1. Phylogeny  
   Serine/threonine‐protein kinase TAO3 (gene TAOK3, also known as MAP3K18, JIK, KDS, DPK, HD‐CL‐09) is classified within the Sterile‐20 (Ste20) kinase superfamily and specifically belongs to the germinal center kinase (GCK) subfamily 8. TAO3 is one of three mammalian paralogs—TAOK1, TAOK2, and TAOK3—that emerged from an evolutionary process common to stress‐activated MAP kinase kinase kinases (MAP3Ks) in vertebrates. The phylogenetic analysis using the framework described by Manning et al. places TAO3 in a conserved clade present across all mammalian species, and the presence of orthologs in other eukaryotes supports its origin from an early common ancestor of eukaryotes. Its evolutionary relationship with TAOK1 and TAOK2 is underscored by a high degree of conservation in the catalytic domains as well as in specific regulatory regions that are characteristic of the GCK family kinases (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, byeon2024pleiotropicfunctionsof pages 1-3).
2. Reaction Catalyzed  
   TAO3 catalyzes a classical kinase reaction in which the γ‐phosphate group from ATP is transferred to a serine or threonine residue of a substrate protein. The reaction mechanism can be summarized as follows:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺.  
   This phosphorylation reaction alters the substrate’s functional state and is central to the propagation of intracellular signaling cascades typically associated with stress responses and cell cycle regulation (goldsmith2007substrateanddocking pages 1-2).
3. Cofactor Requirements  
   In common with most serine/threonine kinases, the catalytic activity of TAO3 is dependent on the presence of divalent metal ions, with Mg²⁺ serving as an essential cofactor. The coordination of Mg²⁺ with ATP is critical to correctly position the nucleotide for effective phosphoryl transfer during the kinase reaction (goldsmith2007substrateanddocking pages 1-2).
4. Substrate Specificity  
   TAO3 exhibits substrate specificity that is consistent with other stress‐activated MAP3Ks and members of the GCK subfamily. It preferentially phosphorylates serine and threonine residues that lie within consensus motifs typical of its kinase family. Key targets include upstream MAP2Ks such as MAP2K3 and MAP2K6, which are subsequently involved in the activation of the p38/MAPK14 stress‐activated cascade. In addition, experimental studies have demonstrated that TAO3 phosphorylates substrates implicated in cytoskeletal dynamics; for example, it phosphorylates cytoplasmic dynein 1 light intermediate chain 2 (LIC2) at serine 202, a modification that modulates invadopodia formation in cancer cells. Although the precise consensus sequence is not fully delineated, data from substrate specificity studies on serine/threonine kinases collectively support a model wherein TAO3 targets motifs that include a phosphorylatable threonine or serine residue, often followed or preceded by basic amino acids, in line with other members of the Ste20 family (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, iizuka2021serinethreoninekinasetao3mediated pages 6-8, byeon2024pleiotropicfunctionsof pages 3-4, goldsmith2007substrateanddocking pages 2-3).
5. Structure  
   TAO3 is organized into several distinct regions that together facilitate its catalytic and regulatory functions. The protein possesses a highly conserved N-terminal serine/threonine kinase domain which comprises approximately the first 319 amino acids. This domain adopts the canonical bilobal structure observed in many protein kinases, including an N-terminal lobe rich in β-strands and a predominantly α-helical C-terminal lobe. Critical structural features of this kinase domain include the activation loop, which undergoes phosphorylation to modulate activity, the hydrophobic spine that is essential for maintaining the active conformation, and the conserved C-helix that contributes to ATP binding (goldsmith2007substrateanddocking pages 3-4, thiriet2013cytoplasmicproteinserinethreonine pages 18-21).

Beyond the kinase domain, TAO3 contains extended C-terminal regulatory regions that are involved in substrate docking and interactions. In particular, conserved PF1 and PF2 domains have been identified within the C-terminal region: the PF1 domain is implicated in the modulation of catalytic activity, while the PF2 domain facilitates protein–protein interactions with downstream effectors and adaptor molecules. Although no experimental crystal structure of full-length TAO3 has been reported, biochemical studies, including those that have engineered constructs for structural investigations, suggest that this modular arrangement is typical for kinases in the TAO family (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, iizuka2021serinethreoninekinasetao3mediated pages 6-8). Furthermore, structural comparisons with other serine/threonine kinases indicate that TAO3 likely features an activation loop whose phosphorylation status is key to its conformational shifts between inactive and active states, a regulatory mechanism that is supported by data on homologous kinases (goldsmith2007substrateanddocking pages 6-7, thiriet2013cytoplasmicproteinserinethreonine pages 4-7).

1. Regulation  
   TAO3 is subject to elaborate regulation that occurs via both post-translational modifications and interaction with upstream signaling components. One significant level of regulation is mediated by phosphorylation events. Upstream kinases such as WNK1 and WNK4 have been reported to phosphorylate TAO3, contributing to its activation in response to specific stimuli. Additionally, TAO3 is capable of autophosphorylation; mutation of key catalytic residues, notably the ATP-binding lysine (e.g., lysine 53), results in a kinase-dead variant that fails to perform its downstream functions, underscoring the critical role of phosphorylation in its regulation (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, iizuka2021serinethreoninekinasetao3mediated pages 8-10).

Moreover, the regulatory PF2 domain present in its C-terminal region facilitates interactions with adaptor proteins and other kinases, thereby integrating TAO3 into broader signaling networks. In the context of stress signaling, TAO3 activates the p38/MAPK14 cascade by phosphorylating MAP2K3 and MAP2K6 while concurrently inhibiting the basal activity of the MAPK8/JNK pathway. The dual regulation—activation of one pathway and inhibition of another—allows TAO3 to fine-tune cellular responses to DNA damage and other stress cues, particularly during the G₂/M transition of the cell cycle (thiriet2013cytoplasmicproteinserinethreonine pages 18-21, byeon2024pleiotropicfunctionsof pages 16-18). This complex regulation is further evidenced by its role in diminishing EGF-induced activation of JNK signaling, suggesting the existence of additional modulation by extracellular receptor-mediated pathways (byeon2024pleiotropicfunctionsof pages 7-9).

1. Function  
   TAO3 plays a central role in the regulation of stress-activated MAPK signaling cascades. Its primary function is to act as an activator of the p38/MAPK14 pathway. In response to DNA damage, TAO3 phosphorylates upstream kinases, specifically MAP2K3 and MAP2K6, which in turn activate p38/MAPK14. This activation is critical for the establishment of the G₂/M cell cycle checkpoint, ensuring that cells do not progress into mitosis with damaged DNA (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, byeon2024pleiotropicfunctionsof pages 4-6).

In addition to its positive regulation of the p38 pathway, TAO3 exerts an inhibitory effect on the MAPK8/JNK cascade. By inhibiting basal JNK activity and reducing its activation in response to epidermal growth factor (EGF), TAO3 ensures a balanced cellular stress response and prevents the unwarranted activation of pro-apoptotic signals under normal conditions (thiriet2013cytoplasmicproteinserinethreonine pages 18-21, byeon2024pleiotropicfunctionsof pages 1-3).

Beyond its role in canonical MAPK signaling, TAO3 has been implicated in the regulation of cytoskeletal dynamics and cancer cell invasion. Studies have shown that TAO3 is highly expressed in several cancer types, including melanoma, breast, and bladder cancers, where it regulates the trafficking of endosomes containing the invadopodia scaffold protein TKS5α. Phosphorylation of targets such as cytoplasmic dynein 1 light intermediate chain 2 (LIC2) at serine 202 by TAO3 contributes to the formation of invadopodia and facilitates extracellular matrix degradation, thereby promoting tumor invasion and metastasis (iizuka2021serinethreoninekinasetao3mediated pages 6-8, pages 11-13).

Expression analyses indicate that TAO3 is significantly enriched in hematopoietic cells, including peripheral blood leukocytes, the thymus, and the spleen, with additional moderate expression observed in organs such as the prostate, testes, and pancreas. This tissue distribution supports its involvement in diverse cellular processes ranging from immune regulation to stress response (thiriet2013cytoplasmicproteinserinethreonine pages 18-21, byeon2024pleiotropicfunctionsof pages 7-9).

1. Other Comments  
   Experimental inhibitors targeting TAO3 have been identified, providing a potential therapeutic avenue for diseases associated with its dysregulation. For example, the small molecule SBI-581 has been reported to inhibit TAO3 kinase activity with an IC₅₀ of approximately 42 nM, and its use in experimental settings has demonstrated a reduction in invadopodia formation and subsequent cancer cell invasion (iizuka2021serinethreoninekinasetao3mediated pages 6-8).

TAO3’s involvement in oncogenic processes is further underscored by its high expression in cancers characterized by aggressive invasion and metastasis. Its dual role in activating the p38/MAPK14 pathway—thereby contributing to DNA damage checkpoint control—and inhibiting the JNK cascade highlights its complex function in promoting cell survival and proliferation in a context-dependent manner (thiriet2013cytoplasmicproteinserinethreonine pages 14-18, byeon2024pleiotropicfunctionsof pages 12-14).

There is an ongoing interest in further characterizing the structural features of TAO3 using techniques such as X-ray crystallography and advanced computational modeling, as these studies are expected to provide additional insights into its regulatory and catalytic mechanisms. Such insights may facilitate the rational design of more selective inhibitors that target TAO3 in pathological conditions, including various invasive cancers (iizuka2021serinethreoninekinasetao3mediated pages 11-13).

1. References
2. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 14-18, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 14-18).
3. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 18-21, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 18-21).
4. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 11-14, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 11-14).
5. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 4-7, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 4-7).
6. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 1-4, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 1-4).
7. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 82-86, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 82-86).
8. Thiriet, M. “Cytoplasmic protein serine/threonine kinases.” Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 113-116, Jul 2013 (thiriet2013cytoplasmicproteinserinethreonine pages 113-116).
9. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 1-3, Jan 2024 (byeon2024pleiotropicfunctionsof pages 1-3).
10. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 16-18, Jan 2024 (byeon2024pleiotropicfunctionsof pages 16-18).
11. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 23-29, Jan 2024 (byeon2024pleiotropicfunctionsof pages 23-29).
12. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 3-4, Jan 2024 (byeon2024pleiotropicfunctionsof pages 3-4).
13. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 19-20, Jan 2024 (byeon2024pleiotropicfunctionsof pages 19-20).
14. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 20-21, Jan 2024 (byeon2024pleiotropicfunctionsof pages 20-21).
15. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 4-6, Jan 2024 (byeon2024pleiotropicfunctionsof pages 4-6).
16. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 12-14, Jan 2024 (byeon2024pleiotropicfunctionsof pages 12-14).
17. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 6-7, Jan 2024 (byeon2024pleiotropicfunctionsof pages 6-7).
18. Byeon, S., & Yadav, S. “Pleiotropic functions of TAO kinases and their dysregulation in neurological disorders.” Science Signaling, pages 7-9, Jan 2024 (byeon2024pleiotropicfunctionsof pages 7-9).
19. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, pages 1-2, Oct 2007 (goldsmith2007substrateanddocking pages 1-2).
20. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, pages 2-3, Oct 2007 (goldsmith2007substrateanddocking pages 2-3).
21. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, pages 3-4, Oct 2007 (goldsmith2007substrateanddocking pages 3-4).
22. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, pages 6-7, Oct 2007 (goldsmith2007substrateanddocking pages 6-7).
23. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, page 15-15, Oct 2007 (goldsmith2007substrateanddocking pages 15-15).
24. Goldsmith, E. J., Akella, R., Min, X., Zhou, T., & Humphreys, J. M. “Substrate and docking interactions in serine/threonine protein kinases.” Chemical Reviews, pages 16-17, Oct 2007 (goldsmith2007substrateanddocking pages 16-17).
25. Iizuka, S. et al. “Serine-threonine kinase TAO3-mediated trafficking of endosomes containing the invadopodia scaffold TKS5α promotes cancer invasion and tumor growth.” Cancer Research, pages 6-8, Jan 2021 (iizuka2021serinethreoninekinasetao3mediated pages 6-8).
26. Iizuka, S. et al. “Serine-threonine kinase TAO3-mediated trafficking of endosomes containing the invadopodia scaffold TKS5α promotes cancer invasion and tumor growth.” Cancer Research, pages 11-13, Jan 2021 (iizuka2021serinethreoninekinasetao3mediated pages 11-13).
27. Iizuka, S. et al. “Serine-threonine kinase TAO3-mediated trafficking of endosomes containing the invadopodia scaffold TKS5α promotes cancer invasion and tumor growth.” Cancer Research, pages 8-10, Jan 2021 (iizuka2021serinethreoninekinasetao3mediated pages 8-10).
28. Koster, K.-A. et al. “Regulation of the activity of the dual leucine zipper kinase by distinct mechanisms.” Cells, pages 15-16, Feb 2024 (koster2024regulationofthe pages 15-16).
29. Koster, K.-A. et al. “Regulation of the activity of the dual leucine zipper kinase by distinct mechanisms.” Cells, pages 11-12, Feb 2024 (koster2024regulationofthe pages 11-12).

References

1. (thiriet2013cytoplasmicproteinserinethreonine pages 14-18): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
2. (thiriet2013cytoplasmicproteinserinethreonine pages 18-21): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
3. (byeon2024pleiotropicfunctionsof pages 1-3): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
4. (byeon2024pleiotropicfunctionsof pages 16-18): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
5. (byeon2024pleiotropicfunctionsof pages 23-29): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
6. (byeon2024pleiotropicfunctionsof pages 3-4): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
7. (goldsmith2007substrateanddocking pages 2-3): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
8. (iizuka2021serinethreoninekinasetao3mediated pages 6-8): S. Iizuka, M. Quintavalle, Jose Navarro, Kyle P. Gribbin, R. Ardecky, M. Abelman, Chen-Ting Ma, E. Sergienko, F. Zeng, I. Pass, G. Thomas, S. McWeeney, C. Hassig, A. Pinkerton, and S. Courtneidge. Serine-threonine kinase tao3-mediated trafficking of endosomes containing the invadopodia scaffold tks5α promotes cancer invasion and tumor growth. Cancer Research, 81:1472-1485, Jan 2021. URL: https://doi.org/10.1158/0008-5472.can-20-2383, doi:10.1158/0008-5472.can-20-2383. This article has 14 citations and is from a highest quality peer-reviewed journal.
9. (koster2024regulationofthe pages 15-16): Kyra-Alexandra Köster, Marten Dethlefs, Jorge Duque Escobar, and Elke Oetjen. Regulation of the activity of the dual leucine zipper kinase by distinct mechanisms. Cells, 13:333, Feb 2024. URL: https://doi.org/10.3390/cells13040333, doi:10.3390/cells13040333. This article has 4 citations and is from a peer-reviewed journal.
10. (thiriet2013cytoplasmicproteinserinethreonine pages 11-14): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
11. (thiriet2013cytoplasmicproteinserinethreonine pages 4-7): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
12. (byeon2024pleiotropicfunctionsof pages 19-20): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
13. (byeon2024pleiotropicfunctionsof pages 20-21): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
14. (byeon2024pleiotropicfunctionsof pages 4-6): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
15. (goldsmith2007substrateanddocking pages 1-2): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
16. (goldsmith2007substrateanddocking pages 3-4): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
17. (iizuka2021serinethreoninekinasetao3mediated pages 11-13): S. Iizuka, M. Quintavalle, Jose Navarro, Kyle P. Gribbin, R. Ardecky, M. Abelman, Chen-Ting Ma, E. Sergienko, F. Zeng, I. Pass, G. Thomas, S. McWeeney, C. Hassig, A. Pinkerton, and S. Courtneidge. Serine-threonine kinase tao3-mediated trafficking of endosomes containing the invadopodia scaffold tks5α promotes cancer invasion and tumor growth. Cancer Research, 81:1472-1485, Jan 2021. URL: https://doi.org/10.1158/0008-5472.can-20-2383, doi:10.1158/0008-5472.can-20-2383. This article has 14 citations and is from a highest quality peer-reviewed journal.
18. (byeon2024pleiotropicfunctionsof pages 12-14): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
19. (byeon2024pleiotropicfunctionsof pages 6-7): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
20. (byeon2024pleiotropicfunctionsof pages 7-9): Sujin Byeon and Smita Yadav. Pleiotropic functions of tao kinases and their dysregulation in neurological disorders. Science Signaling, Jan 2024. URL: https://doi.org/10.1126/scisignal.adg0876, doi:10.1126/scisignal.adg0876. This article has 2 citations and is from a domain leading peer-reviewed journal.
21. (iizuka2021serinethreoninekinasetao3mediated pages 8-10): S. Iizuka, M. Quintavalle, Jose Navarro, Kyle P. Gribbin, R. Ardecky, M. Abelman, Chen-Ting Ma, E. Sergienko, F. Zeng, I. Pass, G. Thomas, S. McWeeney, C. Hassig, A. Pinkerton, and S. Courtneidge. Serine-threonine kinase tao3-mediated trafficking of endosomes containing the invadopodia scaffold tks5α promotes cancer invasion and tumor growth. Cancer Research, 81:1472-1485, Jan 2021. URL: https://doi.org/10.1158/0008-5472.can-20-2383, doi:10.1158/0008-5472.can-20-2383. This article has 14 citations and is from a highest quality peer-reviewed journal.
22. (koster2024regulationofthe pages 11-12): Kyra-Alexandra Köster, Marten Dethlefs, Jorge Duque Escobar, and Elke Oetjen. Regulation of the activity of the dual leucine zipper kinase by distinct mechanisms. Cells, 13:333, Feb 2024. URL: https://doi.org/10.3390/cells13040333, doi:10.3390/cells13040333. This article has 4 citations and is from a peer-reviewed journal.
23. (goldsmith2007substrateanddocking pages 15-15): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
24. (goldsmith2007substrateanddocking pages 16-17): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
25. (goldsmith2007substrateanddocking pages 6-7): Elizabeth J. Goldsmith, Radha Akella, Xiaoshan Min, Tianjun Zhou, and John M. Humphreys. Substrate and docking interactions in serine/threonine protein kinases. Chemical Reviews, 107:5065-5081, Oct 2007. URL: https://doi.org/10.1021/cr068221w, doi:10.1021/cr068221w. This article has 155 citations and is from a highest quality peer-reviewed journal.
26. (thiriet2013cytoplasmicproteinserinethreonine pages 1-4): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
27. (thiriet2013cytoplasmicproteinserinethreonine pages 113-116): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
28. (thiriet2013cytoplasmicproteinserinethreonine pages 82-86): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.