1. Phylogeny  
   Maternal embryonic leucine zipper kinase (MELK) is a serine/threonine kinase that belongs to the AMPK‐related subfamily within the broader kinome. Its evolutionary origin is deep and it is conserved across a wide range of eukaryotic organisms. Orthologs of MELK have been identified in diverse species, ranging from invertebrates such as Caenorhabditis elegans—where the enzyme is known as PIG‐1—to vertebrates including zebrafish, Xenopus, mouse, and human (ganguly2015melk—aconservedkinase pages 1-2, ganguly2015melk—aconservedkinase pages 4-5). The conservation of key structural domains, most notably the catalytic kinase domain and the ubiquitin-associated (UBA) domain, underlines the evolutionary pressure to maintain the function of MELK during development and cell cycle regulation. In evolutionary terms, MELK clusters with other AMPK‐related kinases, such as the MARK and NUAK families, which share similar domain architectures and play roles in cellular energy sensing and signal transduction pathways (jiang2013maternalembryonicleucine pages 1-3, cao2013structuralbasisfor pages 1-2). Such evolutionary conservation across species suggests that MELK performs fundamental cellular functions, with its orthologs contributing to processes as diverse as asymmetric cell division, neural progenitor proliferation, and embryogenesis. Phylogenetic analyses carried out by earlier studies (e.g., Manning et al.) have confirmed that kinases like MELK, which conserve a bilobal catalytic structure and accessory regulatory motifs, likely originated in the last common eukaryotic ancestor. The persistence of these features in MELK today indicates that the core mechanism of phosphorylation has been critical for the regulation of cell proliferation and developmental processes over evolutionary time (ganguly2015melk—aconservedkinase pages 1-2, jiang2013maternalembryonicleucine pages 1-3).
2. Reaction Catalyzed  
   The catalytic reaction mediated by MELK involves the transfer of the γ‐phosphate from adenosine triphosphate (ATP) to specific serine or threonine residues on substrate proteins. In its enzymatic activity, MELK binds ATP and positions it within a conserved active site such that the γ‐phosphate group is optimally oriented for transfer. The overall chemical reaction can be succinctly represented as follows:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(phospho-L-serine or phospho-L-threonine) + H⁺  
   This phosphorylation event alters the conformation or activity of the target substrate, thereby modulating downstream signaling pathways. By catalyzing this reaction, MELK controls key cellular events such as cell cycle progression, apoptotic signaling, and modulation of splicing processes (beullens2005substratespecificityand pages 1-2, cao2013structuralbasisfor pages 1-2).
3. Cofactor Requirements  
   MELK, like many serine/threonine kinases, is dependent on divalent metal cations to support its catalytic function. Specifically, Mg²⁺ is required for the proper coordination and binding of ATP within the catalytic cleft of the kinase domain. The magnesium ion stabilizes the negative charges on the phosphate groups of ATP, thereby facilitating the phosphoryl transfer reaction. In addition to Mg²⁺, kinase assays involving MELK have often been conducted in the presence of reducing agents—such as dithiothreitol (DTT)—to maintain a reduced cellular environment, which is necessary for preventing the formation of inhibitory disulfide bonds within the activation segment and other regulatory motifs (cao2013structuralbasisfor pages 2-3, beullens2005substratespecificityand pages 7-8). The combined requirement for Mg²⁺ and an appropriate redox balance ensures that MELK is maintained in an active conformation ready to interact with both ATP and its protein substrates (cao2013structuralbasisfor pages 2-3).
4. Substrate Specificity  
   MELK is characterized by a notably broad substrate specificity. Unlike many kinases that phosphorylate target proteins based on strict consensus motifs, MELK can recognize and phosphorylate serine/threonine residues embedded within diverse sequence contexts. In vitro experiments have demonstrated that MELK phosphorylates a wide spectrum of poly(peptides), and it has been shown to modify substrates such as BCL2L14, CDC25B, MAP3K5/ASK1, and ZNF622 according to the published literature (beullens2005substratespecificityand pages 7-8). In assays utilizing peptide arrays with as many as 192 different peptide substrates, MELK did not demonstrate a narrow consensus sequence; instead, its substrate recognition appears to be influenced by factors such as the autophosphorylation state of the enzyme and its interactions with additional substrate-specifying proteins (beullens2005substratespecificityand pages 1-1, beullens2005substratespecificityand pages 4-5). Although certain studies have employed synthetic substrates such as the AMARA and SAMS peptides to analyze kinetic parameters—yielding information on Km and kcat values—the overall picture indicates that MELK does not rely on a highly conserved linear motif for substrate selection (cao2013structuralbasisfor pages 2-3). Consequently, in a cellular context, the specificity of MELK toward its targets may be refined by protein–protein interactions, post-translational modifications, and conformational changes within its catalytic domain rather than by a fixed consensus sequence (beullens2005substratespecificityand pages 7-8, cao2013structuralbasisfor pages 2-3).
5. Structure  
   MELK’s structure is organized into several distinct domains that together underpin its catalytic function and regulatory control. The N-terminal portion of the protein comprises the kinase domain, a hallmark of serine/threonine kinases, which exhibits a bilobal arrangement. The smaller N-terminal lobe is predominantly composed of β-strands and includes a glycine-rich loop (P-loop) that is involved in ATP binding, whereas the larger C-terminal lobe is mainly α-helical and houses the catalytic loop, the activation segment, and critical regulatory motifs (cao2013structuralbasisfor pages 1-2, cao2013structuralbasisfor pages 3-5). Within the catalytic domain, the activation loop contains a key threonine residue (Thr167) whose autophosphorylation is essential for achieving a catalytically competent configuration (cao2013structuralbasisfor pages 2-3, cao2013structuralbasisfor pages 7-10).  
   Beyond the kinase domain, MELK features a ubiquitin-associated (UBA) domain that is structurally noncanonical in its fold. This UBA domain binds tightly to the backside of the kinase domain, stabilizing its overall conformation and contributing to both the solubility and the intrinsic catalytic activity of the enzyme (cao2013structuralbasisfor pages 5-6, cao2013structuralbasisfor pages 7-10). In addition, MELK possesses a TP-rich region and a kinase-associated 1 (KA1) domain. The KA1 domain is implicated in membrane association and may play a role in targeting the kinase to specific subcellular locales. Structural studies, including crystallographic analyses of MELK fragments expressed with kinase-dead mutations, have revealed that the kinase can bind ATP–Mg²⁺ effectively, yet the positioning of a protruding P+1 loop from the activation segment appears to impede the binding of exogenous substrates in the unphosphorylated state (cao2013structuralbasisfor pages 6-7, cao2013structuralbasisfor pages 7-10). The structural integrity of MELK is further maintained by the proper assembly of hydrophobic spines—the catalytic spine (C-spine) and the regulatory spine (R-spine)—which are characteristic features of active kinases and facilitate efficient intramolecular phosphotransfer reactions (cao2013structuralbasisfor pages 7-10, cao2013structuralbasisfor pages 10-11).
6. Regulation  
   The regulation of MELK is multifactorial, involving autophosphorylation, redox-dependent control, and intramolecular domain interactions. A central regulatory mechanism is the autophosphorylation of key residues within the activation loop, particularly at Thr167, which is indispensable for catalytic activation (beullens2005substratespecificityand pages 1-1, beullens2005substratespecificityand pages 1-2, cao2013structuralbasisfor pages 2-3). Additional phosphorylation events, including modifications at Ser171 and other residues within the autoinhibitory regions, further contribute to fine-tuning its catalytic activity (beullens2005substratespecificityand pages 8-9).  
   Redox regulation plays a significant role in MELK activity as well. Under oxidizing conditions, the formation of intramolecular disulfide bonds—particularly in the activation segment—can distort the proper conformation of the enzyme, leading to reduced activity. The presence of reducing agents such as DTT is therefore critical to disrupt these bonds and maintain MELK in an active state (cao2013structuralbasisfor pages 7-10, beullens2005substratespecificityand pages 7-8). Moreover, structural studies have highlighted the importance of the tight interaction between the kinase domain and the UBA domain. This interaction not only aids in the proper folding and solubility of MELK but also exerts an autoinhibitory effect that regulates substrate access. Mutational analyses disrupting the interface between these domains yield decreases in solubility and catalytic function, indicating that intramolecular domain integrity is essential for balanced kinase activity (cao2013structuralbasisfor pages 5-6, cao2013structuralbasisfor pages 7-10). Unlike many other AMPK-related kinases that are activated by upstream kinases such as LKB1, MELK is capable of undergoing autophosphorylation independently, underscoring its unique mode of regulation within its kinase family (beullens2005substratespecificityand pages 1-2, faisal2020developmentandtherapeutic pages 1-2).
7. Function  
   MELK performs an array of biological functions that intersect multiple cellular pathways. It is centrally involved in the regulation of the cell cycle; one pivotal role is the phosphorylation of CDC25B, a key phosphatase that drives mitotic progression by dephosphorylating and activating cyclin-dependent kinases. Phosphorylation of CDC25B by MELK is required for its proper localization to centrosomes and spindle poles during mitosis, thereby ensuring accurate cell division (cao2013structuralbasisfor pages 10-11, beullens2005substratespecificityand pages 4-5).  
   In addition to its role in the cell cycle, MELK is implicated in apoptotic signaling. It phosphorylates apoptosis signal-regulating kinase 1 (ASK1/MAP3K5), thereby activating apoptotic cascades under certain conditions. Conversely, MELK phosphorylates and inhibits the pro-apoptotic factor BCL2L14, which may contribute to the promotion of cell survival in the context of mammary carcinogenesis (information provided; beullens2005substratespecificityand pages 1-1). Another important functional aspect of MELK is its participation in the regulation of RNA processing. By phosphorylating the splicing factor ZNF622, MELK is able to interfere with spliceosome assembly, resulting in a redistribution of ZNF622 to the nucleus during mitosis. This modulation of RNA splicing may be critical for cell cycle-dependent gene expression programs (information provided, cao2013structuralbasisfor pages 10-11).  
   MELK is highly expressed in embryonic tissues and in the neural progenitor populations of both embryonic and postnatal brains, where it is essential for the self-renewal and proliferation of multipotent neural cells. Its expression pattern—and the observation that MELK is frequently upregulated in various malignancies such as breast, prostate, and head and neck cancers—points to a dual role in both normal development and oncogenesis (ganguly2015melk—aconservedkinase pages 1-2, almansour2022computationalexplorationof pages 1-2, walton2023abstract1766targeting pages 113-117). Through its capacity to integrate signals from cell cycle checkpoints, apoptotic cues, and RNA splicing pathways, MELK functions as an important regulatory node that contributes to cellular proliferation, survival, and differentiation.
8. Other Comments  
   MELK’s multifaceted roles in crucial cellular processes have made it an attractive target for therapeutic intervention, particularly in oncology. Several small-molecule inhibitors have been identified that target MELK’s catalytic activity; for example, compounds such as OTSSP167 have been developed with the aim of suppressing MELK-driven tumorigenesis (walton2023abstract1766targeting pages 113-117, almansour2022computationalexplorationof pages 7-9). In addition, fragment-based drug discovery approaches have led to the identification of novel kinase hinge-binding fragments, such as 8-Amino-2H-isoquinolin-1-one (MR1), which inhibits MELK with a reported Ki in the low-to-mid micromolar range (rachman2020discoveryofa pages 3-4, rachman2020discoveryofa pages 5-6). The broad substrate specificity of MELK, while complicating the identification of a unique phosphorylation motif, also suggests that its inhibition could disrupt multiple signaling pathways simultaneously—including those governing cell cycle progression, apoptosis, and RNA processing. Furthermore, MELK’s involvement in processes such as primitive hematopoiesis and maintenance of neural progenitor populations highlights its potential importance beyond cancer biology. Although overexpression of MELK has been correlated with aggressive tumor phenotypes and poor clinical outcomes, questions remain regarding whether MELK is universally essential for cancer cell proliferation; ongoing research continues to clarify its role in intracellular signaling networks (beullens2005substratespecificityand pages 1-1, information provided).  
   The complexity of MELK’s regulation—combining autophosphorylation, redox sensitivity, and intramolecular domain interactions—suggests that therapeutic strategies aimed at targeting this kinase must account for these multilayered control mechanisms. Advances in structural and computational biology have provided insights into the three-dimensional conformation of MELK, thereby facilitating the rational design of inhibitors that exploit its unique active site topology (cao2013structuralbasisfor pages 6-7, rachman2020discoveryofa pages 6-7). Overall, the continuing investigation into MELK’s function, regulation, and structural biology underscores its potential as both a biomarker and a target for the treatment of various malignancies.
9. References
10. Beullens, M., Vancauwenbergh, S., Morrice, N., Derua, R., Ceulemans, H., Waelkens, E., & Bollen, M. (2005). Substrate specificity and activity regulation of protein kinase MELK\*. Journal of Biological Chemistry, 280, 40003–40011. (beullens2005substratespecificityand pages 1-1, beullens2005substratespecificityand pages 1-2, beullens2005substratespecificityand pages 4-5, beullens2005substratespecificityand pages 7-8, beullens2005substratespecificityand pages 8-9, beullens2005substratespecificityand pages 9-10)
11. Cao, Lu-Sha, Wang, Jue, Chen, Yuling, Deng, Haiteng, Wang, Zhi-Xin, & Wu, Jia‐Wei. (2013). Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8, e70031. (cao2013structuralbasisfor pages 1-2, cao2013structuralbasisfor pages 2-3, cao2013structuralbasisfor pages 3-5, cao2013structuralbasisfor pages 5-6, cao2013structuralbasisfor pages 6-7, cao2013structuralbasisfor pages 7-10, cao2013structuralbasisfor pages 10-10, cao2013structuralbasisfor pages 10-11)
12. Ganguly, R., Mohyeldin, A., Thiel, J., Kornblum, H. I., Beullens, M., & Nakano, I. (2015). MELK—a conserved kinase: functions, signaling, cancer, and controversy. Clinical and Translational Medicine, Mar 2015. (ganguly2015melk—aconservedkinase pages 1-2, ganguly2015melk—aconservedkinase pages 4-5)
13. Rachman, M. M., Bajusz, D., Hetényi, A., Scarpino, A., Merő, B., Egyed, A., Buday, L., Barril, X., & Keserű, G. (2020). Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11, 552–558. (rachman2020discoveryofa pages 1-3, rachman2020discoveryofa pages 3-4, rachman2020discoveryofa pages 5-6, rachman2020discoveryofa pages 4-5, rachman2020discoveryofa pages 6-7)
14. Walton, J. P., Apostoli, A., Meens, J., Dmytryshyn, J., Arrowsmith, C., & AIlles, L. (2023). Abstract 1766: Targeting epigenetic regulation in clear cell renal cell carcinoma reveals PRMT1 as a novel target. Cancer Research, 80, 1766–1766. (walton2023abstract1766targeting pages 113-117)
15. Almansour, N. M. (2022). Computational exploration of maternal embryonic leucine zipper kinase (MELK) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. (almansour2022computationalexplorationof pages 1-2, almansour2022computationalexplorationof pages 2-3, almansour2022computationalexplorationof pages 5-6, almansour2022computationalexplorationof pages 6-7, almansour2022computationalexplorationof pages 7-9, almansour2022computationalexplorationof pages 9-9, almansour2022computationalexplorationof pages 3-5)
16. Jiang, P., & Zhang, D. (2013). Maternal embryonic leucine zipper kinase (MELK): A novel regulator in cell cycle control, embryonic development, and cancer. International Journal of Molecular Sciences, 14, 21551–21560. (jiang2013maternalembryonicleucine pages 1-3)
17. Faisal, M., Kim, J. H., Yoo, K. H., Roh, E. J., Hong, S. S., & Lee, S. H. (2020). Development and therapeutic potential of NUAKs inhibitors. Journal of Medicinal Chemistry, 64, 2–25. (faisal2020developmentandtherapeutic pages 1-2, faisal2020developmentandtherapeutic pages 2-3, faisal2020developmentandtherapeutic pages 8-9, faisal2020developmentandtherapeutic pages 17-18, faisal2020developmentandtherapeutic pages 21-22, faisal2020developmentandtherapeutic pages 23-23, faisal2020developmentandtherapeutic pages 18-18)
18. Timm, T., Marx, A., Panneerselvam, S., Mandelkow, E., & Mandelkow, E-M. (2008). Structure and regulation of MARK, a kinase involved in abnormal phosphorylation of tau protein. BMC Neuroscience, 9 Suppl 2, S9. (timm2008structureandregulation pages 1-2, timm2008structureandregulation pages 2-4)

References

1. (beullens2005substratespecificityand pages 7-8): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
2. (cao2013structuralbasisfor pages 1-2): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
3. (cao2013structuralbasisfor pages 2-3): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
4. (cao2013structuralbasisfor pages 5-6): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
5. (cao2013structuralbasisfor pages 7-10): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
6. (ganguly2015melk—aconservedkinase pages 4-5): Ranjit Ganguly, Ahmed Mohyeldin, Jordyn Thiel, Harley I Kornblum, Monique Beullens, and Ichiro Nakano. Melk—a conserved kinase: functions, signaling, cancer, and controversy. Clinical and Translational Medicine, Mar 2015. URL: https://doi.org/10.1186/s40169-014-0045-y, doi:10.1186/s40169-014-0045-y. This article has 120 citations and is from a peer-reviewed journal.
7. (almansour2022computationalexplorationof pages 1-2): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
8. (almansour2022computationalexplorationof pages 7-9): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
9. (almansour2022computationalexplorationof pages 9-9): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
10. (beullens2005substratespecificityand pages 1-1): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
11. (beullens2005substratespecificityand pages 1-2): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
12. (beullens2005substratespecificityand pages 4-5): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
13. (cao2013structuralbasisfor pages 10-10): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
14. (cao2013structuralbasisfor pages 10-11): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
15. (cao2013structuralbasisfor pages 3-5): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
16. (cao2013structuralbasisfor pages 6-7): Lu-Sha Cao, Jue Wang, Yuling Chen, Haiteng Deng, Zhi-Xin Wang, and Jia‐Wei Wu. Structural basis for the regulation of maternal embryonic leucine zipper kinase. PLoS ONE, 8:e70031, Jul 2013. URL: https://doi.org/10.1371/journal.pone.0070031, doi:10.1371/journal.pone.0070031. This article has 27 citations and is from a peer-reviewed journal.
17. (ganguly2015melk—aconservedkinase pages 1-2): Ranjit Ganguly, Ahmed Mohyeldin, Jordyn Thiel, Harley I Kornblum, Monique Beullens, and Ichiro Nakano. Melk—a conserved kinase: functions, signaling, cancer, and controversy. Clinical and Translational Medicine, Mar 2015. URL: https://doi.org/10.1186/s40169-014-0045-y, doi:10.1186/s40169-014-0045-y. This article has 120 citations and is from a peer-reviewed journal.
18. (rachman2020discoveryofa pages 3-4): Moira M. Rachman, D. Bajusz, A. Hetényi, A. Scarpino, B. Merő, Attila Egyed, L. Buday, X. Barril, and G. Keserű. Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11:552-558, Mar 2020. URL: https://doi.org/10.1039/c9md00519f, doi:10.1039/c9md00519f. This article has 14 citations and is from a peer-reviewed journal.
19. (rachman2020discoveryofa pages 5-6): Moira M. Rachman, D. Bajusz, A. Hetényi, A. Scarpino, B. Merő, Attila Egyed, L. Buday, X. Barril, and G. Keserű. Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11:552-558, Mar 2020. URL: https://doi.org/10.1039/c9md00519f, doi:10.1039/c9md00519f. This article has 14 citations and is from a peer-reviewed journal.
20. (walton2023abstract1766targeting pages 113-117): Joseph Paul Walton, Anthony Apostoli, Jalna Meens, Julia Dmytryshyn, Cheryl Arrowsmith, and Laurie AIlles. Abstract 1766: targeting epigenetic regulation in clear cell renal cell carcinoma reveals prmt1 as a novel target. Cancer Research, 80:1766-1766, Aug 2023. URL: https://doi.org/10.1158/1538-7445.am2020-1766, doi:10.1158/1538-7445.am2020-1766. This article has 0 citations and is from a highest quality peer-reviewed journal.
21. (almansour2022computationalexplorationof pages 2-3): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
22. (almansour2022computationalexplorationof pages 5-6): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
23. (almansour2022computationalexplorationof pages 6-7): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
24. (beullens2005substratespecificityand pages 8-9): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
25. (beullens2005substratespecificityand pages 9-10): M. Beullens, Sadia Vancauwenbergh, N. Morrice, R. Derua, H. Ceulemans, E. Waelkens, and M. Bollen. Substrate specificity and activity regulation of protein kinase melk\*. Journal of Biological Chemistry, 280:40003-40011, Dec 2005. URL: https://doi.org/10.1074/jbc.m507274200, doi:10.1074/jbc.m507274200. This article has 117 citations and is from a domain leading peer-reviewed journal.
26. (faisal2020developmentandtherapeutic pages 8-9): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
27. (jiang2013maternalembryonicleucine pages 1-3): Pengfei Jiang and Deli Zhang. Maternal embryonic leucine zipper kinase (melk): a novel regulator in cell cycle control, embryonic development, and cancer. International Journal of Molecular Sciences, 14:21551-21560, Oct 2013. URL: https://doi.org/10.3390/ijms141121551, doi:10.3390/ijms141121551. This article has 89 citations and is from a peer-reviewed journal.
28. (rachman2020discoveryofa pages 4-5): Moira M. Rachman, D. Bajusz, A. Hetényi, A. Scarpino, B. Merő, Attila Egyed, L. Buday, X. Barril, and G. Keserű. Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11:552-558, Mar 2020. URL: https://doi.org/10.1039/c9md00519f, doi:10.1039/c9md00519f. This article has 14 citations and is from a peer-reviewed journal.
29. (faisal2020developmentandtherapeutic pages 1-2): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
30. (faisal2020developmentandtherapeutic pages 2-3): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
31. (faisal2020developmentandtherapeutic pages 21-22): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
32. (faisal2020developmentandtherapeutic pages 23-23): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
33. (rachman2020discoveryofa pages 1-3): Moira M. Rachman, D. Bajusz, A. Hetényi, A. Scarpino, B. Merő, Attila Egyed, L. Buday, X. Barril, and G. Keserű. Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11:552-558, Mar 2020. URL: https://doi.org/10.1039/c9md00519f, doi:10.1039/c9md00519f. This article has 14 citations and is from a peer-reviewed journal.
34. (rachman2020discoveryofa pages 6-7): Moira M. Rachman, D. Bajusz, A. Hetényi, A. Scarpino, B. Merő, Attila Egyed, L. Buday, X. Barril, and G. Keserű. Discovery of a novel kinase hinge binder fragment by dynamic undocking. RSC Medicinal Chemistry, 11:552-558, Mar 2020. URL: https://doi.org/10.1039/c9md00519f, doi:10.1039/c9md00519f. This article has 14 citations and is from a peer-reviewed journal.
35. (timm2008structureandregulation pages 1-2): Thomas Timm, Alexander Marx, Saravanan Panneerselvam, Eckhard Mandelkow, and Eva-Maria Mandelkow. Structure and regulation of mark, a kinase involved in abnormal phosphorylation of tau protein. BMC Neuroscience, Dec 2008. URL: https://doi.org/10.1186/1471-2202-9-s2-s9, doi:10.1186/1471-2202-9-s2-s9. This article has 83 citations and is from a peer-reviewed journal.
36. (timm2008structureandregulation pages 2-4): Thomas Timm, Alexander Marx, Saravanan Panneerselvam, Eckhard Mandelkow, and Eva-Maria Mandelkow. Structure and regulation of mark, a kinase involved in abnormal phosphorylation of tau protein. BMC Neuroscience, Dec 2008. URL: https://doi.org/10.1186/1471-2202-9-s2-s9, doi:10.1186/1471-2202-9-s2-s9. This article has 83 citations and is from a peer-reviewed journal.
37. (almansour2022computationalexplorationof pages 3-5): Nahlah Makki Almansour. Computational exploration of maternal embryonic leucine zipper kinase (melk) as a cancer drug target. Saudi Journal of Biological Sciences, Jun 2022. URL: https://doi.org/10.1016/j.sjbs.2022.103335, doi:10.1016/j.sjbs.2022.103335. This article has 2 citations and is from a peer-reviewed journal.
38. (faisal2020developmentandtherapeutic pages 17-18): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.
39. (faisal2020developmentandtherapeutic pages 18-18): Muhammad Faisal, Jae Ho Kim, Kyung Ho Yoo, Eun Joo Roh, Soon Sun Hong, and So Ha Lee. Development and therapeutic potential of nuaks inhibitors. Journal of Medicinal Chemistry, 64:2-25, Dec 2020. URL: https://doi.org/10.1021/acs.jmedchem.0c00533, doi:10.1021/acs.jmedchem.0c00533. This article has 24 citations and is from a highest quality peer-reviewed journal.