1. Phylogeny  
   MAPK6, commonly known as ERK3 or MAP kinase isoform p97, belongs to the atypical subclass of the MAPK family that distinguishes itself from classical MAPKs by possessing an unusual activation loop motif and unique regulatory features. Unlike conventional MAPKs such as ERK1/2 that are activated via a canonical three‐tier cascade involving MAP3K, MAP2K, and MAPK, ERK3 is characterized by the presence of a single phosphorylation acceptor within its Ser-Glu-Gly (SEG) activation motif. This atypical motif not only sets it apart functionally but also defines its evolutionary trajectory. Phylogenetic analyses indicate that ERK3/ MAPK6 is restricted to vertebrates, suggesting that its emergence is a relatively recent adaptation compared to the broadly conserved classical MAPKs found in all eukaryotes. Comparative studies have revealed that its kinase domain shares approximately 50% homology with conventional ERK1, yet its domain architecture and regulatory sequences such as the conserved C34 domain (shared with ERK4) highlight a divergence that is maintained across mammalian species and possibly other vertebrates (akunapuram2023regulationoferk3 pages 15-18, al2015identificationofnovel pages 19-23). Furthermore, experiments in which ERK3 is genetically ablated in animal models underscore its evolutionary conservation and developmental importance, indicating that despite being “atypical” from a regulatory mechanism standpoint, it represents an integral element of the MAPK signalling network conserved from the early vertebrate lineage (aldharee2017roleoferk3c pages 7-15, barbagallo2018exploringtherolesa pages 15-19). Thus, ERK3/MAPK6 is phylogenetically embedded within the CMGC group of kinases, sharing its broader classification with cyclin-dependent kinases, glycogen synthase kinases, and casein kinases, while its specific evolutionary adaptations reflect its specialized roles in vertebrate cellular physiology (albuquerque2024identificationdenouveaux pages 68-71).
2. Reaction Catalyzed  
   ERK3 catalyzes the ATP-dependent transfer of a phosphate group to serine and threonine residues on its substrates, operating as a serine/threonine kinase. The classical reaction it mediates is of the form: ATP + [target protein] – OH → ADP + [target protein] – O‑phosphate + H⁺. In particular, ERK3 phosphorylates two well-documented substrates: microtubule-associated protein 2 (MAP2) and MAPK-activated protein kinase 5 (MAPKAPK5). Upon interaction with MAPKAPK5, ERK3 is phosphorylated on its activation loop residue Ser-189; this event is critical because it not only serves as a switch for activating ERK3 but also triggers subsequent phosphorylation events whereby ERK3 then phosphorylates and activates MAPKAPK5. Thereafter, MAPKAPK5 reciprocates the process by phosphorylating ERK3, establishing a regulatory feedback loop. Although the precise stoichiometry and kinetic parameters of these phosphorylation events are not fully characterized, the mechanism is understood to populate a sequential series of events wherein substrate recognition, binding, and catalysis proceed in an ordered manner resulting in cellular responses such as cell cycle progression (akunapuram2023regulationoferk3 pages 8-15, al2015identificationofnovelb pages 19-23).
3. Cofactor Requirements  
   The catalytic activity of ERK3, like that of most serine/threonine kinases, is dependent on the presence of divalent metal ions that serve as essential cofactors for effective ATP binding and phosphotransfer. In particular, Mg²⁺ is the central cofactor presumed to coordinate the ATP molecule in the enzyme’s catalytic site by stabilizing the negative charges of the phosphate groups, thereby facilitating nucleophilic attack during the transfer reaction. Although detailed studies on the potential involvement of other metal ions such as Mn²⁺ are not elaborated in the current literature, the structural conservation observed in the kinase domains of similarly related MAPKs strongly suggests that Mg²⁺ is the primary cofactor required for ERK3 activity. Additionally, conserved motifs within the kinase domain, such as the AXK motif that includes a key catalytic lysine, contribute to the coordination of ATP and ensure the integrity of the catalytic mechanism (dahm2025atypicalmapksin pages 1-3, akunapuram2023regulationoferk3 pages 15-18).
4. Substrate Specificity  
   The substrate specificity of ERK3 distinguishes it from classical MAPKs through its restricted repertoire of physiological targets and the reliance on specific protein–protein interactions for substrate recognition. Notable substrates for ERK3 include microtubule-associated protein 2 (MAP2), which plays an instrumental role in modulating microtubule dynamics and cytoskeletal organization, and MAPK-activated protein kinase 5 (MAPKAPK5), also known as MK5 or PRAK, which forms a stable complex with ERK3. In this complex, the initial binding to MAPKAPK5 triggers phosphorylation of ERK3 at the activation loop residue Ser-189, an event necessary for the activation of MAPKAPK5. The reciprocal nature of this interaction, where MAPKAPK5 also phosphorylates ERK3, strongly implies a tightly coordinated regulatory mechanism governing downstream signal transduction processes such as cell cycle entry and cytoskeletal rearrangement (akunapuram2023regulationoferk3 pages 8-15, bi2018receptorlikecytoplasmickinases pages 18-21). Although a consensus substrate motif exclusive for ERK3 has not been definitively delineated in the literature, its substrate recognition appears to involve docking sequences and structural features that differ from the classical D-domain interactions observed in other MAPKs. This specificity is further reinforced by the observation that ERK3 does not efficiently phosphorylate a broad array of conventional MAPK substrates like c-Jun or MyoD, instead demonstrating a narrow substrate range that supports its specialized role in cell cycle progression and cytoskeletal control (boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 1-4, akunapuram2023regulationoferk3 pages 15-18).
5. Structure  
   The structural organization of ERK3 is emblematic of the MAP kinase family yet incorporates several atypical features that distinguish it from classic kinases such as ERK1/2. The protein can be broadly divided into three main segments:  
    • The N-terminal kinase domain: This globular domain, comprising approximately 720 amino acids overall with about 50% homology to ERK1’s catalytic domain, harbors the catalytic core responsible for ATP binding and phosphate transfer. Key to its activity is the atypical activation loop, which, unlike the canonical TXY motif found in conventional MAPKs, contains a single phosphorylatable site within the SEG motif (Ser-189). This residue is critical for kinase activity and modulates substrate binding, particularly in the context of its interaction with MAPKAPK5 (akunapuram2023regulationoferk3 pages 15-18, dahm2025atypicalmapksin pages 1-3).  
    • The conserved C34 domain: Unique to the ERK3/ERK4 subfamily, the C34 domain is highly conserved among vertebrates and is absent from classical MAP kinases. Although its specific function remains incompletely defined, the C34 region is implicated in mediating protein–protein interactions that are essential for the stable formation of complexes, such as that observed with MAPKAPK5, and may contribute to subcellular localization dynamics (al2015identificationofnovel pages 19-23, barbagallo2018exploringtherolesa pages 19-24).  
    • The extended C-terminal tail: This region is characterized by a high concentration of serine/threonine residues, which serve as potential sites for additional phosphorylation and regulatory modifications. The C-terminal tail is believed to play a significant role in modulating kinase stability, protein–protein interactions, and possibly intracellular trafficking. Structural modeling and crystallographic studies suggest that the atypical nature of the activation loop combined with the extended tail imparts ERK3 with its characteristic regulatory behavior distinct from the more rigidly structured classical MAPKs (akunapuram2023regulationoferk3 pages 15-18, albuquerque2024identificationdenouveauxa pages 68-71).  
   Overall, the bilobal architecture common to serine/threonine kinases is maintained, with the N-terminal lobe primarily binding ATP and the C-terminal lobe contributing to substrate specificity. However, the divergence in the activation loop, the presence of the C34 domain, and the extended C-terminal tail are structural hallmarks that underlie the unique functional properties of ERK3 (barbagallo2018exploringtheroles pages 15-19, elkhadragy2017regulationofthe pages 17-22).
6. Regulation  
   The regulation of ERK3 is multifaceted and encompasses a spectrum of post-translational modifications and protein–protein interactions that fine-tune its activity, stability, and localization. One of the central regulatory events is the phosphorylation of the activation loop, specifically at the Ser-189 residue located in the SEG motif. This phosphorylation is critical for activating ERK3’s kinase activity, and it is mediated either via autophosphorylation or by upstream kinases such as group I p21-activated kinases (PAKs) (akunapuram2023regulationoferk3 pages 15-18, aldharee2017roleoferk3c pages 7-15). Once phosphorylated, ERK3 can engage in a reciprocal relationship with MAPKAPK5; binding of MAPKAPK5 leads to further phosphorylation of ERK3 and, in return, ERK3 phosphorylates and activates MAPKAPK5, establishing a regulatory complex that is thought to promote cell cycle entry and other downstream signaling events (boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 12-15, barbagallo2018exploringtheroles pages 19-24).  
   In addition to phosphorylation, ERK3 is subject to ubiquitination-mediated proteolysis, which contributes to its notably short half-life in proliferating cells. Specific motifs within its N-terminal region act as degradation signals, and ubiquitination targets ERK3 for proteasomal degradation. The stability of ERK3 is counteracted by deubiquitinating enzymes such as USP20, which remove ubiquitin chains and thereby stabilize the kinase, ensuring its availability for signaling functions such as promoting cell migration (elkhadragy2019aradioactivein pages 7-9, barbagallo2018exploringtheroles pages 74-76).  
   Furthermore, additional regulatory phosphorylation events have been identified within the extended C-terminal tail of ERK3. These phosphorylations, occurring on multiple serine/threonine residues, are often cell cycle–dependent and correlate with enhanced protein stability during mitosis. The precise kinases involved in modifying the C-terminal tail are still under investigation, but evidence suggests that cyclin-dependent kinases such as Cyclin B-Cdk1 may be involved, with subsequent dephosphorylation by phosphatases potentially triggering degradation upon mitotic exit (elkhadragy2024roleofthe pages 15-16, barbagallo2018exploringtherolesa pages 15-19).  
   Subcellular localization also plays a key role in regulating ERK3 function. Although ERK3 lacks classical nuclear localization signals, its distribution between the nucleus and cytoplasm is controlled by its regulatory domains and may be affected by post-translational modifications. The binding of ERK3 to proteins such as MAPKAPK5 has been implicated in facilitating cytoplasmic retention, while its phosphorylation status can influence nuclear export signals, with CRM1-dependent mechanisms mediating nuclear-cytoplasmic shuttling (elkhadragy2024roleofthe pages 11-13, bi2018receptorlikecytoplasmickinases pages 18-21).  
   Collectively, the regulation of ERK3 is emblematic of an atypical kinase that integrates multiple layers of control—from activation loop phosphorylation and reciprocal kinase interactions to ubiquitin-mediated degradation and spatial localization effects—to ensure that its signaling output is precise and context-dependent (akunapuram2023regulationoferk3 pages 15-18, dahm2025atypicalmapksin pages 1-3).
7. Function  
   ERK3 exerts its biological functions through a combination of its enzymatic activity and its capacity to form multi-kinase complexes. One of the primary roles of ERK3 appears to be the regulation of cell cycle progression. The reciprocal phosphorylation cycle established between ERK3 and MAPKAPK5 is believed to drive cell cycle entry by triggering signaling pathways that transition cells from quiescence to proliferation. Despite remaining “atypical” in its activation relative to canonical MAPKs, the ERK3–MAPKAPK5 complex has been functionally associated with promoting mitogenic responses (akunapuram2023regulationoferk3 pages 15-18, alsaran2016functionalcharacterizationof pages 8-15).  
   In addition to its cell cycle regulatory role, ERK3 has been implicated in the control of cytoskeletal dynamics, a function closely linked to its phosphorylation of substrates such as MAP2. By phosphorylating MAP2, ERK3 can influence microtubule organization and, by extension, regulate processes such as cell morphology, migration, and potentially invasion. This function is particularly relevant in the context of cancer, where increased ERK3 expression and activity have been correlated with enhanced cell motility and metastatic phenotypes in tumor models (boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 1-4, barbagallo2018exploringtheroles pages 15-19).  
   Moreover, ERK3 is widely expressed in various tissues including skeletal muscle, brain, and multiple epithelial cell types, indicating its involvement in diverse physiological processes. Its developmental regulation, as evidenced by increasing mRNA levels during embryogenesis followed by tissue-specific expression in adults, suggests that ERK3 contributes to tissue differentiation and organ maturation. Genetic studies in model organisms have demonstrated that perturbations in ERK3 expression can lead to defects in tissue development and homeostasis (aldharee2017roleoferk3c pages 7-15, elkhadragy2024roleofthe pages 1-2).  
   Beyond cell cycle and cytoskeletal regulation, ERK3 has also been implicated in broader signal transduction networks. Its interactions with coactivators and transcriptional regulators, such as the interplay with MAPKAPK5 and potential indirect effects on proteins like SRC-3, suggest a role in integrating mitogenic and stress signals. This places ERK3 as a nodal point where atypical MAPK signaling converges with other pathways that regulate cellular responses to environmental cues, thereby impacting proliferation, differentiation, and survival (barbagallo2018exploringtheroles pages 19-24, akunapuram2023regulationoferk3 pages 8-15).  
   Finally, while ERK3 is predominantly studied in the context of cancer due to its association with increased invasiveness and altered signaling in tumor cells, its roles in immune cell function and inflammatory signaling are also emerging. For instance, there is evidence to suggest that ERK3 may influence thymocyte development and T-cell activation, further underlining its involvement in discrete aspects of cell regulation beyond cytoskeletal dynamics and proliferation (elkhadragy2017regulationofthe pages 22-26, albuquerque2024identificationdenouveauxa pages 68-71).
8. Other Comments  
   Several additional facets of ERK3 regulation and function warrant further discussion. First, the development of selective inhibitors for ERK3 remains in its early stages. Owing to its atypical activation mechanism and the unique features of its ATP-binding pocket and regulatory domains, standard kinase inhibitors display off-target effects when applied to ERK3. Current efforts are focused on identifying molecules that can selectively target the unique structural features of ERK3 without broadly inhibiting other MAPK family members; however, such compounds are not yet widely available in clinical practice (albuquerque2024identificationdenouveaux pages 68-71, schroder2020crystalstructureand pages 10-12).  
   Second, disease associations of ERK3 are becoming increasingly evident, particularly in oncology. Increased expression and dysregulated activity of ERK3 have been observed in various cancers, including lung adenocarcinoma and breast cancer, where its kinase activity is linked to enhanced cell migration, invasiveness, and potential chemoresistance. Furthermore, certain cancer-associated mutations, such as alterations in the L290 residue, have been reported to influence both the subcellular localization and functional output of ERK3. These mutations may enhance cytosolic localization, thereby promoting interactions with cytoplasmic substrates and signaling molecules that drive oncogenic processes (elkhadragy2024roleofthe pages 11-13, barbagallo2018exploringtheroles pages 74-76).  
   Third, the interplay between phosphorylation and ubiquitination in regulating ERK3 stability offers promising avenues for therapeutic intervention. The rapid turnover of ERK3, mediated by ubiquitination events at its N-terminal regions, is counterbalanced by deubiquitinating enzymes such as USP20. This balance is critical for maintaining appropriate levels of active kinase in the cell and suggests that modulation of these regulatory processes could be exploited to alter ERK3 activity in pathological conditions (elkhadragy2019aradioactivein pages 7-9, barbagallo2018exploringtherolesa pages 15-19).  
   Finally, current research is oriented toward elucidating the broader network of interacting proteins and the complete spectrum of downstream substrates associated with ERK3. High-throughput proteomic approaches coupled with advanced data analysis techniques are being employed to map the transient phosphorylation events coordinated by ERK3, with the goal of fully integrating its role within the complex signaling milieu of the cell. These studies are critical for uncovering novel interactions and identifying additional regulatory checkpoints that could serve as targets for therapeutic intervention in diseases where ERK3 signaling is aberrant (akunapuram2023regulationoferk3 pages 15-18, boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 12-15).
9. References  
   akunapuram2023regulationoferk3 pages 15-18; akunapuram2023regulationoferk3 pages 8-15; al2015identificationofnovel pages 19-23; al2015identificationofnovelb pages 19-23; albuquerque2024identificationdenouveaux pages 68-71; albuquerque2024identificationdenouveauxa pages 68-71; aldharee2017roleoferk3c pages 7-15; alsaran2016functionalcharacterizationof pages 8-15; barbagallo2018exploringtheroles pages 15-19; barbagallo2018exploringtheroles pages 19-24; barbagallo2018exploringtheroles pages 74-76; barbagallo2018exploringtherolesa pages 10-15; barbagallo2018exploringtherolesa pages 15-19; barbagallo2018exploringtherolesa pages 19-24; bi2018receptorlikecytoplasmickinases pages 18-21; boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 1-4; boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 12-15; dahm2025atypicalmapksin pages 1-3; elkhadragy2017regulationofthe pages 17-22; elkhadragy2017regulationofthe pages 22-26; elkhadragy2017regulationofthe pages 26-31; elkhadragy2017regulationofthe pages 35-39; elkhadragy2019aradioactivein pages 7-9; elkhadragy2024roleofthe pages 1-2; elkhadragy2024roleofthe pages 11-13; elkhadragy2024roleofthe pages 15-16; elkhadragy2024roleofthe pages 2-4; huang2024reconstructingthedeep pages 1-3.

References

1. (akunapuram2023regulationoferk3 pages 15-18): S Akunapuram. Regulation of erk3 by kras signalling and its role in the growth of lung adenocarcinoma (luad) cells. Unknown journal, 2023.
2. (akunapuram2023regulationoferk3 pages 8-15): S Akunapuram. Regulation of erk3 by kras signalling and its role in the growth of lung adenocarcinoma (luad) cells. Unknown journal, 2023.
3. (al2015identificationofnovel pages 19-23): R Al. Identification of novel roles and new modes of regulation for the atypical map kinases erk3 and erk4. Unknown journal, 2015.
4. (al2015identificationofnovelb pages 19-23): R Al. Identification of novel roles and new modes of regulation for the atypical map kinases erk3 and erk4. Unknown journal, 2015.
5. (albuquerque2024identificationdenouveaux pages 68-71): F Nullans De Albuquerque. Identification de nouveaux mécanismes de régulation de la mapk atypique erk3. Unknown journal, 2024.
6. (albuquerque2024identificationdenouveauxa pages 68-71): F Nullans De Albuquerque. Identification de nouveaux mécanismes de régulation de la mapk atypique erk3. Unknown journal, 2024.
7. (aldharee2017roleoferk3c pages 7-15): HA Aldharee. Role of erk3 in regulating rhogdi1-paks signaling axis. Unknown journal, 2017.
8. (alsaran2016functionalcharacterizationof pages 8-15): HM Alsaran. Functional characterization of cancer-related mutations of erk3. Unknown journal, 2016.
9. (barbagallo2018exploringtheroles pages 15-19): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
10. (barbagallo2018exploringtheroles pages 19-24): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
11. (barbagallo2018exploringtheroles pages 74-76): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
12. (barbagallo2018exploringtherolesa pages 10-15): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
13. (barbagallo2018exploringtherolesa pages 15-19): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
14. (barbagallo2018exploringtherolesa pages 19-24): M Barbagallo. Exploring the roles of atypical map kinases erk3 and erk4 during inflammation. Unknown journal, 2018.
15. (bi2018receptorlikecytoplasmickinases pages 18-21): Guozhi Bi, Zhaoyang Zhou, Weibing Wang, Lin Li, Shaofei Rao, Ying Wu, Xiaojuan Zhang, Frank L. H. Menke, She Chen, and Jian-Min Zhou. Receptor-like cytoplasmic kinases directly link diverse pattern recognition receptors to the activation of mitogen-activated protein kinase cascades in arabidopsis. The Plant Cell, 30:1543-1561, Jun 2018. URL: https://doi.org/10.1105/tpc.17.00981, doi:10.1105/tpc.17.00981. This article has 316 citations.
16. (boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 1-4): Katarzyna Bogucka-Janczi, Gregory Harms, Mary May-Coissieux, Mohamad Bentires-Alj, Bernd Thiede, and Krishnaraj Rajalingam. Erk3/mapk6 dictates cdc42/rac1 activity and arp2/3-dependent actin polymerization. BioRxiv, Oct 2023. URL: https://doi.org/10.1101/2022.10.12.511969, doi:10.1101/2022.10.12.511969. This article has 21 citations.
17. (boguckajanczi2023erk3mapk6dictatescdc42rac1 pages 12-15): Katarzyna Bogucka-Janczi, Gregory Harms, Mary May-Coissieux, Mohamad Bentires-Alj, Bernd Thiede, and Krishnaraj Rajalingam. Erk3/mapk6 dictates cdc42/rac1 activity and arp2/3-dependent actin polymerization. BioRxiv, Oct 2023. URL: https://doi.org/10.1101/2022.10.12.511969, doi:10.1101/2022.10.12.511969. This article has 21 citations.
18. (dahm2025atypicalmapksin pages 1-3): Katrin Dahm, Parthiban Vijayarangakannan, Hans‐Peter Wollscheid, Hansjörg Schild, and Krishnaraj Rajalingam. Atypical mapks in cancer. The FEBS Journal, Sep 2025. URL: https://doi.org/10.1111/febs.17283, doi:10.1111/febs.17283. This article has 1 citations.
19. (elkhadragy2017regulationofthe pages 17-22): L Elkhadragy. Regulation of the expression and activity of extracellular signal-regulated kinase 3 (erk3). Unknown journal, 2017.
20. (elkhadragy2017regulationofthe pages 22-26): L Elkhadragy. Regulation of the expression and activity of extracellular signal-regulated kinase 3 (erk3). Unknown journal, 2017.
21. (elkhadragy2017regulationofthe pages 26-31): L Elkhadragy. Regulation of the expression and activity of extracellular signal-regulated kinase 3 (erk3). Unknown journal, 2017.
22. (elkhadragy2017regulationofthe pages 35-39): L Elkhadragy. Regulation of the expression and activity of extracellular signal-regulated kinase 3 (erk3). Unknown journal, 2017.
23. (elkhadragy2019aradioactivein pages 7-9): Lobna Elkhadragy and Weiwen Long. A radioactive in vitro erk3 kinase assay. Bio-protocol, Aug 2019. URL: https://doi.org/10.21769/bioprotoc.3332, doi:10.21769/bioprotoc.3332. This article has 5 citations and is from a poor quality or predatory journal.
24. (elkhadragy2024roleofthe pages 1-2): L. Elkhadragy, Amanda K Myers, and Weiwen Long. Role of the atypical mapk erk3 in cancer growth and progression. Cancers, Mar 2024. URL: https://doi.org/10.3390/cancers16071381, doi:10.3390/cancers16071381. This article has 1 citations and is from a peer-reviewed journal.
25. (elkhadragy2024roleofthe pages 11-13): L. Elkhadragy, Amanda K Myers, and Weiwen Long. Role of the atypical mapk erk3 in cancer growth and progression. Cancers, Mar 2024. URL: https://doi.org/10.3390/cancers16071381, doi:10.3390/cancers16071381. This article has 1 citations and is from a peer-reviewed journal.
26. (elkhadragy2024roleofthe pages 15-16): L. Elkhadragy, Amanda K Myers, and Weiwen Long. Role of the atypical mapk erk3 in cancer growth and progression. Cancers, Mar 2024. URL: https://doi.org/10.3390/cancers16071381, doi:10.3390/cancers16071381. This article has 1 citations and is from a peer-reviewed journal.
27. (elkhadragy2024roleofthe pages 2-4): L. Elkhadragy, Amanda K Myers, and Weiwen Long. Role of the atypical mapk erk3 in cancer growth and progression. Cancers, Mar 2024. URL: https://doi.org/10.3390/cancers16071381, doi:10.3390/cancers16071381. This article has 1 citations and is from a peer-reviewed journal.
28. (huang2024reconstructingthedeep pages 1-3): EJ Huang, Jeeun Parksong, Amy F. Peterson, Fernando Torres, Sergi Regot, and Gabriel S. Bever. Reconstructing the deep phylogeny of the mapk signaling network: functional specialization via multi-tier coevolutionary expansion. BioRxiv, Oct 2024. URL: https://doi.org/10.1101/2024.10.01.616093, doi:10.1101/2024.10.01.616093. This article has 0 citations.