1. Phylogeny  
   TAOK1 is an evolutionarily conserved serine/threonine kinase that belongs to the STE20 kinase family and specifically to the MAP3K subgroup, with close relationships to its paralogs TAOK2 and TAOK3 (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, byeon2024pleiotropicfunctionsof pages 1-3). Its kinase domain is highly conserved across species, with orthologs documented in invertebrates such as Drosophila (Tao) and Caenorhabditis elegans (KIN-18), as well as in vertebrates including fish, rodents, and humans, indicating that the core signaling roles of TAOK1 were established early in evolution (beeman2023neurodevelopmentaldisorder–associatedmutations pages 3-4, ying2024roleofste20typea pages 25-27). Phylogenetic studies position TAOK1 within a cluster of protein kinases that mediate stress-activated MAPK cascades, underscoring its ancestral connection to the STE20 family members that predominate in eukaryotic signaling networks (byeon2024pleiotropicfunctionsof pages 29-34, fang2020thediverseroles pages 1-3).
2. Reaction Catalyzed  
   TAOK1 functions as a serine/threonine-protein kinase, catalyzing the phosphorylation of specific protein substrates by transferring a phosphate group from ATP to serine or threonine residues on the target proteins. The chemical reaction can be summarized as: ATP + [protein substrate] → ADP + [protein substrate]-phosphate + H⁺ (beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7, ning2025multiomicsanalysisrevealed pages 14-16). Physiologically, TAOK1 directly phosphorylates MAP kinase kinase substrates such as MAP2K3 and MAP2K6, thereby acting as an upstream activator of the p38/MAPK14 stress-activated cascade. In addition, it phosphorylates MARK2 at Thr208, which leads to its activation and subsequent phosphorylation of MAPT/tau, resulting in the detachment of tau from microtubules and thereby regulating cytoskeletal stability (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, fang2020thediverseroles pages 11-13).
3. Cofactor Requirements  
   The catalytic activity of TAOK1, similar to many serine/threonine kinases, is dependent on the binding of ATP as a phosphate donor along with essential divalent metal ions such as Mg²⁺ which are required to stabilize the charged phosphate groups during the transfer reaction (ning2025multiomicsanalysisrevealed pages 14-16, ying2024roleofste20typea pages 74-76). Although the detailed characterization of additional cofactor or regulatory molecule requirements for TAOK1 remains to be fully elucidated, it follows the canonical mechanism observed in the kinase superfamily where ATP and Mg²⁺ are indispensable for phosphorylation activity (byeon2024pleiotropicfunctionsof pages 4-6, hu2021clinicalandneurobiological pages 2-3).
4. Substrate Specificity  
   TAOK1 exhibits substrate specificity for serine/threonine residues and has been demonstrated to phosphorylate proteins critical for MAP kinase cascades and cytoskeletal regulation. Its physiological substrates include MAP2K3 and MAP2K6, whose phosphorylation leads to downstream activation of p38 MAPK; it also phosphorylates MARK2, thereby modulating microtubule dynamics through the detachment of tau protein from microtubules (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7). The consensus specificity likely revolves around recognition of target motifs that include nearby basic residues, and the kinase shows a preference for threonine phosphorylation, as suggested by analyses of phosphorylation events and the autophosphorylation regulation that involves key threonine residues (byeon2024pleiotropicfunctionsof pages 12-14, fang2020thediverseroles pages 13-15). Although precise consensus motifs have not been universally defined for TAOK1, experimental evidence points to its pivotal role in modulating substrates that govern both stress responses and cytoskeletal reorganization (byeon2024pleiotropicfunctionsof pages 7-9, yoder2023geneexpressionanalysis pages 10-12).
5. Structure  
   TAOK1 is a large protein, approximately 1001 amino acids in length, with a multidomain architecture that is crucial for its function. The N-terminal region contains a highly conserved kinase domain (approximately residues 1–320), which is responsible for its catalytic activity. This domain is characterized by the conventional serine/threonine kinase fold that binds ATP and facilitates phosphoryl transfer (beeman2023neurodevelopmentaldisorder–associatedmutations pages 3-4, dulovicmahlow2019denovovariants pages 3-4). Immediately following the kinase domain, TAOK1 features a series of predicted coiled-coil motifs that mediate protein–protein interactions, and these regions are implicated in subcellular localization and membrane association. Notably, the C-terminal segment also contains a unique triple helix structure that directly binds phosphoinositides and is essential for plasma membrane association and membrane remodeling (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, byeon2024pleiotropicfunctionsof pages 3-4). Critical regulatory residues include autophosphorylation sites such as Thr440 and Thr443, whose phosphorylation status controls the enzyme’s switch between an active cytosolic state and an inactive membrane-bound state (beeman2023neurodevelopmentaldisorder–associatedmutations pages 25-26, byeon2024pleiotropicfunctionsof pages 18-19). Structural predictions from AlphaFold2.0 further support this domain organization and highlight the spatial separation between the catalytic core and membrane-interactive regions (ying2024roleofste20typea pages 25-27, fang2020thediverseroles pages 5-8).
6. Regulation  
   The activity of TAOK1 is intricately regulated by multiple post-translational modifications and protein–protein interactions. One of the central regulatory mechanisms is autophosphorylation; TAOK1 autophosphorylates residue Ser181 within its catalytic loop, which is crucial for its kinase activity, and it also phosphorylates residues Thr440 and Thr443 that regulate its localization by modulating plasma membrane association (beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7, beeman2023neurodevelopmentaldisorder–associatedmutations pages 25-26). In addition, TAOK1 is subject to regulation by upstream kinases such as MST3, part of the Hippo signaling pathway, which phosphorylates TAOK1 and influences dendritic spine formation in neuronal cells (beeman2023neurodevelopmentaldisorder–associatedmutations pages 15-20, byeon2024pleiotropicfunctionsof pages 16-18). Its kinase activity is also modulated by interactions with cellular lipids; the triple helix in the C-terminal region binds phosphoinositides, and this membrane interaction is negatively regulated by autophosphorylation (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, byeon2024pleiotropicfunctionsof pages 3-4). Furthermore, disease-associated mutations within the kinase domain render TAOK1 catalytically inactive, trapping it in a membrane-bound state and leading to aberrant membrane remodeling, which in turn distorts neuronal development (dulovicmahlow2019denovovariants pages 1-2, byeon2024pleiotropicfunctionsof pages 11-12). These layers of regulation ensure that TAOK1’s activity is tightly controlled in response to diverse cellular signals such as DNA damage, stress, and extracellular cues (hu2021clinicalandneurobiological pages 3-4, fang2020thediverseroles pages 17-19).
7. Function  
   TAOK1 plays multifaceted roles in cellular physiology, prominently functioning as a key mediator in stress-activated signaling pathways and in maintaining cytoskeletal stability. It contributes to the activation of the p38/MAPK14 cascade by phosphorylating MAP2K3 and MAP2K6, thus playing a critical role in the DNA damage response and in cell cycle checkpoint regulation at the G2/M transition (beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7, ning2025multiomicsanalysisrevealed pages 11-14). By phosphorylating MARK2 at Thr208, TAOK1 indirectly influences the stability of microtubules through the timely detachment of MAPT/tau from microtubule filaments; this function is vital for proper cytoskeletal remodeling and neuronal polarity (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3, fang2020thediverseroles pages 11-13). In addition to its role in stress response and cytoskeletal dynamics, TAOK1 is implicated in the regulation of apoptosis. It participates in the activation of the MAPK8/JNK cascade, thereby modulating apoptotic morphological changes such as cell contraction, membrane blebbing, and the formation of apoptotic bodies (byeon2024pleiotropicfunctionsof pages 29-34, fang2020thediverseroles pages 19-20). TAOK1 is also essential in the nervous system; its high expression in mammalian brain regions including the hippocampus, neocortex, and cerebellum underscores its involvement in neuronal development, dendritic arborization, and migration to the cortical plate. Dysfunctional TAOK1, as observed in cases with de novo missense and truncating mutations, leads to impaired dendritic branching and abnormal neuronal morphology, which are linked to neurodevelopmental disorders such as intellectual disability, developmental delay, and autism spectrum disorder (beeman2023neurodevelopmentaldisorder–associatedmutations pages 15-20, dulovicmahlow2019denovovariants pages 1-2, hu2021clinicalandneurobiological pages 5-6). Therefore, TAOK1 integrates extracellular stress signals with intracellular responses by modulating key signaling cascades that influence cell growth, survival, cytoskeletal rearrangements, and neural connectivity (byeon2024pleiotropicfunctionsof pages 16-18, hu2021clinicalandneurobiological pages 6-8).
8. Other Comments  
   Inhibitor development targeting TAOK1 is an area of active research given its central roles in stress response, apoptosis, and neurodevelopment. Although specific TAOK1 inhibitors have not yet been fully optimized, related studies using broad-spectrum or TAOK family inhibitors have provided insights into its potential as a therapeutic target in neurodevelopmental disorders and certain cancers (ying2024roleofste20type pages 74-76, ning2025multiomicsanalysisrevealed pages 11-14). Mutations in TAOK1 have been identified in patients with neurodevelopmental disorders, and these variants often result in a loss of kinase activity and aberrant plasma membrane association, thus leading to defects in neuronal morphology and connectivity (dulovicmahlow2019denovovariants pages 2-3, beeman2023neurodevelopmentaldisorder–associatedmutations pages 3-4). In addition, TAOK1′s involvement in pathways such as the p38 MAPK cascade and JNK activation links it to responses to DNA damage and cellular stress, which may further connect its dysregulation to oncogenic processes (ning2025multiomicsanalysisrevealed pages 14-16, byeon2024pleiotropicfunctionsof pages 18-19). Ongoing research is focused on elucidating the precise mechanisms that control its autophosphorylation, subcellular localization, and interactions with regulatory proteins, as well as on mapping its complete substrate spectrum using phosphoproteomics (fang2020thediverseroles pages 16-17, hu2021clinicalandneurobiological pages 1-2). The multiplicity of TAOK1’s roles in neuronal migration, cytoskeletal dynamics, and stress signaling makes it a critical node in both normal physiology and disease, and further studies are anticipated to clarify its potential as a target for therapeutic intervention in neurodevelopmental and neurodegenerative disorders (byeon2024pleiotropicfunctionsof pages 19-20, wernigg2025theserinethreoninekinase pages 88-91).
9. References  
   beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3; beeman2023neurodevelopmentaldisorder–associatedmutations pages 3-4; beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7; beeman2023neurodevelopmentaldisorder–associatedmutations pages 15-20; beeman2023neurodevelopmentaldisorder–associatedmutations pages 25-26; byeon2024pleiotropicfunctionsof pages 1-3; byeon2024pleiotropicfunctionsof pages 3-4; byeon2024pleiotropicfunctionsof pages 4-6; byeon2024pleiotropicfunctionsof pages 6-7; byeon2024pleiotropicfunctionsof pages 7-9; byeon2024pleiotropicfunctionsof pages 11-12; byeon2024pleiotropicfunctionsof pages 12-14; byeon2024pleiotropicfunctionsof pages 16-18; byeon2024pleiotropicfunctionsof pages 18-19; byeon2024pleiotropicfunctionsof pages 19-20; dulovicmahlow2019denovovariants pages 1-2; dulovicmahlow2019denovovariants pages 2-3; dulovicmahlow2019denovovariants pages 3-4; fang2020thediverseroles pages 1-3; fang2020thediverseroles pages 5-8; fang2020thediverseroles pages 8-11; fang2020thediverseroles pages 11-13; fang2020thediverseroles pages 13-15; fang2020thediverseroles pages 15-16; fang2020thediverseroles pages 16-17; fang2020thediverseroles pages 17-19; fang2020thediverseroles pages 19-20; hu2021clinicalandneurobiological pages 1-2; hu2021clinicalandneurobiological pages 2-3; hu2021clinicalandneurobiological pages 3-4; hu2021clinicalandneurobiological pages 4-5; hu2021clinicalandneurobiological pages 5-6; hu2021clinicalandneurobiological pages 6-8; ning2025multiomicsanalysisrevealed pages 11-14; ning2025multiomicsanalysisrevealed pages 14-16; ying2024roleofste20type pages 25-27; ying2024roleofste20type pages 74-76; ying2024roleofste20typea pages 25-27; ying2024roleofste20typea pages 74-76; yoder2023geneexpressionanalysis pages 10-12; wernigg2025theserinethreoninekinase pages 23-27; wernigg2025theserinethreoninekinase pages 78-81; wernigg2025theserinethreoninekinase pages 88-91.

References

1. (beeman2023neurodevelopmentaldisorder–associatedmutations pages 1-3): N. Beeman, Tanmay R. Sapre, Shao-En Ong, and Smita Yadav. Neurodevelopmental disorder–associated mutations in taok1 reveal its function as a plasma membrane remodeling kinase. Science Signaling, Jan 2023. URL: https://doi.org/10.1126/scisignal.add3269, doi:10.1126/scisignal.add3269. This article has 13 citations and is from a domain leading peer-reviewed journal.
2. (beeman2023neurodevelopmentaldisorder–associatedmutations pages 15-20): N. Beeman, Tanmay R. Sapre, Shao-En Ong, and Smita Yadav. Neurodevelopmental disorder–associated mutations in taok1 reveal its function as a plasma membrane remodeling kinase. Science Signaling, Jan 2023. URL: https://doi.org/10.1126/scisignal.add3269, doi:10.1126/scisignal.add3269. This article has 13 citations and is from a domain leading peer-reviewed journal.
3. (beeman2023neurodevelopmentaldisorder–associatedmutations pages 6-7): N. Beeman, Tanmay R. Sapre, Shao-En Ong, and Smita Yadav. Neurodevelopmental disorder–associated mutations in taok1 reveal its function as a plasma membrane remodeling kinase. Science Signaling, Jan 2023. URL: https://doi.org/10.1126/scisignal.add3269, doi:10.1126/scisignal.add3269. This article has 13 citations and is from a domain leading peer-reviewed journal.
4. (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.
5. (byeon2024pleiotropicfunctionsof pages 29-34): 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. (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.
8. (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.
9. (fang2020thediverseroles pages 1-3): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
10. (fang2020thediverseroles pages 11-13): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
11. (fang2020thediverseroles pages 13-15): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
12. (fang2020thediverseroles pages 17-19): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
13. (fang2020thediverseroles pages 5-8): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
14. (fang2020thediverseroles pages 8-11): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
15. (hu2021clinicalandneurobiological pages 2-3): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
16. (hu2021clinicalandneurobiological pages 3-4): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
17. (ning2025multiomicsanalysisrevealed pages 11-14): Li Ning, Xiu Li, Yating Xu, Yu Si, Hongting Zhao, and Qingling Ren. Multi-omics analysis revealed that taok1 can be used as a prognostic marker and target in a variety of tumors, especially in cervical cancer. OncoTargets and Therapy, Volume 18:335-353, Mar 2025. URL: https://doi.org/10.2147/ott.s506582, doi:10.2147/ott.s506582. This article has 0 citations and is from a peer-reviewed journal.
18. (ning2025multiomicsanalysisrevealed pages 14-16): Li Ning, Xiu Li, Yating Xu, Yu Si, Hongting Zhao, and Qingling Ren. Multi-omics analysis revealed that taok1 can be used as a prognostic marker and target in a variety of tumors, especially in cervical cancer. OncoTargets and Therapy, Volume 18:335-353, Mar 2025. URL: https://doi.org/10.2147/ott.s506582, doi:10.2147/ott.s506582. This article has 0 citations and is from a peer-reviewed journal.
19. (ying2024roleofste20type pages 25-27): X Ying. Role of ste20-type kinases in liver lipid metabolism and hepatocarcinogenesis: insights from in vitro and in vivo studies. Unknown journal, 2024.
20. (ying2024roleofste20type pages 74-76): X Ying. Role of ste20-type kinases in liver lipid metabolism and hepatocarcinogenesis: insights from in vitro and in vivo studies. Unknown journal, 2024.
21. (ying2024roleofste20typea pages 25-27): X Ying. Role of ste20-type kinases in liver lipid metabolism and hepatocarcinogenesis: insights from in vitro and in vivo studies. Unknown journal, 2024.
22. (ying2024roleofste20typea pages 74-76): X Ying. Role of ste20-type kinases in liver lipid metabolism and hepatocarcinogenesis: insights from in vitro and in vivo studies. Unknown journal, 2024.
23. (yoder2023geneexpressionanalysis pages 10-12): Michael D. Yoder, Steven Van Osten, and Gregory F. Weber. Gene expression analysis of the tao kinase family of ste20p-like map kinase kinase kinases during early embryonic development in xenopus laevis. Gene Expression Patterns, 48:119318, Jun 2023. URL: https://doi.org/10.1016/j.gep.2023.119318, doi:10.1016/j.gep.2023.119318. This article has 2 citations and is from a peer-reviewed journal.
24. (beeman2023neurodevelopmentaldisorder–associatedmutations pages 25-26): N. Beeman, Tanmay R. Sapre, Shao-En Ong, and Smita Yadav. Neurodevelopmental disorder–associated mutations in taok1 reveal its function as a plasma membrane remodeling kinase. Science Signaling, Jan 2023. URL: https://doi.org/10.1126/scisignal.add3269, doi:10.1126/scisignal.add3269. This article has 13 citations and is from a domain leading peer-reviewed journal.
25. (beeman2023neurodevelopmentaldisorder–associatedmutations pages 3-4): N. Beeman, Tanmay R. Sapre, Shao-En Ong, and Smita Yadav. Neurodevelopmental disorder–associated mutations in taok1 reveal its function as a plasma membrane remodeling kinase. Science Signaling, Jan 2023. URL: https://doi.org/10.1126/scisignal.add3269, doi:10.1126/scisignal.add3269. This article has 13 citations and is from a domain leading peer-reviewed journal.
26. (byeon2024pleiotropicfunctionsof pages 11-12): 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.
27. (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.
28. (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.
29. (byeon2024pleiotropicfunctionsof pages 18-19): 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.
30. (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.
31. (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.
32. (dulovicmahlow2019denovovariants pages 1-2): Marija Dulovic-Mahlow, Joanne Trinh, Krishna Kumar Kandaswamy, Geir Julius Braathen, Nataliya Di Donato, Elisa Rahikkala, Skadi Beblo, Martin Werber, Victor Krajka, Øyvind L. Busk, Hauke Baumann, Nouriya Abbas Al-Sannaa, Frauke Hinrichs, Rabea Affan, Nir Navot, Mohammed A. Al Balwi, Gabriela Oprea, Øystein L. Holla, Maximilian E.R. Weiss, Rami A. Jamra, Anne-Karin Kahlert, Shivendra Kishore, Kristian Tveten, Melissa Vos, Arndt Rolfs, and Katja Lohmann. De novo variants in taok1 cause neurodevelopmental disorders. The American Journal of Human Genetics, 105:213-220, Jul 2019. URL: https://doi.org/10.1016/j.ajhg.2019.05.005, doi:10.1016/j.ajhg.2019.05.005. This article has 46 citations.
33. (dulovicmahlow2019denovovariants pages 2-3): Marija Dulovic-Mahlow, Joanne Trinh, Krishna Kumar Kandaswamy, Geir Julius Braathen, Nataliya Di Donato, Elisa Rahikkala, Skadi Beblo, Martin Werber, Victor Krajka, Øyvind L. Busk, Hauke Baumann, Nouriya Abbas Al-Sannaa, Frauke Hinrichs, Rabea Affan, Nir Navot, Mohammed A. Al Balwi, Gabriela Oprea, Øystein L. Holla, Maximilian E.R. Weiss, Rami A. Jamra, Anne-Karin Kahlert, Shivendra Kishore, Kristian Tveten, Melissa Vos, Arndt Rolfs, and Katja Lohmann. De novo variants in taok1 cause neurodevelopmental disorders. The American Journal of Human Genetics, 105:213-220, Jul 2019. URL: https://doi.org/10.1016/j.ajhg.2019.05.005, doi:10.1016/j.ajhg.2019.05.005. This article has 46 citations.
34. (dulovicmahlow2019denovovariants pages 3-4): Marija Dulovic-Mahlow, Joanne Trinh, Krishna Kumar Kandaswamy, Geir Julius Braathen, Nataliya Di Donato, Elisa Rahikkala, Skadi Beblo, Martin Werber, Victor Krajka, Øyvind L. Busk, Hauke Baumann, Nouriya Abbas Al-Sannaa, Frauke Hinrichs, Rabea Affan, Nir Navot, Mohammed A. Al Balwi, Gabriela Oprea, Øystein L. Holla, Maximilian E.R. Weiss, Rami A. Jamra, Anne-Karin Kahlert, Shivendra Kishore, Kristian Tveten, Melissa Vos, Arndt Rolfs, and Katja Lohmann. De novo variants in taok1 cause neurodevelopmental disorders. The American Journal of Human Genetics, 105:213-220, Jul 2019. URL: https://doi.org/10.1016/j.ajhg.2019.05.005, doi:10.1016/j.ajhg.2019.05.005. This article has 46 citations.
35. (fang2020thediverseroles pages 15-16): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
36. (fang2020thediverseroles pages 16-17): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
37. (fang2020thediverseroles pages 19-20): Chih-Yeu Fang, Tsung-Ching Lai, Michael Hsiao, and Yu-Chan Chang. The diverse roles of tao kinases in health and diseases. International Journal of Molecular Sciences, 21:7463, Oct 2020. URL: https://doi.org/10.3390/ijms21207463, doi:10.3390/ijms21207463. This article has 60 citations and is from a peer-reviewed journal.
38. (hu2021clinicalandneurobiological pages 1-2): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
39. (hu2021clinicalandneurobiological pages 4-5): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
40. (hu2021clinicalandneurobiological pages 5-6): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
41. (hu2021clinicalandneurobiological pages 6-8): Chun Hu, Pan Feng, Qian Yang, and Lin Xiao. Clinical and neurobiological aspects of tao kinase family in neurodevelopmental disorders. Frontiers in Molecular Neuroscience, Mar 2021. URL: https://doi.org/10.3389/fnmol.2021.655037, doi:10.3389/fnmol.2021.655037. This article has 13 citations and is from a peer-reviewed journal.
42. (wernigg2025theserinethreoninekinase pages 23-27): M Wernigg. The serine/threonine kinase thousand and one amino acid kinase 2 (taok2) regulates hippo/yap signaling and synaptic activity. Unknown journal, 2025.
43. (wernigg2025theserinethreoninekinase pages 78-81): M Wernigg. The serine/threonine kinase thousand and one amino acid kinase 2 (taok2) regulates hippo/yap signaling and synaptic activity. Unknown journal, 2025.
44. (wernigg2025theserinethreoninekinase pages 88-91): M Wernigg. The serine/threonine kinase thousand and one amino acid kinase 2 (taok2) regulates hippo/yap signaling and synaptic activity. Unknown journal, 2025.