therapeutic targets for myelodysplastic syndrome and acute myeloid leukemia. Unknown journal, 2014.
22. (smith2019alternativesplicingand pages 47-53): MA Smith. Alternative splicing and regulation of innate immune mediators in normal and malignant hematopoiesis. Unknown journal, 2019.
23. (turek2023mutationsinthe pages 7-10): Ilona Turek, Trang H. Nguyen, Charles Galea, Isaiah Abad, Lubna Freihat, David T. Manallack, Tony Velkov, and Helen Irving. Mutations in the vicinity of the irak3 guanylate cyclase center impact its subcellular localization and ability to modulate inflammatory signaling in immortalized cell lines. International Journal of Molecular Sciences, 24:8572, May 2023. URL: https://doi.org/10.3390/ijms24108572, doi:10.3390/ijms24108572. This article has 8 citations and is from a peer-reviewed journal.
24. (wang2013functionalandepidemiological pages 40-44): Hui Wang. Functional and epidemiological characterization ofnon-synonymous single nucleotide polymorphisms in irak2. Unknown journal, 2013. URL: https://doi.org/10.11588/heidok.00015481, doi:10.11588/heidok.00015481. This article has 0 citations.
25. (wang2013functionalandepidemiological pages 44-49): Hui Wang. Functional and epidemiological characterization ofnon-synonymous single nucleotide polymorphisms in irak2. Unknown journal, 2013. URL: https://doi.org/10.11588/heidok.00015481, doi:10.11588/heidok.00015481. This article has 0 citations.
26. (wang2013functionalandepidemiological pages 49-53): Hui Wang. Functional and epidemiological characterization ofnon-synonymous single nucleotide polymorphisms in irak2. Unknown journal, 2013. URL: https://doi.org/10.11588/heidok.00015481, doi:10.11588/heidok.00015481. This article has 0 citations.
27. (blaum2014structureofthe pages 4-5): Bärbel S. Blaum, Sara Mazzotta, Erik R. Nöldeke, Thierry Halter, Johannes Madlung, Birgit Kemmerling, and Thilo Stehle. Structure of the pseudokinase domain of bir2, a regulator of bak1-mediated immune signaling in arabidopsis. Journal of Structural Biology, 186:112-121, Apr 2014. URL: https://doi.org/10.1016/j.jsb.2014.02.005, doi:10.1016/j.jsb.2014.02.005. This article has 72 citations and is from a peer-reviewed journal.
28. (borghese2025irak3isupregulated pages 1-2): Federica Borghese, Richard O. Williams, and Felix I. L. Clanchy. Irak3 is upregulated in rheumatoid arthritis synovium and delays the onset of experimental arthritis. Frontiers in Immunology, Apr 2025. URL: https://doi.org/10.3389/fimmu.2025.1468341, doi:10.3389/fimmu.2025.1468341. This article has 0 citations and is from a peer-reviewed journal.
29. (dardick2006plantandanimal pages 9-10): 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 323 citations and is from a highest quality peer-reviewed journal.
30. (flannery2010theinterleukin1receptorassociated pages 20-24): Sinead Flannery and Andrew G. Bowie. The interleukin-1 receptor-associated kinases: critical regulators of innate immune signalling. Biochemical Pharmacology, 80:1981-1991, Dec 2010. URL: https://doi.org/10.1016/j.bcp.2010.06.020, doi:10.1016/j.bcp.2010.06.020. This article has 392 citations and is from a domain leading peer-reviewed journal.
31. (flannery2010theinterleukin1receptorassociated pages 43-47): Sinead Flannery and Andrew G. Bowie. The interleukin-1 receptor-associated kinases: critical regulators of innate immune signalling. Biochemical Pharmacology, 80:1981-1991, Dec 2010. URL: https://doi.org/10.1016/j.bcp.2010.06.020, doi:10.1016/j.bcp.2010.06.020. This article has 392 citations and is from a domain leading peer-reviewed journal.