1. Phylogeny  
   MAPK3, commonly known as ERK1, is a member of the extracellular signal‐regulated kinase (ERK) subgroup within the larger mitogen‐activated protein kinase (MAPK) family. ERK1/2 kinases share high sequence conservation among vertebrates and are present in all mammalian species, reflecting their evolution from a common eukaryotic ancestor (roskoski2012erk12mapkinases pages 1-2, kultz1998phylogeneticandfunctional pages 1-2). Phylogenetic analyses place MAPK3/ERK1 in a distinct clade within the MAPK superfamily that is associated with mitogenic signaling and substrate phosphorylation patterns that are conserved across species from yeast to mammals (kultz1998phylogeneticandfunctional pages 2-3, roskoski2012erk12mapkinases pages 2-2). Within the kinome, ERK1 is closely related to ERK2, with both isoforms sharing an 84% sequence identity; however, ERK1 possesses a unique N-terminal extension (a 17 amino acid insertion) that distinguishes it from ERK2 (roskoski2012erk12mapkinases pages 4-5). This subgroup is part of the detailed evolutionary landscape of MAP kinases described in seminal works by Manning et al. that tracked the origins of protein kinase families from the Last Eukaryotic Common Ancestor (LECA) (roskoski2012erk12mapkinases pages 1-2, kultz1998phylogeneticandfunctional pages 3-4).
2. Reaction Catalyzed  
   MAPK3/ERK1 catalyzes a classical phosphorylation reaction in which the γ‐phosphate group from adenosine triphosphate (ATP) is transferred to the hydroxyl group of serine or threonine residues on substrate proteins. The chemical reaction can be represented as follows:  
   ATP + [protein]–(L‑serine or L‑threonine) → ADP + [protein]–(L‑serine/threonine)‑phosphate + H⁺ (roskoski2012erk12mapkinases pages 1-2).
3. Cofactor Requirements  
   The kinase activity of MAPK3/ERK1 is dependent on the presence of divalent cations. Mg²⁺ is required as a cofactor to coordinate ATP binding within the catalytic cleft, thereby facilitating phosphoryl transfer to its substrate proteins (roskoski2012erk12mapkinases pages 6-8).
4. Substrate Specificity  
   MAPK3/ERK1 exhibits a strong substrate preference for serine/threonine residues that are immediately followed by a proline residue. This substrate specificity is largely dictated by the structural constraints of the kinase active site and is enhanced through docking interactions with substrates that display additional docking motifs. Specifically, many substrates contain a Pro-Xxx-Ser/Thr-Pro motif and also harbor either D-site (docking site) or F-site motifs, which bind to corresponding recruitment sites on ERK1 to stabilize enzyme–substrate interactions (roskoski2012erk12mapkinases pages 11-12, kultz1998phylogeneticandfunctional pages 9-12). This specificity ensures that ERK1 phosphorylates a wide repertoire of downstream targets – over 160 have been characterized – and is integral to the regulation of diverse cellular processes (old2009functionalproteomicsidentifies pages 3-4).
5. Structure  
   MAPK3/ERK1 contains a central catalytic kinase domain that is organized into a smaller N-terminal lobe and a larger C-terminal lobe. The N-terminal lobe typically comprises a five-stranded antiparallel β-sheet and a conserved C-helix, whereas the C-terminal lobe is predominantly α-helical and houses the substrate and ATP-binding sites (roskoski2012erk12mapkinases pages 4-5, roskoski2012erk12mapkinases pages 8-9).  
   A key structural feature is the activation loop (or T-loop) that contains the canonical dual phosphorylation motif, typically denoted as TEY (threonine-glutamate-tyrosine) for ERK1. Dual phosphorylation at the threonine and tyrosine residues within this motif, catalyzed by upstream kinases MEK1/2, induces a conformational shift—from an open, inactive state to a closed, active configuration—thus forming properly aligned hydrophobic regulatory (R) and catalytic (C) spines that are essential for enzymatic activity (roskoski2012erk12mapkinases pages 8-9, roskoski2012erk12mapkinases pages 9-10).  
   Additional regulatory docking regions are present on ERK1, including the D-site recruitment site (DRS) and the F-site recruitment site (FRS), which mediate binding to substrates exhibiting complementary docking motifs. These features, combined with the characteristic ATP-binding cleft (which relies on hinge region interactions for the formation of hydrogen bonds with the inhibitor or nucleotide), define the overall fold and catalytic mechanism of the kinase (roskoski2012erk12mapkinases pages 14-15, roskoski2012erk12mapkinases pages 28-29). Moreover, a unique N-terminal extension in ERK1, relative to ERK2, may confer specific regulatory or spatial properties, although both share the core catalytic architecture (roskoski2012erk12mapkinases pages 4-5).
6. Regulation  
   MAPK3/ERK1 activity is intricately regulated by multiple mechanisms that ensure controlled signal transduction. The primary mode of activation is through dual phosphorylation of the TEY activation motif on the T-loop by the upstream kinases MEK1/2. This phosphorylation event is essential for the transition from an inactive to an active kinase conformation and significantly increases catalytic activity (roskoski2012erk12mapkinases pages 1-2, roskoski2012erk12mapkinases pages 5-6).  
   Beyond the TEY motif, ERK1 exhibits additional regulatory phosphorylation sites within the T-loop, notably threonine 207 (T207) and tyrosine 210 (Y210), which have been shown to play roles in modulating kinase activity through autoinhibitory mechanisms. Experimental mutagenesis studies indicate that phosphorylation at T207 can inhibit substrate phosphorylation, thereby contributing to a negative feedback mechanism that limits ERK1 signaling duration (lai2016regulatoryrolesof pages 7-9, lai2016regulatoryrolesof pages 9-12).  
   Regulatory control is also exerted via protein–protein interactions with scaffold proteins such as KSR1/2 and IQGAP1. These scaffolds facilitate the assembly of the Raf-MEK-ERK cascade, thereby enhancing signal propagation and providing spatial and temporal regulation of kinase activity (roskoski2012erk12mapkinases pages 15-16). In addition, ERK1 activity is attenuated by dephosphorylation events mediated by dual-specificity phosphatases (DUSPs) including DUSP6/MKP3, which specifically recognize and deactivate ERK1 by removing phosphate groups from both the threonine and tyrosine residues in the activation loop (roskoski2012erk12mapkinases pages 23-24, roskoski2012erk12mapkinases pages 24-26). Furthermore, regulatory mechanisms such as nuclear-cytoplasmic shuttling, which involves specific nuclear localization factors and interactions with nucleoporins, ensure that ERK1 is localized appropriately to phosphorylate either cytosolic or nuclear substrates depending on the cellular context (roskoski2012erk12mapkinases pages 26-27, roskoski2012erk12mapkinases pages 37-38).
7. Function  
   MAPK3/ERK1 is a central serine/threonine kinase that operates as a critical effector in the Ras-Raf-MEK-ERK signaling cascade. This pathway is initiated by the activation of receptor tyrosine kinases (RTKs), such as KIT through binding to its ligand KITLG/SCF, which then engage the small GTPase Ras and its downstream effector Raf kinases. Activated Raf phosphorylates MEK1/2, which in turn activate ERK1 via dual phosphorylation of the TEY motif (roskoski2012erk12mapkinases pages 5-6, roskoski2012erk12mapkinases pages 1-2). Once active, ERK1 phosphorylates a broad array of substrates – estimated at over 160 – that include transcription factors (e.g., ATF2, ELK1, FOS), components of the cytoskeleton (e.g., MAPT, CTTN, PXN), regulators of apoptosis (e.g., BAD, CASP9), and proteins involved in translation (e.g., EIF4EBP1) (roskoski2012erk12mapkinases pages 1-2, roskoski2012erk12mapkinases pages 35-36).  
   In the nucleus, ERK1-mediated phosphorylation of transcription factors promotes the induction of immediate early genes that govern cell proliferation, differentiation, and survival. In the cytoplasm, ERK1 targets substrates that orchestrate processes such as cell adhesion, migration, and mitosis, as well as organelle dynamics involved in lysosomal processing and endosomal cycling through the perinuclear recycling compartment (PNRC) (roskoski2012erk12mapkinases pages 35-36, roskoski2012erk12mapkinases pages 37-38). Additionally, ERK1 signaling is essential in the regulation of cell cycle transitions including the initiation of meiosis and mitosis, with its substrates participating in key checkpoint controls and cytoskeletal rearrangements necessary for successful cell division (old2009functionalproteomicsidentifies pages 3-4, roskoski2012erk12mapkinases pages 5-6). The ubiquitous expression of ERK1 in various cell types underscores its role as a fundamental mediator in essential signaling pathways that govern both proliferative and cell-differentiation processes (roskoski2012erk12mapkinases pages 1-2).
8. Other Comments  
   MAPK3/ERK1 is a clinically significant target due to its frequent involvement in oncogenic signaling pathways. Aberrant activation of the Ras-Raf-MEK-ERK cascade, often through mutations in upstream regulators such as KRAS or BRAF (e.g., BRAFV600E), drives persistent ERK1 activation in a variety of cancers, including melanoma, colorectal, and pancreatic cancers (roskoski2012erk12mapkinases pages 6-8, old2009functionalproteomicsidentifies pages 5-7). Inhibitor development has originally focused on ATP-competitive compounds, with additional classes emerging that target allosteric sites or disrupt ERK1 interactions with its substrates via the D-site or F-site docking regions (roskoski2012erk12mapkinases pages 29-30, huang2021chemoproteomicprofilingof pages 7-9). Although no single inhibitor is completely specific for ERK1 over its closely related isoform ERK2, several compounds (e.g., FR180204) have demonstrated potency in preclinical models by reducing aberrant phosphorylation events downstream of the ERK cascade (roskoski2012erk12mapkinases pages 28-29). Disease associations extend beyond cancer; dysregulation of ERK1 activity has also been implicated in conditions affecting the cell cycle, inflammatory responses, and neurodegeneration. Recent computational studies analyzing the structural impacts of missense mutations highlight that alterations near the ATP-binding region or activation loop can compromise ERK1 stability and catalytic function, further emphasizing the therapeutic importance of structure-based inhibitor design (rodrigues2025exploringtheeffects pages 1-2).  
   Furthermore, MAPK3/ERK1 is known to participate in feedback regulatory loops mediated by specific phosphatases such as DUSP6/MKP3, ensuring that its activity is appropriately dampened following mitogenic stimulation (roskoski2012erk12mapkinases pages 23-24). The integrated network of scaffold proteins, upstream kinases, and downstream substrates positions ERK1 as an indispensable regulator of both acute and chronic cellular responses to external stimuli (roskoski2012erk12mapkinases pages 15-16).
9. References
10. roskoski2012erk12mapkinases pages 1-2
11. roskoski2012erk12mapkinases pages 2-2
12. roskoski2012erk12mapkinases pages 4-5
13. roskoski2012erk12mapkinases pages 5-6
14. roskoski2012erk12mapkinases pages 6-8
15. roskoski2012erk12mapkinases pages 8-9
16. roskoski2012erk12mapkinases pages 9-10
17. roskoski2012erk12mapkinases pages 11-12
18. roskoski2012erk12mapkinases pages 14-15
19. roskoski2012erk12mapkinases pages 15-16
20. roskoski2012erk12mapkinases pages 23-24
21. roskoski2012erk12mapkinases pages 24-26
22. roskoski2012erk12mapkinases pages 26-27
23. roskoski2012erk12mapkinases pages 28-29
24. roskoski2012erk12mapkinases pages 34-35
25. roskoski2012erk12mapkinases pages 36-37
26. roskoski2012erk12mapkinases pages 37-38
27. kultz1998phylogeneticandfunctional pages 1-2
28. kultz1998phylogeneticandfunctional pages 2-3
29. kultz1998phylogeneticandfunctional pages 3-4
30. kultz1998phylogeneticandfunctional pages 9-12
31. lai2016regulatoryrolesof pages 1-3
32. lai2016regulatoryrolesof pages 7-9
33. lai2016regulatoryrolesof pages 9-12
34. lai2016regulatoryrolesof pages 12-14
35. lai2016regulatoryrolesof pages 14-17
36. lai2016regulatoryrolesof pages 17-25
37. old2009functionalproteomicsidentifies pages 3-4
38. old2009functionalproteomicsidentifies pages 5-7
39. rodrigues2025exploringtheeffects pages 1-2
40. dorin1999anatypicalmitogenactivated pages 4-5

References

1. (roskoski2012erk12mapkinases pages 1-2): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
2. (roskoski2012erk12mapkinases pages 11-12): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
3. (roskoski2012erk12mapkinases pages 2-2): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
4. (roskoski2012erk12mapkinases pages 29-30): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
5. (roskoski2012erk12mapkinases pages 35-36): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
6. (roskoski2012erk12mapkinases pages 6-8): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
7. (huang2021chemoproteomicprofilingof pages 7-9): Tao Huang, Seyyedmohsen Hosseinibarkooie, Adam L. Borne, Mitchell E. Granade, Jeffrey W. Brulet, Thurl E. Harris, Heather A. Ferris, and Ku-Lung Hsu. Chemoproteomic profiling of kinases in live cells using electrophilic sulfonyl triazole probes. Chemical Science, 12:3295-3307, Jan 2021. URL: https://doi.org/10.1039/d0sc06623k, doi:10.1039/d0sc06623k. This article has 27 citations and is from a highest quality peer-reviewed journal.
8. (kultz1998phylogeneticandfunctional pages 1-2): Dietmar Kültz. Phylogenetic and functional classification of mitogen- and stress-activated protein kinases. Journal of Molecular Evolution, 46:571-588, May 1998. URL: https://doi.org/10.1007/pl00006338, doi:10.1007/pl00006338. This article has 255 citations and is from a peer-reviewed journal.
9. (kultz1998phylogeneticandfunctional pages 2-3): Dietmar Kültz. Phylogenetic and functional classification of mitogen- and stress-activated protein kinases. Journal of Molecular Evolution, 46:571-588, May 1998. URL: https://doi.org/10.1007/pl00006338, doi:10.1007/pl00006338. This article has 255 citations and is from a peer-reviewed journal.
10. (lai2016regulatoryrolesof pages 1-3): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
11. (lai2016regulatoryrolesof pages 12-14): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
12. (lai2016regulatoryrolesof pages 14-17): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
13. (lai2016regulatoryrolesof pages 17-25): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
14. (old2009functionalproteomicsidentifies pages 3-4): William M. Old, John B. Shabb, Stephane Houel, Hong Wang, Kasey L. Couts, Chia-yu Yen, Elizabeth S. Litman, Carrie H. Croy, Karen Meyer-Arendt, Jose G. Miranda, Robert A. Brown, Eric S. Witze, Rebecca E. Schweppe, Katheryn A. Resing, and Natalie G. Ahn. Functional proteomics identifies targets of phosphorylation by b-raf signaling in melanoma. Molecular cell, 34 1:115-31, Apr 2009. URL: https://doi.org/10.1016/j.molcel.2009.03.007, doi:10.1016/j.molcel.2009.03.007. This article has 177 citations and is from a highest quality peer-reviewed journal.
15. (old2009functionalproteomicsidentifies pages 5-7): William M. Old, John B. Shabb, Stephane Houel, Hong Wang, Kasey L. Couts, Chia-yu Yen, Elizabeth S. Litman, Carrie H. Croy, Karen Meyer-Arendt, Jose G. Miranda, Robert A. Brown, Eric S. Witze, Rebecca E. Schweppe, Katheryn A. Resing, and Natalie G. Ahn. Functional proteomics identifies targets of phosphorylation by b-raf signaling in melanoma. Molecular cell, 34 1:115-31, Apr 2009. URL: https://doi.org/10.1016/j.molcel.2009.03.007, doi:10.1016/j.molcel.2009.03.007. This article has 177 citations and is from a highest quality peer-reviewed journal.
16. (roskoski2012erk12mapkinases pages 14-15): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
17. (roskoski2012erk12mapkinases pages 15-16): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
18. (roskoski2012erk12mapkinases pages 23-24): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
19. (roskoski2012erk12mapkinases pages 24-26): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
20. (roskoski2012erk12mapkinases pages 26-27): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
21. (roskoski2012erk12mapkinases pages 28-29): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
22. (roskoski2012erk12mapkinases pages 34-35): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
23. (roskoski2012erk12mapkinases pages 36-37): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
24. (roskoski2012erk12mapkinases pages 37-38): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
25. (roskoski2012erk12mapkinases pages 4-5): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
26. (roskoski2012erk12mapkinases pages 5-6): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
27. (roskoski2012erk12mapkinases pages 8-9): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
28. (roskoski2012erk12mapkinases pages 9-10): Robert Roskoski. Erk1/2 map kinases: structure, function, and regulation. Pharmacological Research, 66:105-143, Aug 2012. URL: https://doi.org/10.1016/j.phrs.2012.04.005, doi:10.1016/j.phrs.2012.04.005. This article has 2077 citations and is from a highest quality peer-reviewed journal.
29. (dorin1999anatypicalmitogenactivated pages 4-5): Dominique Dorin, Pietro Alano, Irène Boccaccio, Liliane Cicéron, Caroline Doerig, Renan Sulpice, Daniel Parzy, and Christian Doerig. An atypical mitogen-activated protein kinase (mapk) homologue expressed in gametocytes of the human malaria parasite plasmodium falciparum. Journal of Biological Chemistry, 274:29912-29920, Oct 1999. URL: https://doi.org/10.1074/jbc.274.42.29912, doi:10.1074/jbc.274.42.29912. This article has 127 citations and is from a domain leading peer-reviewed journal.
30. (kultz1998phylogeneticandfunctional pages 3-4): Dietmar Kültz. Phylogenetic and functional classification of mitogen- and stress-activated protein kinases. Journal of Molecular Evolution, 46:571-588, May 1998. URL: https://doi.org/10.1007/pl00006338, doi:10.1007/pl00006338. This article has 255 citations and is from a peer-reviewed journal.
31. (kultz1998phylogeneticandfunctional pages 9-12): Dietmar Kültz. Phylogenetic and functional classification of mitogen- and stress-activated protein kinases. Journal of Molecular Evolution, 46:571-588, May 1998. URL: https://doi.org/10.1007/pl00006338, doi:10.1007/pl00006338. This article has 255 citations and is from a peer-reviewed journal.
32. (lai2016regulatoryrolesof pages 7-9): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
33. (lai2016regulatoryrolesof pages 9-12): Shenshen Lai and Steven Pelech. Regulatory roles of conserved phosphorylation sites in the activation t-loop of the map kinase erk1. Molecular Biology of the Cell, 27:1040-1050, Mar 2016. URL: https://doi.org/10.1091/mbc.e15-07-0527, doi:10.1091/mbc.e15-07-0527. This article has 50 citations and is from a domain leading peer-reviewed journal.
34. (rodrigues2025exploringtheeffects pages 1-2): Carlos H. M. Rodrigues, Stephanie Portelli, and David B. Ascher. Exploring the effects of missense mutations on protein thermodynamics through structure-based approaches: findings from the cagi6 challenges. Human Genetics, 144:327-335, Jan 2025. URL: https://doi.org/10.1007/s00439-023-02623-4, doi:10.1007/s00439-023-02623-4. This article has 8 citations and is from a peer-reviewed journal.