1. Phylogeny  
   Death‐associated protein kinase 1 (DAPK1) is a member of the DAPK family of calcium/calmodulin‐dependent serine/threonine kinases, which also includes DAPK2, DAPK3 (ZIP kinase), DRAK1, and DRAK2. The catalytic domain of DAPK1 is highly conserved among eukaryotes and orthologs of DAPK1 have been identified in all mammalian species. DAPK1 shares evolutionary relationships with its family members through conserved kinase subdomains, although its extra‐catalytic regions (including ankyrin repeats, ROC-COR domains, and the death domain) set it apart functionally from the other DAPK isoforms (chen2019deathassociatedproteinkinase pages 2-3, nair2013deathassociatedprotein pages 1-3, singh2016deathassociatedprotein pages 2-4).
2. Reaction Catalyzed  
   DAPK1 catalyzes the phosphorylation of serine/threonine residues on protein substrates using ATP as the phosphate donor. The generalized reaction is:  
     ATP + [protein]-(L‑serine or L‑threonine) → ADP + [protein]-(L‑serine/threonine)-phosphate + H⁺  
   This reaction transfers the γ-phosphate of ATP to the hydroxyl group of target serine or threonine residues (chen2019deathassociatedproteinkinase pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of DAPK1 depends on the presence of Mg²⁺, which coordinates ATP binding in the active site, and on Ca²⁺ in complex with calmodulin. The Ca²⁺/calmodulin binding is essential for relieving autoinhibition imposed by the autoregulatory domain and for full activation of the kinase (chen2019deathassociatedproteinkinase pages 1-2, singh2016deathassociatedprotein pages 1-2).
4. Substrate Specificity  
   DAPK1 exhibits substrate specificity toward serine/threonine residues in regulatory proteins. Its consensus substrate recognition involves the preference for sequences in which phosphorylatable serine or threonine residues are flanked by certain charged amino acids. Documented substrates include PIN1, which is phosphorylated at Ser71 leading to inhibition of its isomerase activity and altered nuclear localization; TPM1, where phosphorylation results in enhanced stress fiber formation in endothelial cells; STX1A, whose binding to STXBP1 is significantly reduced upon phosphorylation; PRKD1, whose activation via phosphorylation modulates JNK signaling under conditions of oxidative stress; BECN1, in which phosphorylation decreases its binding to the antiapoptotic proteins BCL2 and BCL2L1 and promotes autophagy; and TSC2, whose phosphorylation disrupts the TSC1–TSC2 complex and stimulates mTORC1 activity in a growth factor-dependent manner (chen2019deathassociatedproteinkinase pages 1-2, kim2014deathassociatedproteinkinase pages 1-2).
5. Structure  
   DAPK1 is a 160 kDa multidomain enzyme composed of several distinct regions that contribute to its catalytic and regulatory functions. The N-terminal catalytic domain is composed of approximately 11 conserved subdomains, with a characteristic protein kinase fold consisting of a small N-terminal lobe (rich in β-sheets) and a larger C-terminal lobe (rich in α-helices). A key residue within the ATP-binding site is Lys42, which is crucial for catalysis. Flanking the kinase domain is a Ca²⁺/calmodulin (CaM) regulatory domain that functions as an autoinhibitory segment; binding of CaM in the presence of Ca²⁺ relieves this inhibition. Following this region are eight ankyrin repeats that mediate protein–protein interactions and contribute to the localization of DAPK1 to actin microfilaments, as well as facilitating its degradation via ubiquitin–proteasome pathways. A ROC (Ras of complex proteins)-COR (C-terminal of ROC) tandem domain is present and is responsible for binding and hydrolyzing GTP, thereby modulating kinase activity through intramolecular signaling. Near the C-terminus, DAPK1 contains a death domain that mediates interactions with other apoptosis-related proteins such as FADD, TNFR, and APP, and a serine-rich tail that serves as an additional regulatory module (chen2019deathassociatedproteinkinase pages 3-4, pages 4-5, singh2016deathassociatedprotein pages 2-4).
6. Regulation  
   DAPK1 is regulated by multiple mechanisms that modulate its catalytic activity in response to cellular signals. One critical regulatory mechanism is autoinhibition mediated by the Ca²⁺/calmodulin-binding autoregulatory domain; in the absence of Ca²⁺-bound calmodulin, this domain obstructs the catalytic cleft. Autophosphorylation at Ser308 within this domain stabilizes the inhibitory conformation, and dephosphorylation by specific phosphatases such as protein phosphatase 2A (PP2A) reactivates the enzyme. In addition, phosphorylation at sites in other domains, such as Ser289 (phosphorylation by p90 ribosomal S6 kinase) and at Tyr491/Tyr492 (phosphorylation by Src family kinases), further fine-tunes kinase activity. Binding of GTP to the ROC domain also exerts negative regulation by promoting Ser308 phosphorylation via an intramolecular mechanism independent of PP2A. DAPK1 is additionally regulated post-translationally through ubiquitination mediated by E3 ubiquitin ligases, such as CHIP and DIP1, which target the protein for proteasomal degradation. These regulatory modifications coordinate to ensure that DAPK1 activity is precisely controlled in response to stress, growth factor signaling, and calcium flux (chen2019deathassociatedproteinkinase pages 4-5, farag2019death‐associatedproteinkinase pages 14-15, widau2010proteinphosphatase2a pages 1-1, singh2016deathassociatedprotein pages 10-10).
7. Function  
   DAPK1 is centrally involved in the regulation of cell death and survival pathways. It functions as a positive mediator of type I apoptotic cell death, which is caspase-dependent, and also triggers type II autophagic cell death characterized by the accumulation of autophagic vesicles. In neuronal cells, DAPK1 contributes to apoptosis by phosphorylating substrates such as PIN1, thereby inhibiting its catalytic function and altering nuclear localization, which has implications in the pathogenesis of neurodegenerative diseases such as Alzheimer’s disease. In endothelial cells, phosphorylation of TPM1 by DAPK1 enhances stress fiber formation, impacting cytoskeletal dynamics. Moreover, phosphorylation of STX1A leads to reduced binding to its partner STXBP1, and phosphorylation of PRKD1 modulates JNK signaling under oxidative stress conditions. DAPK1 also phosphorylates BECN1, weakening its interaction with the antiapoptotic proteins BCL2 and BCL2L1 and thus promoting autophagy. Phosphorylation of TSC2 disrupts the TSC1–TSC2 complex and stimulates mTORC1 activity in a growth factor-dependent context. These functions delineate a role for DAPK1 in both tumor suppression and neuronal cell death, with its activity being frequently downregulated in cancers via promoter hypermethylation and other mechanisms, while its overactivity in neurons contributes to neurodegenerative processes (chen2019deathassociatedproteinkinase pages 1-2, pages 6-7, kim2019deathassociatedproteinkinase pages 1-3, singh2016deathassociatedprotein pages 1-2, li2023ablationofdeathassociated pages 11-13).
8. Other Comments  
   DAPK1 is the subject of considerable interest as a therapeutic target due to its roles in apoptosis, autophagy, and cytoskeletal regulation. Several small molecule inhibitors have been identified through structure-based virtual screening and medicinal chemistry efforts; examples include aminopyridazine-based inhibitors and alkylated 3-amino-6-phenylpyridazine derivatives, which have been shown to reduce brain tissue loss in stroke models. Peptide-directed strategies that trigger lysosomal degradation of DAPK1 have also been explored. In parallel, natural compounds such as curcumin and grifolin can modulate DAPK1 activity. Clinically, DAPK1 is associated with various pathologies including Alzheimer’s disease, ischemic brain injury, certain cancers, and inflammatory conditions. Its overexpression in AD patients and its role in enhancing phosphorylation of tau and APP processing underline its importance as a mediator of neurodegeneration. Conversely, loss of DAPK1 expression in various cancers correlates with advanced disease stage and metastasis. These associations underscore the dual roles of DAPK1 in promoting cell death in neurons while acting as a tumor suppressor in other tissues (chen2019deathassociatedproteinkinase pages 6-7, farag2019death‐associatedproteinkinase pages 15-17, kim2019deathassociatedproteinkinase pages 7-9, yu2017deathassociatedproteinkinase pages 1-2).
9. References
10. chen2019deathassociatedproteinkinase pages 1-2
11. chen2019deathassociatedproteinkinase pages 2-3
12. chen2019deathassociatedproteinkinase pages 3-4
13. chen2019deathassociatedproteinkinase pages 4-5
14. chen2019deathassociatedproteinkinase pages 6-7
15. elbadawy2018novelfunctionsof pages 1-3
16. elbadawy2018novelfunctionsof pages 10-12
17. elbadawy2018novelfunctionsof pages 12-14
18. elbadawy2018novelfunctionsof pages 15-16
19. elbadawy2018novelfunctionsof pages 3-5
20. elbadawy2018novelfunctionsof pages 8-10
21. farag2019death‐associatedproteinkinase pages 1-2
22. farag2019death‐associatedproteinkinase pages 10-12
23. farag2019death‐associatedproteinkinase pages 12-14
24. farag2019death‐associatedproteinkinase pages 14-15
25. farag2019death‐associatedproteinkinase pages 15-17
26. farag2019death‐associatedproteinkinase pages 17-19
27. farag2019death‐associatedproteinkinase pages 19-22
28. farag2019death‐associatedproteinkinase pages 2-4
29. farag2019death‐associatedproteinkinase pages 26-28
30. farag2019death‐associatedproteinkinase pages 28-30
31. farag2019death‐associatedproteinkinase pages 31-33
32. farag2019death‐associatedproteinkinase pages 33-35
33. farag2019death‐associatedproteinkinase pages 4-6
34. farag2019death‐associatedproteinkinase pages 6-9
35. farag2019death‐associatedproteinkinase pages 9-10
36. gozuacik2006dapkproteinfamily pages 2-3
37. gozuacik2006dapkproteinfamily pages 3-4
38. gozuacik2006dapkproteinfamily pages 4-5
39. inbal2000deathassociatedproteinkinaserelated pages 1-2
40. kim2014deathassociatedproteinkinase pages 1-2
41. kim2019deathassociatedproteinkinase pages 1-3
42. kim2019deathassociatedproteinkinase pages 11-12
43. kim2019deathassociatedproteinkinase pages 12-14
44. kim2019deathassociatedproteinkinase pages 14-16
45. kim2019deathassociatedproteinkinase pages 3-5
46. kim2019deathassociatedproteinkinase pages 5-7
47. kim2019deathassociatedproteinkinase pages 7-9
48. kim2019deathassociatedproteinkinase pages 9-11
49. li2023ablationofdeathassociated pages 11-13
50. li2023ablationofdeathassociated pages 13-14
51. nair2013deathassociatedprotein pages 1-3
52. nair2013deathassociatedprotein pages 12-15
53. nair2013deathassociatedprotein pages 3-7
54. singh2016deathassociatedprotein pages 1-2
55. singh2016deathassociatedprotein pages 10-10
56. singh2016deathassociatedprotein pages 10-11
57. singh2016deathassociatedprotein pages 2-4
58. singh2016deathassociatedprotein pages 4-5
59. singh2016deathassociatedprotein pages 6-8
60. singh2016deathassociatedprotein pages 8-9
61. stevens2009peptidecombinatoriallibraries pages 1-2
62. widau2010proteinphosphatase2a pages 1-1
63. yokoyama2015structuralinsightinto pages 1-5
64. you2017deathassociatedproteinkinase pages 1-2
65. gade2014regulationofthe pages 1-2
66. farag2019death‐associatedproteinkinase pages 23-26
67. farag2019death‐associatedproteinkinase pages 30-31

References

1. (chen2019deathassociatedproteinkinase pages 1-2): Dongmei Chen, Xiao Z. Zhou, and Tae H. Lee. Death-associated protein kinase 1 as a promising drug target in cancer and alzheimer’s disease. Recent Patents on Anti-Cancer Drug Discovery, 14:144-157, Aug 2019. URL: https://doi.org/10.2174/1574892814666181218170257, doi:10.2174/1574892814666181218170257. This article has 57 citations and is from a peer-reviewed journal.
2. (chen2019deathassociatedproteinkinase pages 2-3): Dongmei Chen, Xiao Z. Zhou, and Tae H. Lee. Death-associated protein kinase 1 as a promising drug target in cancer and alzheimer’s disease. Recent Patents on Anti-Cancer Drug Discovery, 14:144-157, Aug 2019. URL: https://doi.org/10.2174/1574892814666181218170257, doi:10.2174/1574892814666181218170257. This article has 57 citations and is from a peer-reviewed journal.
3. (chen2019deathassociatedproteinkinase pages 3-4): Dongmei Chen, Xiao Z. Zhou, and Tae H. Lee. Death-associated protein kinase 1 as a promising drug target in cancer and alzheimer’s disease. Recent Patents on Anti-Cancer Drug Discovery, 14:144-157, Aug 2019. URL: https://doi.org/10.2174/1574892814666181218170257, doi:10.2174/1574892814666181218170257. This article has 57 citations and is from a peer-reviewed journal.
4. (chen2019deathassociatedproteinkinase pages 4-5): Dongmei Chen, Xiao Z. Zhou, and Tae H. Lee. Death-associated protein kinase 1 as a promising drug target in cancer and alzheimer’s disease. Recent Patents on Anti-Cancer Drug Discovery, 14:144-157, Aug 2019. URL: https://doi.org/10.2174/1574892814666181218170257, doi:10.2174/1574892814666181218170257. This article has 57 citations and is from a peer-reviewed journal.
5. (chen2019deathassociatedproteinkinase pages 6-7): Dongmei Chen, Xiao Z. Zhou, and Tae H. Lee. Death-associated protein kinase 1 as a promising drug target in cancer and alzheimer’s disease. Recent Patents on Anti-Cancer Drug Discovery, 14:144-157, Aug 2019. URL: https://doi.org/10.2174/1574892814666181218170257, doi:10.2174/1574892814666181218170257. This article has 57 citations and is from a peer-reviewed journal.
6. (elbadawy2018novelfunctionsof pages 1-3): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
7. (elbadawy2018novelfunctionsof pages 10-12): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
8. (elbadawy2018novelfunctionsof pages 12-14): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
9. (elbadawy2018novelfunctionsof pages 15-16): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
10. (elbadawy2018novelfunctionsof pages 3-5): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
11. (elbadawy2018novelfunctionsof pages 8-10): M. Elbadawy, T. Usui, H. Yamawaki, and K. Sasaki. Novel functions of death-associated protein kinases through mitogen-activated protein kinase-related signals. International Journal of Molecular Sciences, Oct 2018. URL: https://doi.org/10.3390/ijms19103031, doi:10.3390/ijms19103031. This article has 61 citations and is from a peer-reviewed journal.
12. (farag2019death‐associatedproteinkinase pages 1-2): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
13. (farag2019death‐associatedproteinkinase pages 10-12): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
14. (farag2019death‐associatedproteinkinase pages 12-14): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
15. (farag2019death‐associatedproteinkinase pages 14-15): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
16. (farag2019death‐associatedproteinkinase pages 15-17): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
17. (farag2019death‐associatedproteinkinase pages 17-19): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
18. (farag2019death‐associatedproteinkinase pages 19-22): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
19. (farag2019death‐associatedproteinkinase pages 2-4): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
20. (farag2019death‐associatedproteinkinase pages 26-28): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
21. (farag2019death‐associatedproteinkinase pages 28-30): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
22. (farag2019death‐associatedproteinkinase pages 31-33): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
23. (farag2019death‐associatedproteinkinase pages 33-35): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
24. (farag2019death‐associatedproteinkinase pages 4-6): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
25. (farag2019death‐associatedproteinkinase pages 6-9): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
26. (farag2019death‐associatedproteinkinase pages 9-10): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
27. (gozuacik2006dapkproteinfamily pages 2-3): Devrim Gozuacik and Adi Kimchi. Dapk protein family and cancer. Autophagy, 2:74-79, Jan 2006. URL: https://doi.org/10.4161/auto.2.2.2459, doi:10.4161/auto.2.2.2459. This article has 254 citations and is from a domain leading peer-reviewed journal.
28. (gozuacik2006dapkproteinfamily pages 3-4): Devrim Gozuacik and Adi Kimchi. Dapk protein family and cancer. Autophagy, 2:74-79, Jan 2006. URL: https://doi.org/10.4161/auto.2.2.2459, doi:10.4161/auto.2.2.2459. This article has 254 citations and is from a domain leading peer-reviewed journal.
29. (gozuacik2006dapkproteinfamily pages 4-5): Devrim Gozuacik and Adi Kimchi. Dapk protein family and cancer. Autophagy, 2:74-79, Jan 2006. URL: https://doi.org/10.4161/auto.2.2.2459, doi:10.4161/auto.2.2.2459. This article has 254 citations and is from a domain leading peer-reviewed journal.
30. (inbal2000deathassociatedproteinkinaserelated pages 1-2): Boaz Inbal, Gidi Shani, Ofer Cohen, Joseph L. Kissil, and Adi Kimchi. Death-associated protein kinase-related protein 1, a novel serine/threonine kinase involved in apoptosis. Molecular and Cellular Biology, 20:1044-1054, Feb 2000. URL: https://doi.org/10.1128/mcb.20.3.1044-1054.2000, doi:10.1128/mcb.20.3.1044-1054.2000. This article has 201 citations and is from a domain leading peer-reviewed journal.
31. (kim2014deathassociatedproteinkinase pages 1-2): B. M. Kim, M. You, C. Chen, S. Lee, Y. Hong, Y. Hong, A. Kimchi, X. Z. Zhou, and T. H. Lee. Death-associated protein kinase 1 has a critical role in aberrant tau protein regulation and function. Cell Death & Disease, May 2014. URL: https://doi.org/10.1038/cddis.2014.216, doi:10.1038/cddis.2014.216. This article has 104 citations.
32. (kim2019deathassociatedproteinkinase pages 1-3): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
33. (kim2019deathassociatedproteinkinase pages 11-12): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
34. (kim2019deathassociatedproteinkinase pages 12-14): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
35. (kim2019deathassociatedproteinkinase pages 14-16): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
36. (kim2019deathassociatedproteinkinase pages 3-5): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
37. (kim2019deathassociatedproteinkinase pages 5-7): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
38. (kim2019deathassociatedproteinkinase pages 7-9): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
39. (kim2019deathassociatedproteinkinase pages 9-11): Nami Kim, Dongmei Chen, Xiao Zhen Zhou, and Tae Ho Lee. Death-associated protein kinase 1 phosphorylation in neuronal cell death and neurodegenerative disease. International Journal of Molecular Sciences, 20:3131, Jun 2019. URL: https://doi.org/10.3390/ijms20133131, doi:10.3390/ijms20133131. This article has 77 citations and is from a peer-reviewed journal.
40. (li2023ablationofdeathassociated pages 11-13): Ruomeng Li, Shuai Zhi, Guihua Lan, Xiaotong Chen, Xiuzhi Zheng, Li Hu, Long Wang, Tao Zhang, Tae Ho Lee, Shitao Rao, and Dongmei Chen. Ablation of death-associated protein kinase 1 changes the transcriptomic profile and alters neural-related pathways in the brain. International Journal of Molecular Sciences, 24:6542, Mar 2023. URL: https://doi.org/10.3390/ijms24076542, doi:10.3390/ijms24076542. This article has 6 citations and is from a peer-reviewed journal.
41. (li2023ablationofdeathassociated pages 13-14): Ruomeng Li, Shuai Zhi, Guihua Lan, Xiaotong Chen, Xiuzhi Zheng, Li Hu, Long Wang, Tao Zhang, Tae Ho Lee, Shitao Rao, and Dongmei Chen. Ablation of death-associated protein kinase 1 changes the transcriptomic profile and alters neural-related pathways in the brain. International Journal of Molecular Sciences, 24:6542, Mar 2023. URL: https://doi.org/10.3390/ijms24076542, doi:10.3390/ijms24076542. This article has 6 citations and is from a peer-reviewed journal.
42. (nair2013deathassociatedprotein pages 1-3): Syam Nair, Henrik Hagberg, Rajanikant Krishnamurthy, Claire Thornton, and Carina Mallard. Death associated protein kinases: molecular structure and brain injury. International Journal of Molecular Sciences, 14:13858-13872, Jul 2013. URL: https://doi.org/10.3390/ijms140713858, doi:10.3390/ijms140713858. This article has 56 citations and is from a peer-reviewed journal.
43. (nair2013deathassociatedprotein pages 12-15): Syam Nair, Henrik Hagberg, Rajanikant Krishnamurthy, Claire Thornton, and Carina Mallard. Death associated protein kinases: molecular structure and brain injury. International Journal of Molecular Sciences, 14:13858-13872, Jul 2013. URL: https://doi.org/10.3390/ijms140713858, doi:10.3390/ijms140713858. This article has 56 citations and is from a peer-reviewed journal.
44. (nair2013deathassociatedprotein pages 3-7): Syam Nair, Henrik Hagberg, Rajanikant Krishnamurthy, Claire Thornton, and Carina Mallard. Death associated protein kinases: molecular structure and brain injury. International Journal of Molecular Sciences, 14:13858-13872, Jul 2013. URL: https://doi.org/10.3390/ijms140713858, doi:10.3390/ijms140713858. This article has 56 citations and is from a peer-reviewed journal.
45. (singh2016deathassociatedprotein pages 1-2): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
46. (singh2016deathassociatedprotein pages 10-10): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
47. (singh2016deathassociatedprotein pages 10-11): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
48. (singh2016deathassociatedprotein pages 2-4): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
49. (singh2016deathassociatedprotein pages 4-5): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
50. (singh2016deathassociatedprotein pages 6-8): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
51. (singh2016deathassociatedprotein pages 8-9): Pratibha Singh, Palaniyandi Ravanan, and Priti Talwar. Death associated protein kinase 1 (dapk1): a regulator of apoptosis and autophagy. Frontiers in Molecular Neuroscience, Jun 2016. URL: https://doi.org/10.3389/fnmol.2016.00046, doi:10.3389/fnmol.2016.00046. This article has 210 citations and is from a peer-reviewed journal.
52. (stevens2009peptidecombinatoriallibraries pages 1-2): C. Stevens, Yao-neng Lin, B. Harrison, L. Burch, R. Ridgway, O. Sansom, and T. Hupp. Peptide combinatorial libraries identify tsc2 as a death-associated protein kinase (dapk) death domain-binding protein and reveal a stimulatory role for dapk in mtorc1 signaling\*. Journal of Biological Chemistry, 284:334-344, Jan 2009. URL: https://doi.org/10.1074/jbc.m805165200, doi:10.1074/jbc.m805165200. This article has 98 citations and is from a domain leading peer-reviewed journal.
53. (widau2010proteinphosphatase2a pages 1-1): Ryan C. Widau, Yijun Jin, Shelley A. Dixon, Brian E. Wadzinski, and Patricia J. Gallagher. Protein phosphatase 2a (pp2a) holoenzymes regulate death-associated protein kinase (dapk) in ceramide-induced anoikis. Journal of Biological Chemistry, 285:13827-13838, Apr 2010. URL: https://doi.org/10.1074/jbc.m109.085076, doi:10.1074/jbc.m109.085076. This article has 78 citations and is from a domain leading peer-reviewed journal.
54. (yokoyama2015structuralinsightinto pages 1-5): Takeshi Yokoyama, Yuto Kosaka, and Mineyuki Mizuguchi. Structural insight into the interactions between death-associated protein kinase 1 and natural flavonoids. Journal of Medicinal Chemistry, 58:7400-7408, Sep 2015. URL: https://doi.org/10.1021/acs.jmedchem.5b00893, doi:10.1021/acs.jmedchem.5b00893. This article has 85 citations and is from a highest quality peer-reviewed journal.
55. (you2017deathassociatedproteinkinase pages 1-2): M. You, B. Kim, Chun-Hau Chen, M. Begley, L. Cantley, and T. Lee. Death-associated protein kinase 1 phosphorylates ndrg2 and induces neuronal cell death. Cell Death and Differentiation, 24:238-250, Nov 2017. URL: https://doi.org/10.1038/cdd.2016.114, doi:10.1038/cdd.2016.114. This article has 54 citations and is from a domain leading peer-reviewed journal.
56. (farag2019death‐associatedproteinkinase pages 23-26): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
57. (farag2019death‐associatedproteinkinase pages 30-31): Ahmed Karam Farag and Eun Joo Roh. Death‐associated protein kinase (dapk) family modulators: current and future therapeutic outcomes. Medicinal Research Reviews, 39:349-385, Jun 2019. URL: https://doi.org/10.1002/med.21518, doi:10.1002/med.21518. This article has 116 citations and is from a domain leading peer-reviewed journal.
58. (gade2014regulationofthe pages 1-2): Padmaja Gade, Srikanta B. Manjegowda, Shreeram C. Nallar, Uday B. Maachani, Alan S. Cross, and Dhananjaya V. Kalvakolanu. Regulation of the death-associated protein kinase 1 expression and autophagy via atf6 requires apoptosis signal-regulating kinase 1. Molecular and Cellular Biology, 34:4033-4048, Nov 2014. URL: https://doi.org/10.1128/mcb.00397-14, doi:10.1128/mcb.00397-14. This article has 80 citations and is from a domain leading peer-reviewed journal.