1. Phylogeny  
   Cyclin-dependent kinase 5 (CDK5) is an atypical member of the cyclin-dependent kinase family that—unlike its classical cell‐cycle counterparts—is primarily restricted to postmitotic neuronal tissues. CDK5 is conserved across vertebrates and is found in species ranging from rodents to humans, indicating that its critical functions in neural development and maintenance have been maintained during evolution (dhariwala2008anunusualmember pages 1-3, su2011cyclindependentkinasesin pages 1-3). Unlike the classical CDKs that cluster with regulators of cell cycle transitions, CDK5 groups phylogenetically with kinases that evolved specialized roles in postmitotic cells. This specialization is supported by the evolutionary studies of the protein kinase complement of the human genome and analyses of kinase family evolution, which show that while CDK5 shares approximately 60% sequence homology with kinases such as CDK1 and CDK2, it has diverged in its regulatory mechanism by adopting neuron‐specific activators rather than canonical cyclins (lopes2011cdk5multitaskingbetween pages 1-6, su2011cyclindependentkinasesin pages 3-5). In addition, phylogenetic surveys based on datasets from Manning et al. reveal that CDK5 occupies a unique branch of the kinome that is separate from cyclin-dependent kinases involved in cell cycle control, consistent with its distinct functions in neuronal signaling and cytoskeletal dynamics.
2. Reaction Catalyzed  
   The reaction catalyzed by CDK5 follows the typical mechanism of protein kinases. In the presence of Mg²⁺ as a cofactor, CDK5 transfers a phosphate group from ATP onto the hydroxyl group of serine or threonine residues on target proteins that are followed immediately by a proline residue. The overall reaction can be summarized as follows:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (dhariwala2008anunusualmember pages 1-3, su2011cyclindependentkinasesin pages 1-3).
3. Cofactor Requirements  
   CDK5 requires divalent metal ions, most notably Mg²⁺, as a critical cofactor for its kinase activity. The presence of Mg²⁺ facilitates the proper alignment of ATP in the active site of CDK5, thereby enabling efficient catalysis of the phosphate transfer reaction (dhariwala2008anunusualmember pages 1-3, su2011cyclindependentkinasesin pages 1-3).
4. Substrate Specificity  
   CDK5 is a proline-directed serine/threonine kinase that exhibits a distinct substrate specificity primarily recognizing a consensus motif in which a serine or threonine residue is immediately followed by a proline (S/TP motif). Studies have indicated that substrates are often further defined by the presence of basic residues, such as lysine or arginine, at the +3 position relative to the phosphorylated site (dhariwala2008anunusualmember pages 1-3, lopes2011cdk5multitaskingbetween pages 1-6). Recent large-scale analyses of substrate specificities within the human serine/threonine kinome further support the notion that CDK5 preferentially phosphorylates sites that conform to such proline-directed motifs (Johnson2023Journal pages 759-766). Although CDK5 is not classified as a tyrosine kinase, considerations of tyrosine kinase substrate preferences from studies by Yaron-Barir2024Journal provide complementary insights into kinase specificity landscapes; however, for CDK5 the defining consensus remains the (S/T)P motif. Hence, substrates that contain sequences such as –R/K–X–(S/T)–P–X–(K/R) are often phosphorylated by CDK5 (dhariwala2008anunusualmember pages 1-3, Johnson2023Journal pages 759-766).
5. Structure  
   The three-dimensional structure of CDK5 is organized around a central kinase domain that is typical of the cyclin-dependent kinase family. This kinase domain is composed of a smaller N-terminal lobe, rich in β-strands, and a larger C-terminal lobe that is predominantly α-helical. The active site, which includes an ATP-binding pocket, is formed at the interface between these two lobes and contains critical features such as a glycine-rich loop that stabilizes the phosphate groups of ATP and an activation loop that is central to substrate alignment (mapelli2003thestructuralperspective pages 5-6, lim2003proteinproteininteractionsin pages 3-4). Unlike classical CDKs, activation of CDK5 does not require phosphorylation of its activation loop. Instead, CDK5 is activated upon binding to specific non-cyclin regulatory proteins such as p35 and p39. Structural studies have demonstrated that these activators—despite lacking overall sequence similarity to cyclins—adopt a cyclin-like fold that supports the conformational rearrangements needed for full kinase activity (dhariwala2008anunusualmember pages 1-3, mapelli2005mechanismofcdk5p25 pages 1-2). Furthermore, cleavage of p35 by calpain generates p25, whose binding leads to an open activation loop conformation that compromises normal subcellular localization and alters substrate specificity (mapelli2005mechanismofcdk5p25 pages 8-9, lim2003proteinproteininteractionsin pages 8-8). Key catalytic features include a conserved lysine residue in the ATP-binding region that forms a salt bridge with a glutamic acid in the C-helix and a hydrophobic spine that contributes to structural stability and the proper positioning of the substrate (mapelli2003thestructuralperspective pages 6-8, malumbres2014cyclindependentkinases pages 5-6).
6. Regulation  
   The regulation of CDK5 is unique among the cyclin-dependent kinases. Rather than relying on periodic expression of cyclin partners and phosphorylation of a T-loop for activation, CDK5 is controlled almost exclusively through its interaction with neuron-specific regulatory subunits p35 and p39, as well as the pathological p25 fragment derived from p35 by calpain-mediated proteolysis (dhariwala2008anunusualmember pages 1-3, lim2003proteinproteininteractionsin pages 3-4). Binding of these activators induces a conformational change in CDK5 that results in full catalytic activation without the need for activating phosphorylation on the T-loop (mapelli2003thestructuralperspective pages 5-6, sharma2020akinaseof pages 1-2). Additionally, CDK5 activity is modulated by phosphorylation at specific residues. For instance, phosphorylation at Tyr15—catalyzed by non-receptor tyrosine kinases such as c-Abl and Fyn—enhances its activity, while phosphorylation at Thr14 can have inhibitory effects (dhariwala2008anunusualmember pages 3-5, sharma2020akinaseof pages 3-4). CDK5 is further regulated by negative feedback mechanisms; for example, autophosphorylation events on its activator p35 enhance its stability by preventing calpain-mediated cleavage. Conversely, cleavage of p35 to p25 disrupts membrane targeting and prolongs the kinase’s activity, contributing to aberrant phosphorylation of substrates implicated in neurodegenerative disorders (dhariwala2008anunusualmember pages 6-8, roufayel2019cdk5keyregulator pages 5-7). Regulation also occurs via protein–protein interactions with additional modulators such as cyclin I in non-neuronal cells, which can influence apoptotic and pro-survival signaling pathways (roufayel2019cdk5keyregulator pages 9-11, shupp2017biologicalfunctionsof pages 4-6).
7. Function  
   CDK5 plays an essential role in multiple neuronal processes as well as in some non-neuronal functions. In the central nervous system, CDK5 is critical for neuronal migration, neurite outgrowth, axon guidance, and dendritic spine morphogenesis. It accomplishes these tasks by phosphorylating a broad array of substrates involved in cytoskeletal dynamics, such as microtubule-associated proteins (MAPT/TAU, MAP1B, and MAP2) and regulators of actin remodeling (PAK1, RAC1, RHOA, and CDC42) (dhariwala2008anunusualmember pages 1-3, lopes2011cdk5multitaskingbetween pages 1-6). In synaptic terminals, CDK5 modulates neurotransmitter release by phosphorylating proteins that govern both exocytosis (e.g., SYN1, MUNC18, AMPH) and endocytosis processes (e.g., DNM1 and SYNJ1). In addition, CDK5 regulates cell survival by phosphorylating factors such as p53 and by activating anti-apoptotic proteins including BCL2 and STAT3, thereby influencing the balance between cell death and survival in neurons experiencing genotoxic or oxidative stress (roufayel2019cdk5keyregulator pages 7-9, su2011cyclindependentkinasesin pages 3-5). Beyond neuronal cells, CDK5 is expressed in pancreatic beta cells, where it has been shown to regulate insulin secretion through modulation of calcium channel activity; it also plays roles in immune cells, endothelial cells, and certain cancer cell types, affecting proliferation, migration, and angiogenesis (sharma2020akinaseof pages 1-2, pozo2016theemergingrole pages 1-3). Moreover, CDK5 has been found to negatively regulate the Wnt/β-catenin signaling pathway and to activate the GAIT pathway in myeloid cells, further demonstrating its involvement in diverse cellular signaling networks (dhariwala2008anunusualmember pages 1-3, peyressatre2015targetingcyclindependentkinases pages 4-6).
8. Other Comments  
   Several inhibitors targeting CDK5 have been identified, including small molecules such as roscovitine (also known as seliciclib) and olomoucine, which inhibit its kinase activity by competing for the ATP-binding site; however, these compounds generally lack specificity due to overlapping inhibition of other CDKs (shupp2017biologicalfunctionsof pages 7-8, sharma2020akinaseof pages 8-9). In addition, peptide inhibitors like the CDK5 inhibitory peptide (CIP), which is derived from the p35 activator domain, have been developed to selectively block the hyperactive CDK5/p25 complex without affecting physiological CDK5/p35 function, thereby offering potential therapeutic benefits in neurodegenerative conditions such as Alzheimer’s disease (shah2018taleofthe pages 1-3, shupp2017biologicalfunctionsof pages 8-9). Disease associations for CDK5 include neurodegenerative disorders—where deregulated kinase activity, particularly through the generation of p25, leads to abnormal phosphorylation of tau and other substrates—and certain cancers, in which CDK5 overexpression or hyperactivation contributes to tumor cell proliferation, migration, angiogenesis, and immune escape (pozo2016theemergingrole pages 12-13, shupp2017biologicalfunctionsof pages 7-8). Notable substrates of CDK5 in the context of disease are microtubule-binding proteins such as MAPT/TAU, whose hyperphosphorylation is a hallmark of Alzheimer’s pathology, as well as critical regulators of apoptosis like p53 (dhariwala2008anunusualmember pages 6-8, roufayel2019cdk5keyregulator pages 9-11). In addition, CDK5’s involvement in the regulation of neurotransmitter release and synaptic plasticity underscores its potential as a target in neurological disorders that feature synapse dysfunction.  
   Furthermore, CDK5 has been implicated in the regulation of circadian rhythms through phosphorylation of clock proteins, and it modulates endothelial cell migration and angiogenesis via substrates such as PTK2/FAK1 (dhariwala2008anunusualmember pages 6-8, su2011cyclindependentkinasesin pages 3-5). These diverse roles, together with its unique regulation by non-cyclin activators and the differential outcomes of its hyperactive versus physiological states, establish CDK5 as a multifaceted kinase of considerable therapeutic and investigative interest.
9. References  
   dhariwala2008anunusualmember pages 1-3; dhariwala2008anunusualmember pages 3-5; dhariwala2008anunusualmember pages 6-8; lopes2011cdk5multitaskingbetween pages 1-6; lopes2011cdk5multitaskingbetween pages 6-9; mapelli2005mechanismofcdk5p25 pages 1-2; mapelli2005mechanismofcdk5p25 pages 8-9; pao2021threedecadesof pages 3-4; peyressatre2015targetingcyclindependentkinases pages 4-6; peyressatre2015targetingcyclindependentkinases pages 6-8; roufayel2019cdk5keyregulator pages 1-3; roufayel2019cdk5keyregulator pages 3-5; roufayel2019cdk5keyregulator pages 5-7; roufayel2019cdk5keyregulator pages 7-9; roufayel2019cdk5keyregulator pages 9-11; sharma2020akinaseof pages 1-2; sharma2020akinaseof pages 3-4; sharma2020akinaseof pages 8-9; sharma2020akinaseof pages 9-10; shupp2017biologicalfunctionsof pages 1-2; shupp2017biologicalfunctionsof pages 4-6; shupp2017biologicalfunctionsof pages 7-8; shupp2017biologicalfunctionsof pages 8-9; su2011cyclindependentkinasesin pages 1-3; su2011cyclindependentkinasesin pages 3-5; lim2003proteinproteininteractionsin pages 3-4; lim2003proteinproteininteractionsin pages 4-5; lim2003proteinproteininteractionsin pages 6-7; lim2003proteinproteininteractionsin pages 8-8; mapelli2003thestructuralperspective pages 5-6; mapelli2003thestructuralperspective pages 6-8; mapelli2003thestructuralperspective pages 8-8; mapelli2003thestructuralperspective pages 8-9; mclinden2012atthefulcrum pages 1-3; mclinden2012atthefulcrum pages 14-16; mushtaq2016neuroprotectivemechanismsmediated pages 1-3; pao2021threedecadesof pages 1-3; pao2021threedecadesof pages 13-14; peyressatre2015targetingcyclindependentkinases pages 32-34; pozo2016theemergingrole pages 1-3; pozo2016theemergingrole pages 12-13; pozo2016theemergingrole pages 20-21; rosales2006extraneuronalrolesof pages 9-10; shah2018taleofthe pages 1-3; shah2018taleofthe pages 10-12; smith2001cdk5onthe pages 1-2.  
   Additional substrate specificity details are supported by Johnson, J. L. et al. (2023) and Yaron-Barir, T. M. et al. (2024).  
   For phylogenetic context, see Manning, G. et al. (2002) in Science and Trends in Biochemical Sciences.

References

1. (dhariwala2008anunusualmember pages 1-3): Fatema A. Dhariwala and Medha S. Rajadhyaksha. An unusual member of the cdk family: cdk5. Cellular and Molecular Neurobiology, 28:351-369, Jan 2008. URL: https://doi.org/10.1007/s10571-007-9242-1, doi:10.1007/s10571-007-9242-1. This article has 237 citations and is from a peer-reviewed journal.
2. (lopes2011cdk5multitaskingbetween pages 1-6): Joao P. Lopes and Paula Agostinho. Cdk5: multitasking between physiological and pathological conditions. Progress in Neurobiology, 94:49-63, Jun 2011. URL: https://doi.org/10.1016/j.pneurobio.2011.03.006, doi:10.1016/j.pneurobio.2011.03.006. This article has 153 citations and is from a domain leading peer-reviewed journal.
3. (mapelli2005mechanismofcdk5p25 pages 1-2): Marina Mapelli, Lucia Massimiliano, Claudia Crovace, Markus A. Seeliger, Li-Huei Tsai, Laurent Meijer, and Andrea Musacchio. Mechanism of cdk5/p25 binding by cdk inhibitors. Journal of Medicinal Chemistry, 48:671-679, Jan 2005. URL: https://doi.org/10.1021/jm049323m, doi:10.1021/jm049323m. This article has 251 citations and is from a highest quality peer-reviewed journal.
4. (pao2021threedecadesof pages 3-4): Ping-Chieh Pao and Li-Huei Tsai. Three decades of cdk5. Journal of Biomedical Science, Nov 2021. URL: https://doi.org/10.1186/s12929-021-00774-y, doi:10.1186/s12929-021-00774-y. This article has 116 citations and is from a domain leading peer-reviewed journal.
5. (peyressatre2015targetingcyclindependentkinases pages 4-6): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
6. (peyressatre2015targetingcyclindependentkinases pages 6-8): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
7. (roufayel2019cdk5keyregulator pages 3-5): Rabih Roufayel and Nimer Murshid. Cdk5: key regulator of apoptosis and cell survival. Biomedicines, 7:88, Nov 2019. URL: https://doi.org/10.3390/biomedicines7040088, doi:10.3390/biomedicines7040088. This article has 56 citations and is from a peer-reviewed journal.
8. (roufayel2019cdk5keyregulator pages 7-9): Rabih Roufayel and Nimer Murshid. Cdk5: key regulator of apoptosis and cell survival. Biomedicines, 7:88, Nov 2019. URL: https://doi.org/10.3390/biomedicines7040088, doi:10.3390/biomedicines7040088. This article has 56 citations and is from a peer-reviewed journal.
9. (sharma2020akinaseof pages 3-4): Samanta Sharma and Piotr Sicinski. A kinase of many talents: non-neuronal functions of cdk5 in development and disease. Open Biology, 10:190287, Jan 2020. URL: https://doi.org/10.1098/rsob.190287, doi:10.1098/rsob.190287. This article has 51 citations and is from a peer-reviewed journal.
10. (shupp2017biologicalfunctionsof pages 4-6): Alison Shupp, Mathew C. Casimiro, and Richard G. Pestell. Biological functions of cdk5 and potential cdk5 targeted clinical treatments. Oncotarget, 8:17373-17382, Jan 2017. URL: https://doi.org/10.18632/oncotarget.14538, doi:10.18632/oncotarget.14538. This article has 122 citations and is from a poor quality or predatory journal.
11. (su2011cyclindependentkinasesin pages 1-3): Susan C. Su and Li-Huei Tsai. Cyclin-dependent kinases in brain development and disease. Annual Review of Cell and Developmental Biology, 27:465-491, Nov 2011. URL: https://doi.org/10.1146/annurev-cellbio-092910-154023, doi:10.1146/annurev-cellbio-092910-154023. This article has 356 citations and is from a domain leading peer-reviewed journal.
12. (su2011cyclindependentkinasesin pages 3-5): Susan C. Su and Li-Huei Tsai. Cyclin-dependent kinases in brain development and disease. Annual Review of Cell and Developmental Biology, 27:465-491, Nov 2011. URL: https://doi.org/10.1146/annurev-cellbio-092910-154023, doi:10.1146/annurev-cellbio-092910-154023. This article has 356 citations and is from a domain leading peer-reviewed journal.
13. (dhariwala2008anunusualmember pages 3-5): Fatema A. Dhariwala and Medha S. Rajadhyaksha. An unusual member of the cdk family: cdk5. Cellular and Molecular Neurobiology, 28:351-369, Jan 2008. URL: https://doi.org/10.1007/s10571-007-9242-1, doi:10.1007/s10571-007-9242-1. This article has 237 citations and is from a peer-reviewed journal.
14. (dhariwala2008anunusualmember pages 6-8): Fatema A. Dhariwala and Medha S. Rajadhyaksha. An unusual member of the cdk family: cdk5. Cellular and Molecular Neurobiology, 28:351-369, Jan 2008. URL: https://doi.org/10.1007/s10571-007-9242-1, doi:10.1007/s10571-007-9242-1. This article has 237 citations and is from a peer-reviewed journal.
15. (lim2003proteinproteininteractionsin pages 3-4): Anthony C.B. Lim, Dianbo Qu, and Robert Z. Qi. Protein-protein interactions in cdk5 regulation and function. Neurosignals, 12:230-238, Sep 2003. URL: https://doi.org/10.1159/000074625, doi:10.1159/000074625. This article has 42 citations and is from a peer-reviewed journal.
16. (lim2003proteinproteininteractionsin pages 4-5): Anthony C.B. Lim, Dianbo Qu, and Robert Z. Qi. Protein-protein interactions in cdk5 regulation and function. Neurosignals, 12:230-238, Sep 2003. URL: https://doi.org/10.1159/000074625, doi:10.1159/000074625. This article has 42 citations and is from a peer-reviewed journal.
17. (lim2003proteinproteininteractionsin pages 6-7): Anthony C.B. Lim, Dianbo Qu, and Robert Z. Qi. Protein-protein interactions in cdk5 regulation and function. Neurosignals, 12:230-238, Sep 2003. URL: https://doi.org/10.1159/000074625, doi:10.1159/000074625. This article has 42 citations and is from a peer-reviewed journal.
18. (lim2003proteinproteininteractionsin pages 8-8): Anthony C.B. Lim, Dianbo Qu, and Robert Z. Qi. Protein-protein interactions in cdk5 regulation and function. Neurosignals, 12:230-238, Sep 2003. URL: https://doi.org/10.1159/000074625, doi:10.1159/000074625. This article has 42 citations and is from a peer-reviewed journal.
19. (lopes2011cdk5multitaskingbetween pages 6-9): Joao P. Lopes and Paula Agostinho. Cdk5: multitasking between physiological and pathological conditions. Progress in Neurobiology, 94:49-63, Jun 2011. URL: https://doi.org/10.1016/j.pneurobio.2011.03.006, doi:10.1016/j.pneurobio.2011.03.006. This article has 153 citations and is from a domain leading peer-reviewed journal.
20. (malumbres2014cyclindependentkinases pages 5-6): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1880 citations and is from a highest quality peer-reviewed journal.
21. (mapelli2003thestructuralperspective pages 5-6): Marina Mapelli and Andrea Musacchio. The structural perspective on cdk5. Neurosignals, 12:164-172, Jan 2003. URL: https://doi.org/10.1159/000074617, doi:10.1159/000074617. This article has 49 citations and is from a peer-reviewed journal.
22. (mapelli2003thestructuralperspective pages 6-8): Marina Mapelli and Andrea Musacchio. The structural perspective on cdk5. Neurosignals, 12:164-172, Jan 2003. URL: https://doi.org/10.1159/000074617, doi:10.1159/000074617. This article has 49 citations and is from a peer-reviewed journal.
23. (mapelli2003thestructuralperspective pages 8-8): Marina Mapelli and Andrea Musacchio. The structural perspective on cdk5. Neurosignals, 12:164-172, Jan 2003. URL: https://doi.org/10.1159/000074617, doi:10.1159/000074617. This article has 49 citations and is from a peer-reviewed journal.
24. (mapelli2003thestructuralperspective pages 8-9): Marina Mapelli and Andrea Musacchio. The structural perspective on cdk5. Neurosignals, 12:164-172, Jan 2003. URL: https://doi.org/10.1159/000074617, doi:10.1159/000074617. This article has 49 citations and is from a peer-reviewed journal.
25. (mapelli2005mechanismofcdk5p25 pages 8-9): Marina Mapelli, Lucia Massimiliano, Claudia Crovace, Markus A. Seeliger, Li-Huei Tsai, Laurent Meijer, and Andrea Musacchio. Mechanism of cdk5/p25 binding by cdk inhibitors. Journal of Medicinal Chemistry, 48:671-679, Jan 2005. URL: https://doi.org/10.1021/jm049323m, doi:10.1021/jm049323m. This article has 251 citations and is from a highest quality peer-reviewed journal.
26. (mclinden2012atthefulcrum pages 1-3): K. McLinden, S. Trunova, and E. Giniger. At the fulcrum in health and disease: cdk5 and the balancing acts of neuronal structure and physiology. Brain disorders & therapy, Jul 2012. URL: https://doi.org/10.4172/2168-975x.s1-001, doi:10.4172/2168-975x.s1-001. This article has 35 citations.
27. (mclinden2012atthefulcrum pages 14-16): K. McLinden, S. Trunova, and E. Giniger. At the fulcrum in health and disease: cdk5 and the balancing acts of neuronal structure and physiology. Brain disorders & therapy, Jul 2012. URL: https://doi.org/10.4172/2168-975x.s1-001, doi:10.4172/2168-975x.s1-001. This article has 35 citations.
28. (mushtaq2016neuroprotectivemechanismsmediated pages 1-3): Gohar Mushtaq, Nigel H. Greig, Firoz Anwar, Fahad A. Al-Abbasi, Mazin A. Zamzami, Hasan A. Al-Talhi, and Mohammad A. Kamal. Neuroprotective mechanisms mediated by cdk5 inhibition. Current Pharmaceutical Design, 22:527-534, Jan 2016. URL: https://doi.org/10.2174/1381612822666151124235028, doi:10.2174/1381612822666151124235028. This article has 70 citations and is from a peer-reviewed journal.
29. (pao2021threedecadesof pages 1-3): Ping-Chieh Pao and Li-Huei Tsai. Three decades of cdk5. Journal of Biomedical Science, Nov 2021. URL: https://doi.org/10.1186/s12929-021-00774-y, doi:10.1186/s12929-021-00774-y. This article has 116 citations and is from a domain leading peer-reviewed journal.
30. (pao2021threedecadesof pages 13-14): Ping-Chieh Pao and Li-Huei Tsai. Three decades of cdk5. Journal of Biomedical Science, Nov 2021. URL: https://doi.org/10.1186/s12929-021-00774-y, doi:10.1186/s12929-021-00774-y. This article has 116 citations and is from a domain leading peer-reviewed journal.
31. (peyressatre2015targetingcyclindependentkinases pages 32-34): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
32. (pozo2016theemergingrole pages 1-3): Karine Pozo and James A. Bibb. The emerging role of cdk5 in cancer. Trends in Cancer, 2:606-618, Oct 2016. URL: https://doi.org/10.1016/j.trecan.2016.09.001, doi:10.1016/j.trecan.2016.09.001. This article has 197 citations and is from a peer-reviewed journal.
33. (pozo2016theemergingrole pages 12-13): Karine Pozo and James A. Bibb. The emerging role of cdk5 in cancer. Trends in Cancer, 2:606-618, Oct 2016. URL: https://doi.org/10.1016/j.trecan.2016.09.001, doi:10.1016/j.trecan.2016.09.001. This article has 197 citations and is from a peer-reviewed journal.
34. (pozo2016theemergingrole pages 20-21): Karine Pozo and James A. Bibb. The emerging role of cdk5 in cancer. Trends in Cancer, 2:606-618, Oct 2016. URL: https://doi.org/10.1016/j.trecan.2016.09.001, doi:10.1016/j.trecan.2016.09.001. This article has 197 citations and is from a peer-reviewed journal.
35. (rosales2006extraneuronalrolesof pages 9-10): Jesusa L. Rosales and Ki‐Young Lee. Extraneuronal roles of cyclin‐dependent kinase 5. BioEssays, Oct 2006. URL: https://doi.org/10.1002/bies.20473, doi:10.1002/bies.20473. This article has 155 citations and is from a peer-reviewed journal.
36. (roufayel2019cdk5keyregulator pages 1-3): Rabih Roufayel and Nimer Murshid. Cdk5: key regulator of apoptosis and cell survival. Biomedicines, 7:88, Nov 2019. URL: https://doi.org/10.3390/biomedicines7040088, doi:10.3390/biomedicines7040088. This article has 56 citations and is from a peer-reviewed journal.
37. (roufayel2019cdk5keyregulator pages 5-7): Rabih Roufayel and Nimer Murshid. Cdk5: key regulator of apoptosis and cell survival. Biomedicines, 7:88, Nov 2019. URL: https://doi.org/10.3390/biomedicines7040088, doi:10.3390/biomedicines7040088. This article has 56 citations and is from a peer-reviewed journal.
38. (roufayel2019cdk5keyregulator pages 9-11): Rabih Roufayel and Nimer Murshid. Cdk5: key regulator of apoptosis and cell survival. Biomedicines, 7:88, Nov 2019. URL: https://doi.org/10.3390/biomedicines7040088, doi:10.3390/biomedicines7040088. This article has 56 citations and is from a peer-reviewed journal.
39. (shah2018taleofthe pages 1-3): Kavita Shah and Sandra Rossie. Tale of the good and the bad cdk5: remodeling of the actin cytoskeleton in the brain. Molecular Neurobiology, 55:3426-3438, May 2018. URL: https://doi.org/10.1007/s12035-017-0525-3, doi:10.1007/s12035-017-0525-3. This article has 103 citations and is from a peer-reviewed journal.
40. (shah2018taleofthe pages 10-12): Kavita Shah and Sandra Rossie. Tale of the good and the bad cdk5: remodeling of the actin cytoskeleton in the brain. Molecular Neurobiology, 55:3426-3438, May 2018. URL: https://doi.org/10.1007/s12035-017-0525-3, doi:10.1007/s12035-017-0525-3. This article has 103 citations and is from a peer-reviewed journal.
41. (sharma2020akinaseof pages 1-2): Samanta Sharma and Piotr Sicinski. A kinase of many talents: non-neuronal functions of cdk5 in development and disease. Open Biology, 10:190287, Jan 2020. URL: https://doi.org/10.1098/rsob.190287, doi:10.1098/rsob.190287. This article has 51 citations and is from a peer-reviewed journal.
42. (sharma2020akinaseof pages 8-9): Samanta Sharma and Piotr Sicinski. A kinase of many talents: non-neuronal functions of cdk5 in development and disease. Open Biology, 10:190287, Jan 2020. URL: https://doi.org/10.1098/rsob.190287, doi:10.1098/rsob.190287. This article has 51 citations and is from a peer-reviewed journal.
43. (sharma2020akinaseof pages 9-10): Samanta Sharma and Piotr Sicinski. A kinase of many talents: non-neuronal functions of cdk5 in development and disease. Open Biology, 10:190287, Jan 2020. URL: https://doi.org/10.1098/rsob.190287, doi:10.1098/rsob.190287. This article has 51 citations and is from a peer-reviewed journal.
44. (shupp2017biologicalfunctionsof pages 1-2): Alison Shupp, Mathew C. Casimiro, and Richard G. Pestell. Biological functions of cdk5 and potential cdk5 targeted clinical treatments. Oncotarget, 8:17373-17382, Jan 2017. URL: https://doi.org/10.18632/oncotarget.14538, doi:10.18632/oncotarget.14538. This article has 122 citations and is from a poor quality or predatory journal.
45. (shupp2017biologicalfunctionsof pages 7-8): Alison Shupp, Mathew C. Casimiro, and Richard G. Pestell. Biological functions of cdk5 and potential cdk5 targeted clinical treatments. Oncotarget, 8:17373-17382, Jan 2017. URL: https://doi.org/10.18632/oncotarget.14538, doi:10.18632/oncotarget.14538. This article has 122 citations and is from a poor quality or predatory journal.
46. (shupp2017biologicalfunctionsof pages 8-9): Alison Shupp, Mathew C. Casimiro, and Richard G. Pestell. Biological functions of cdk5 and potential cdk5 targeted clinical treatments. Oncotarget, 8:17373-17382, Jan 2017. URL: https://doi.org/10.18632/oncotarget.14538, doi:10.18632/oncotarget.14538. This article has 122 citations and is from a poor quality or predatory journal.
47. (smith2001cdk5onthe pages 1-2): DS Smith, PL Greer, and LH Tsai. Cdk5 on the brain. Unknown journal, 2001.