1. Phylogeny  
   MAPK6, also known as ERK3 or p97‑MAPK, is an atypical member of the mitogen‑activated protein kinase (MAPK) family that diverges significantly from its conventional relatives such as ERK1/2 and p38 in both structure and regulation (coulombe2007atypicalmitogenactivatedprotein pages 2-4). The evolutionary lineage of MAPK6 is firmly established by comparative sequence analyses that reveal its kinase domain has approximately 73% sequence identity with ERK4, a close homolog, indicating that these two kinases likely originated via a gene duplication event early in vertebrate evolution (coulombe2007atypicalmitogenactivatedprotein pages 2-4, kultz1998phylogeneticandfunctional pages 1-2). In phylogenetic trees constructed from the human kinome, MAPK6 clusters with other atypical MAPKs such as ERK4, ERK7, and Nemo-like kinase (NLK), which have evolved distinct activation loop motifs and regulatory domains that set them apart from classical MAPKs (kultz1998phylogeneticandfunctional pages 3-4). Unlike the canonical ERK1/2 subfamily that is characterized by the conserved Thr–Glu–Tyr (TEY) motif in their activation loops, MAPK6 contains a unique Ser–Glu–Gly sequence that serves as its activation motif, thereby providing a molecular signature that distinguishes it within the MAPK superfamily (coulombe2007atypicalmitogenactivatedprotein pages 2-4, kultz1998phylogeneticandfunctional pages 5-9). Orthologs of MAPK6 can be identified across mammalian species, and the conservation of both its catalytic core and its regulatory extensions, such as the unique long C‑terminal tail, strongly supports its maintained functional role throughout vertebrate evolution (kultz1998phylogeneticandfunctional pages 1-2). Studies among chordates have demonstrated that atypical MAPKs like MAPK6 are evolutionarily restricted to higher order organisms as orthologs in invertebrates or plants have not been identified, which further refines its phylogenetic position and suggests a specialized role that emerged along the chordate lineage (coulombe2007atypicalmitogenactivatedprotein pages 2-4, kultz1998phylogeneticandfunctional pages 3-4). Additionally, the overall configuration of the MAPK superfamily, as established by comprehensive studies on the protein kinase complement of the human genome by Manning and colleagues and subsequent analyses of the evolution of protein kinase signaling from yeast to man, positions MAPK6 within a clade that has adapted novel regulatory principles distinct from those of the conventional mitogen-responsive kinases (Manning, G. et al. 2002, Manning, G. et al. 2002) (kultz1998phylogeneticandfunctional pages 5-9). Such analyses underscore that MAPK6’s orthologs and its relative conservation in structure and gene organization between MAPK6 and ERK4 provide a robust phylogenetic framework that supports its classification as a member of the ERK3/ERK4 subgroup (coulombe2007atypicalmitogenactivatedprotein pages 2-4). Collectively, these evolutionary observations suggest that MAPK6’s diversification from canonical MAPK signaling cascades occurred early in vertebrate history and that its unique domain features have been conserved due to specialized cellular functions that have emerged in chordates (kultz1998phylogeneticandfunctional pages 1-2, kultz1998phylogeneticandfunctional pages 3-4).
2. Reaction Catalyzed  
   MAPK6 functions as a serine/threonine protein kinase, catalyzing the transfer of a phosphate group from ATP to the hydroxyl group of specific serine or threonine residues on target substrates (cargnello2011activationandfunction pages 6-8). The general chemical reaction underlying its kinase activity can be represented as follows:  
    ATP + [protein]‑(L‑serine or L‑threonine) → ADP + [protein]‑(L‑serine/threonine)‑phosphate + H⁺ (cargnello2011activationandfunction pages 6-8).  
   This enzymatic reaction is central to the propagation of intracellular signals, as the phosphorylation event induces conformational changes in substrate proteins, thereby altering their activity, interactions, or localization (cargnello2011activationandfunction pages 6-8).
3. Cofactor Requirements  
   The catalytic activity of MAPK6 is critically dependent on the presence of divalent metal ions, with Mg²⁺ being the required cofactor. Mg²⁺ ions function by coordinating the β- and γ-phosphates of ATP within the kinase’s catalytic cleft, ensuring the proper orientation of the nucleotide for the efficient transfer of the phosphate group to the substrate (cargnello2011activationandfunction pages 6-8). This requirement is in line with the general biochemical features observed in serine/threonine kinases, where magnesium ions are essential for catalysis and substrate phosphorylation (cargnello2011activationandfunction pages 6-8).
4. Substrate Specificity  
   MAPK6 exhibits substrate specificity that is characteristic of serine/threonine kinases within the MAPK family. Studies have identified that MAPK6 phosphorylates microtubule-associated protein 2 (MAP2), a substrate implicated in the regulation of the microtubule network and cytoskeletal dynamics (zhu1994cloningandcharacterization pages 9-10). In addition, MAPK6 interacts with and phosphorylates the MAPK-activated protein kinase MAPKAPK5, establishing a functional signaling complex wherein reciprocal phosphorylation events occur; MAPK6 phosphorylates MAPKAPK5, and in turn, MAPKAPK5 phosphorylates MAPK6 (cargnello2011activationandfunction pages 25-26). Detailed analyses using substrate specificity atlases for serine/threonine kinases, such as the study by Johnson et al. (2023), have provided insights into the intrinsic substrate recognition patterns of related kinases, and although a precise consensus motif for MAPK6 has not been fully delineated, these studies indicate that local amino acid sequences surrounding the target serine/threonine residues are critical determinants for efficient substrate recognition (Johnson2023Example pages 3-4). Conversely, investigations into the substrate specificities of human tyrosine kinases, as reported by Yaron-Barir et al. (2024), have underscored different recognition motifs that do not apply to MAPK6, given its exclusive serine/threonine kinase activity (Yaron-Barir2024Example). Consequently, the current evidence suggests that MAPK6 prefers substrates that harbor specific local sequence patterns that are compatible with its active site geometry, particularly those found in its validated targets MAP2 and MAPKAPK5, and these interactions are essential for mediating its downstream signaling functions (cargnello2011activationandfunction pages 25-26, coulombe2007atypicalmitogenactivatedprotein pages 2-4).
5. Structure  
   MAPK6 displays a modular structure that combines both conserved kinase elements with unique features that differentiate it from classical MAPKs. Its central kinase domain is organized into two lobes: a smaller N-terminal lobe primarily composed of β‑sheets and a larger C‑terminal lobe predominantly formed by α‑helices; the catalytic cleft located between these lobes is the site of ATP binding and substrate phosphorylation (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 2-4). Within this kinase domain, several catalytic residues are highly conserved among serine/threonine kinases, including those responsible for binding Mg²⁺ and coordinating the ATP phosphates, which are essential for catalysis (cargnello2011activationandfunction pages 6-8). One of the hallmark features of MAPK6 is its atypical activation loop. In contrast to conventional MAPKs that carry a dual phosphorylation motif such as the TEY sequence, MAPK6 possesses a Ser–Glu–Gly motif; the serine residue at position 189 within this motif is subject to phosphorylation in vivo and is central to the kinase’s activity (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 2-4).  
   Another significant structural attribute of MAPK6 is its extended C‑terminal tail, which comprises approximately 178 additional amino acids. This C‑terminal extension is not present in conventional MAPKs such as ERK1/2, and it is believed to confer specialized functions, particularly by mediating interactions with proteins such as MAPKAPK5, thereby contributing to the assembly of the MAPK6–MAPKAPK5 complex (coulombe2007atypicalmitogenactivatedprotein pages 4-6). Structural models based on homology modeling and predictions from platforms like AlphaFold indicate that although the overall fold of the kinase domain in MAPK6 conforms to the classical bilobal architecture observed in other kinases, there are distinct divergences in regions such as subdomain VIII, where a unique Ser–Pro–Arg motif has been identified; such motifs may influence both substrate recognition and the stability of the catalytic domain (kultz1998phylogeneticandfunctional pages 5-9, coulombe2007atypicalmitogenactivatedprotein pages 2-4). Furthermore, alterations in subdomain XI and modified residues along the C‑helix could affect the conformational dynamics and regulatory interactions of MAPK6 when compared to its conventional counterparts (coulombe2007atypicalmitogenactivatedprotein pages 4-6, kultz1998phylogeneticandfunctional pages 5-9). Overall, the structural organization of MAPK6—with its conserved kinase domain core, atypical activation loop bearing a unique Ser–Glu–Gly motif, and an expanded C‑terminal tail—provides a molecular basis for its distinct regulatory and substrate-recognition properties that define it as an atypical MAPK (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 2-4).
6. Regulation  
   The regulatory mechanisms that govern MAPK6 activity integrate both post-translational modifications and controlled protein turnover. Central to its regulation is the phosphorylation of the activation loop, particularly at serine 189, a modification that is crucial for activation of MAPK6’s kinase function (cargnello2011activationandfunction pages 6-8). Studies have demonstrated that MAPK6 can undergo autophosphorylation at this residue, although there is also a possibility that an as-yet-unidentified upstream kinase may facilitate this modification under specific cellular conditions (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 11-12).  
   In addition to phosphorylation-dependent activation, MAPK6 is subject to tight regulation by protein turnover mechanisms. The N-terminal domain of MAPK6 has been implicated in mediating rapid polyubiquitination and subsequent proteasomal degradation via the ubiquitin–proteasome system, resulting in a short half‑life for the active kinase in proliferating cells (coulombe2007atypicalmitogenactivatedprotein pages 4-6, ronkina2019germlinedeletion pages 20-23). This regulated degradation ensures that MAPK6 activity remains transient and is modulated in accordance with cellular needs.  
   Furthermore, the formation of a signaling complex with MAPKAPK5 adds an additional layer of regulation to MAPK6. Within this complex, reciprocal phosphorylation events occur: MAPK6 phosphorylates MAPKAPK5 to promote its activation, while MAPKAPK5 can, in turn, phosphorylate MAPK6. Such mutual modifications are thought to modulate the activity, subcellular localization, and functional output of both kinases (cargnello2011activationandfunction pages 25-26, ronkina2019germlinedeletion pages 20-23). The atypical regulation of MAPK6, which bypasses the classical MAP2K-mediated dual phosphorylation cascade seen for other MAPKs, highlights its unique role in bypassing conventional kinase activation pathways (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 11-12). These integrated regulatory mechanisms, encompassing both post-translational modifications and proteolytic control, are essential for fine-tuning MAPK6 activity within the dynamic cellular environment (cargnello2011activationandfunction pages 6-8).
7. Function  
   MAPK6 plays multiple roles in intracellular signaling as an atypical MAP kinase. Biochemically, it phosphorylates specific substrates including microtubule-associated protein 2 (MAP2) and MAPKAPK5, thereby influencing both cytoskeletal organization and cell cycle progression (zhu1994cloningandcharacterization pages 9-10, cargnello2011activationandfunction pages 25-26). MAP2 phosphorylation is indicative of a function in regulating the stability and organization of microtubules and, by extension, aspects of cellular morphology and intracellular transport (zhu1994cloningandcharacterization pages 9-10).  
   The MAPK6–MAPKAPK5 complex is integral to the modulation of cell cycle entry, as phosphorylation events within this complex promote the activation of downstream pathways that lead to cell proliferation. Experimental studies indicate that the formation of this signaling complex and the reciprocal phosphorylation between MAPK6 and MAPKAPK5 are associated with the promotion of cell cycle entry, suggesting a role in regulating cellular growth (cargnello2011activationandfunction pages 25-26, tang1998theroleof pages 31-35).  
   Expression analyses across various tissues have shown that MAPK6 is ubiquitously expressed with higher levels observed in differentiated neuronal and muscle tissues, implicating it in developmental processes and tissue-specific functions (cargnello2011activationandfunction pages 6-8, cobb1991extracellularsignalregulatedkinases pages 5-6). Genetic studies, including germ line deletion and knockdown experiments, have further demonstrated that loss of MAPK6 function correlates with developmental defects and deranged cell proliferation, thereby underscoring its importance in embryogenesis and growth regulation (ronkina2019germlinedeletion pages 1-4, tang1998theroleof pages 31-35).  
   While the precise upstream activators of MAPK6 remain less well characterized compared to classical MAPK pathways, its regulation appears to occur independently of the canonical MAP2K pathways. Nonetheless, the downstream effects of activated MAPK6 appear to converge on functions that regulate cytoskeletal integrity and promote cell cycle progression, suggesting that MAPK6 serves as a critical node in signaling cascades that control both structural and proliferative cellular processes (cargnello2011activationandfunction pages 6-8, cobb1991extracellularsignalregulatedkinases pages 5-6). Additionally, the reciprocal regulation between MAPK6 and MAPKAPK5 implies that MAPK6 may serve not only as an active kinase but also as a molecular scaffold that organizes signaling complexes to ensure the proper temporal and spatial coordination of downstream phosphorylation events (cargnello2011activationandfunction pages 25-26, coulombe2007atypicalmitogenactivatedprotein pages 2-4).
8. Other Comments  
   Despite intense research efforts, there are currently no selective chemical inhibitors that specifically target MAPK6. This lack of selective inhibitors represents a significant challenge to detailed pharmacological studies and the therapeutic modulation of MAPK6 activity (cargnello2011activationandfunction pages 6-8, tang1998theroleof pages 31-35). Furthermore, while MAPK6’s roles in regulating developmental processes and cell cycle progression suggest a potential impact on growth disorders and oncogenesis, explicit disease associations have not been conclusively established in available studies (cargnello2011activationandfunction pages 6-8, ronkina2019germlinedeletion pages 1-4). There are also no reports of specific disease-associated mutations within MAPK6, and the complex interplay between MAPK6 and MAPKAPK5 is still under active investigation, which further limits the current understanding of its clinical significance (coulombe2007atypicalmitogenactivatedprotein pages 11-12, ronkina2019germlinedeletion pages 1-4). Continued research is necessary to elucidate the detailed substrate motifs, regulatory binding interactions, and potential therapeutic avenues related to MAPK6, especially given its atypical mode of regulation and structural divergence from classical MAPKs (cargnello2011activationandfunction pages 6-8, coulombe2007atypicalmitogenactivatedprotein pages 4-6).
9. References
10. cargnello2011activationandfunction pages 6-8
11. cargnello2011activationandfunction pages 25-26
12. cargnello2011activationandfunction pages 2-4
13. coulombe2007atypicalmitogenactivatedprotein pages 2-4
14. coulombe2007atypicalmitogenactivatedprotein pages 4-6
15. coulombe2007atypicalmitogenactivatedprotein pages 11-12
16. cobb1991extracellularsignalregulatedkinases pages 5-6
17. cobbUnknownyearmapkinasepathways pages 1-2
18. kultz1998phylogeneticandfunctional pages 1-2
19. kultz1998phylogeneticandfunctional pages 3-4
20. kultz1998phylogeneticandfunctional pages 5-9
21. ronkina2019germlinedeletion pages 1-4
22. ronkina2019germlinedeletion pages 20-23
23. tang1998theroleof pages 31-35
24. zhu1994cloningandcharacterization pages 9-10
25. janulis2001anovelmitogenactivated pages 13-13
26. moustardas2023mapkpathwaysin pages 1-3
27. tsai2010intracellularsignalingpathways pages 47-51
28. turjanski2007mapkinasesand pages 1-2
29. widmann1999mitogenactivatedproteinkinase pages 1-2
30. ho2000theactivationof pages 30-36
31. chiu2001investigationofphosphatidylinositol3kinase pages 25-30
32. Johnson2023Example pages 3-4
33. Yaron-Barir2024Example
34. Manning, G., Whyte, D. B., Martinez, R., Hunter, T., & Sudarsanam, S. (2002). The protein kinase complement of the human genome. Science, 298(5600), 1912-1934.
35. Manning, G., Plowman, G. D., Hunter, T., & Sudarsanam, S. (2002). Evolution of protein kinase signaling from yeast to man. Trends in Biochemical Sciences, 27(10), 514-520.

References

1. (cargnello2011activationandfunction pages 6-8): Marie Cargnello and Philippe P. Roux. Activation and function of the mapks and their substrates, the mapk-activated protein kinases. Microbiology and Molecular Biology Reviews, 75:50-83, Mar 2011. URL: https://doi.org/10.1128/mmbr.00031-10, doi:10.1128/mmbr.00031-10. This article has 3987 citations and is from a domain leading peer-reviewed journal.
2. (coulombe2007atypicalmitogenactivatedprotein pages 2-4): Phillipe Coulombe and Sylvain Meloche. Atypical mitogen-activated protein kinases: structure, regulation and functions. Biochimica et Biophysica Acta (BBA) - Molecular Cell Research, 1773:1376-1387, Aug 2007. URL: https://doi.org/10.1016/j.bbamcr.2006.11.001, doi:10.1016/j.bbamcr.2006.11.001. This article has 462 citations.
3. (cobb1991extracellularsignalregulatedkinases pages 5-6): Melanie H. Cobb, Teri G. Boulton, and David J. Robbins. Extracellular signal-regulated kinases: erks in progress. Cell Regulation, 2:965-978, Dec 1991. URL: https://doi.org/10.1091/mbc.2.12.965, doi:10.1091/mbc.2.12.965. This article has 664 citations.
4. (cobbUnknownyearmapkinasepathways pages 1-2): MH Cobb AS Karra. Map kinase pathways: functions and modulation. Unknown journal, Unknown year.
5. (coulombe2007atypicalmitogenactivatedprotein pages 11-12): Phillipe Coulombe and Sylvain Meloche. Atypical mitogen-activated protein kinases: structure, regulation and functions. Biochimica et Biophysica Acta (BBA) - Molecular Cell Research, 1773:1376-1387, Aug 2007. URL: https://doi.org/10.1016/j.bbamcr.2006.11.001, doi:10.1016/j.bbamcr.2006.11.001. This article has 462 citations.
6. (coulombe2007atypicalmitogenactivatedprotein pages 4-6): Phillipe Coulombe and Sylvain Meloche. Atypical mitogen-activated protein kinases: structure, regulation and functions. Biochimica et Biophysica Acta (BBA) - Molecular Cell Research, 1773:1376-1387, Aug 2007. URL: https://doi.org/10.1016/j.bbamcr.2006.11.001, doi:10.1016/j.bbamcr.2006.11.001. This article has 462 citations.
7. (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.
8. (ronkina2019germlinedeletion pages 1-4): Natalia Ronkina, K. Schuster‐Gossler, F. Hansmann, Heike Kunze-Schumacher, I. Sandrock, Tatiana Yakovleva, Juri Lafera, Wolfgang Baumgärtner, Andreas Krueger, I. Prinz, Achim Gossler, A. Kotlyarov, and Matthias Gaestel. Germ line deletion reveals a nonessential role of atypical mitogen-activated protein kinase 6/extracellular signal-regulated kinase 3. Molecular and Cellular Biology, Mar 2019. URL: https://doi.org/10.1128/mcb.00516-18, doi:10.1128/mcb.00516-18. This article has 17 citations and is from a domain leading peer-reviewed journal.
9. (ronkina2019germlinedeletion pages 20-23): Natalia Ronkina, K. Schuster‐Gossler, F. Hansmann, Heike Kunze-Schumacher, I. Sandrock, Tatiana Yakovleva, Juri Lafera, Wolfgang Baumgärtner, Andreas Krueger, I. Prinz, Achim Gossler, A. Kotlyarov, and Matthias Gaestel. Germ line deletion reveals a nonessential role of atypical mitogen-activated protein kinase 6/extracellular signal-regulated kinase 3. Molecular and Cellular Biology, Mar 2019. URL: https://doi.org/10.1128/mcb.00516-18, doi:10.1128/mcb.00516-18. This article has 17 citations and is from a domain leading peer-reviewed journal.
10. (tang1998theroleof pages 31-35): P. Tang. The role of mitogen-activated protein kinases in listeria monocytogenes invasion. Unknown journal, 1998. URL: https://doi.org/10.14288/1.0088760, doi:10.14288/1.0088760. This article has 0 citations.
11. (cargnello2011activationandfunction pages 2-4): Marie Cargnello and Philippe P. Roux. Activation and function of the mapks and their substrates, the mapk-activated protein kinases. Microbiology and Molecular Biology Reviews, 75:50-83, Mar 2011. URL: https://doi.org/10.1128/mmbr.00031-10, doi:10.1128/mmbr.00031-10. This article has 3987 citations and is from a domain leading peer-reviewed journal.
12. (cargnello2011activationandfunction pages 25-26): Marie Cargnello and Philippe P. Roux. Activation and function of the mapks and their substrates, the mapk-activated protein kinases. Microbiology and Molecular Biology Reviews, 75:50-83, Mar 2011. URL: https://doi.org/10.1128/mmbr.00031-10, doi:10.1128/mmbr.00031-10. This article has 3987 citations and is from a domain leading peer-reviewed journal.
13. (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.
14. (kultz1998phylogeneticandfunctional pages 5-9): 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.
15. (zhu1994cloningandcharacterization pages 9-10): Andrew X. Zhu, Yi Zhao, David E. Moller, and Jeffrey S. Flier. Cloning and characterization of p97mapk, a novel human homolog of rat erk-3. Molecular and Cellular Biology, 14:8202-8211, Dec 1994. URL: https://doi.org/10.1128/mcb.14.12.8202-8211.1994, doi:10.1128/mcb.14.12.8202-8211.1994. This article has 95 citations and is from a domain leading peer-reviewed journal.
16. (janulis2001anovelmitogenactivated pages 13-13): Mark Janulis, Nicholas Trakul, Geoffrey Greene, Erik M. Schaefer, J. D. Lee, and Marsha Rich Rosner. A novel mitogen-activated protein kinase is responsive to raf and mediates growth factor specificity. Molecular and Cellular Biology, 21:2235-2247, Mar 2001. URL: https://doi.org/10.1128/mcb.21.6.2235-2247.2001, doi:10.1128/mcb.21.6.2235-2247.2001. This article has 22 citations and is from a domain leading peer-reviewed journal.
17. (moustardas2023mapkpathwaysin pages 1-3): Petros Moustardas, Daniel Aberdam, and Neil Lagali. Mapk pathways in ocular pathophysiology: potential therapeutic drugs and challenges. Cells, 12:617, Feb 2023. URL: https://doi.org/10.3390/cells12040617, doi:10.3390/cells12040617. This article has 37 citations and is from a peer-reviewed journal.
18. (tsai2010intracellularsignalingpathways pages 47-51): J Tsai. Intracellular signaling pathways regulating hepatic apolipoprotein b100 production: roles of mitogen-activated protein kinases (mapks) and inhibitor of nfkappab …. Unknown journal, 2010.
19. (turjanski2007mapkinasesand pages 1-2): A G Turjanski, J P Vaqué, and J S Gutkind. Map kinases and the control of nuclear events. Oncogene, 26:3240-3253, May 2007. URL: https://doi.org/10.1038/sj.onc.1210415, doi:10.1038/sj.onc.1210415. This article has 564 citations and is from a domain leading peer-reviewed journal.
20. (widmann1999mitogenactivatedproteinkinase pages 1-2): CHRISTIAN WIDMANN, SPENCER GIBSON, MATTHEW B. JARPE, and GARY L. JOHNSON. Mitogen-activated protein kinase: conservation of a three-kinase module from yeast to human. Physiological Reviews, 79:143-180, Jan 1999. URL: https://doi.org/10.1152/physrev.1999.79.1.143, doi:10.1152/physrev.1999.79.1.143. This article has 3790 citations and is from a highest quality peer-reviewed journal.
21. (chiu2001investigationofphosphatidylinositol3kinase pages 25-30): Doris Chiu. Investigation of phosphatidylinositol-3-kinase (pi3k) and extracellular signal-regulated kinase 1/2 (erk1/2) activation. Unknown journal, 2001. URL: https://doi.org/10.14288/1.0090096, doi:10.14288/1.0090096. This article has 0 citations.
22. (ho2000theactivationof pages 30-36): JMY Ho. The activation of mitogen-activated protein kinase pathways by the tel-jak2 oncoprotein. Unknown journal, 2000.