1. Phylogeny  
   Mitogen‐activated protein kinase 9 (MAPK9), widely known as JNK2, is a member of the c‐Jun N‐terminal kinase (JNK) subgroup within the broader MAP kinase (MAPK) family. JNK2 is classified within the CMGC group of protein kinases, which includes cyclin‐dependent kinases (CDKs), glycogen synthase kinases (GSKs), and CLKs. Its evolutionary origin can be traced back to early eukaryotes, with orthologous proteins identified across a wide range of species. In vertebrates, three closely related paralogs exist—JNK1, JNK2, and JNK3—arising from whole‐genome duplication events. Among these, JNK2 is ubiquitously expressed, in contrast to the more tissue‐restricted expression of JNK3, which is primarily neuronal. JNK2 has been preserved in mammalian species, reflecting its critical regulatory function in cellular stress responses and apoptotic signaling (orand2023revealingthemechanism pages 33-38, zeke2016jnksignalingregulation pages 6-7).
2. Reaction Catalyzed  
   MAPK9 catalyzes the transfer of the γ‐phosphate group from ATP to hydroxyl groups on serine or threonine residues of substrate proteins. The reaction can be summarized as follows: ATP + [protein]–(L‐serine or L‐threonine) → ADP + [protein]–phosphoserine/threonine + H⁺. This phosphorylation event is central to the activation or modulation of downstream targets involved in transcription regulation, apoptosis, and other stress responses. In particular, MAPK9 phosphorylates key transcription factors such as c‐Jun and ATF2, thereby regulating the activity of the AP-1 complex. In addition, under conditions of oxidative or ribotoxic stress, MAPK9 phosphorylates the RNA polymerase I transcription initiation factor RRN3 leading to inhibition of rRNA synthesis (ha2019phosphorylationdynamicsof pages 13-15, sun2016themitogenactivatedprotein pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of MAPK9 is ATP-dependent and it requires divalent metal ions for proper enzymatic function. Typically, Mg²⁺ is essential as a cofactor because it coordinates with the phosphate groups of ATP to facilitate the phosphoryl transfer reaction. In some experimental systems, Mn²⁺ may substitute for Mg²⁺; however, Mg²⁺ is considered the physiologically relevant ion. This cofactor dependence is common among serine/threonine kinases and is critical for the precise alignment of substrates within the active site (sun2016themitogenactivatedprotein pages 1-2, han2019phosphorylationdynamicsof pages 1-3).
4. Substrate Specificity  
   MAPK9 exhibits substrate specificity dictated primarily by its recognition of proline-directed serine/threonine motifs. Physiologically, its best-characterized substrates include the transcription factors c-Jun and ATF2. The kinase phosphorylates serine residues located adjacent to proline in these proteins, a hallmark of the substrate preference shared by MAP kinases. In addition to canonical AP-1 components, MAPK9 also phosphorylates other proteins such as p53, Bcl-2, and components linked to RRN3 function, thereby influencing apoptosis, cell cycle progression, and transcriptional regulation. While the core substrate motif is a serine/threonine followed by a proline, docking interactions mediated by short linear motifs (D-motifs) in substrates are also critical for modulating binding affinity and subsequent phosphorylation efficiency (orand2023revealingthemechanism pages 298-299, zeke2016jnksignalingregulation pages 10-13).
5. Structure  
   Structurally, MAPK9 conforms to the classic MAP kinase fold that comprises a bi-lobal catalytic domain. The N-terminal lobe is β-sheet rich and contains the glycine-rich loop (G-loop), which is involved in ATP binding, whereas the C-terminal lobe is predominantly α-helical and houses the substrate binding and catalytic regions. A defining structural feature of MAPK9 is the activation loop that contains a Thr-Pro-Tyr (TPY) motif – dual phosphorylation at these threonine and tyrosine residues is necessary for activation. High-resolution crystallographic studies of related JNK isoforms provide insights into the conserved docking grooves and common docking (CD) regions that are critical for substrate and regulatory protein interactions. Furthermore, alternative splicing of MAPK9 generates isoforms (such as the p46 and p54 variants) that can differ in the length of their regulatory domains, imparting subtle variations in catalytic efficiency and substrate selectivity (ha2019phosphorylationdynamicsof pages 3-6, zeke2016jnksignalingregulation pages 8-10, orand2023revealingthemechanism pages 41-45).
6. Regulation  
   MAPK9 is regulated through a complex interplay of phosphorylation–dephosphorylation events, scaffold-mediated interactions, and subcellular localization dynamics. The kinase is activated by dual phosphorylation on residues within its TPY motif by upstream dual-specificity kinases MAP2K4 (MKK4) and MAP2K7 (MKK7). MKK4 preferentially phosphorylates the tyrosine residue, while MKK7 catalyzes phosphorylation at the threonine residue, and together these modifications shift MAPK9 to an active conformation. Negative regulation is mediated largely by dual-specificity phosphatases (DUSPs), such as DUSP1 and MKP1, which dephosphorylate MAPK9, thus attenuating its signaling. Additionally, scaffold proteins such as JNK-interacting protein 1 (JIP1) and POSH coordinate the assembly of MAPK9 with its upstream kinases and substrates, ensuring both spatial and temporal specificity in signaling. Post-translational modifications beyond phosphorylation, such as ubiquitination in response to cellular stress signals, further modulate the stability and localization of MAPK9 (ha2019phosphorylationdynamicsof pages 16-18, orand2023revealingthemechanism pages 175-178, rehfeldt2020cjunnterminalkinase pages 27-29).
7. Function  
   MAPK9 plays multifaceted roles in cellular processes that are central to both normal physiology and pathophysiological states. Its primary function is to modulate gene expression by phosphorylating transcription factors, thus altering cell behavior in response to extracellular stress stimuli such as pro-inflammatory cytokines, UV irradiation, and oxidative stress. Through activation of the AP-1 complex (via phosphorylation of c-Jun and ATF2), MAPK9 regulates cell proliferation, differentiation, and programmed cell death (apoptosis). In stressed cells, MAPK9 also phosphorylates factors like p53 and YAP1, promoting apoptosis to prevent the propagation of damaged cells. In T lymphocytes, MAPK9, together with MAPK8 (JNK1), directs the differentiation of T-helper cells into a Th1 phenotype subsequent to T cell receptor stimulation mediated by complex assembly with CARMA1 and BCL10. Moreover, MAPK9 is implicated in cytoskeletal regulation—as in the disruption of epithelial tight junctions induced by osmotic stress—as well as in the regulation of canonical signaling pathways such as the Wnt/β-catenin cascade, where its activity leads to beta-catenin degradation. Additional roles include modulating circadian rhythm through phosphorylation of the CLOCK-BMAL1 heterodimer and influencing neurite outgrowth in spiral ganglion neurons. These diverse functions underscore the centrality of MAPK9 in cellular stress responses, immune regulation, and developmental processes (ha2019phosphorylationdynamicsof pages 13-15, orand2023revealingthemechanism pages 298-299, yan2024theroleof pages 19-20).
8. Other Comments  
   From a therapeutic perspective, MAPK9 is an attractive target given its central role in pathways that regulate apoptosis, inflammation, and cellular stress responses. Aberrant MAPK9 activity has been implicated in a variety of disease states, including cancer, neurodegenerative disorders, metabolic diseases, and autoimmune conditions. As a result, several small-molecule inhibitors, such as ATP-competitive agents like SP600125, as well as compounds disrupting MAPK9 interactions with scaffold proteins, are actively being investigated. However, the high degree of conservation in the ATP-binding sites among JNK isoforms poses a challenge for the development of isoform-selective inhibitors. Furthermore, studies using chemical probes and structure-guided drug design have provided valuable insights into the molecular determinants of inhibitor selectivity, with efforts underway to optimize lead compounds to minimize off-target effects (ansideri2018structuraloptimizationof pages 23-23, rehfeldt2020cjunnterminalkinase pages 11-13, shillingford2023mitogenactivatedproteinkinase pages 20-23). Notable among these efforts are clinical and preclinical studies exploring the use of JNK inhibitors for conditions such as Alzheimer’s disease, where reduced neuroinflammation via MAPK9 inhibition may prove beneficial. In addition to small-molecule interventions, regulation through modulation of upstream kinases (MKK4/MKK7) or enhancement of DUSP activity represents alternative therapeutic strategies. Ongoing research continues to dissect the complexities of MAPK9 signaling dynamics, its splicing isoform-specific roles, and the interplay of its regulatory networks in different cell types (orand2023revealingthemechanism pages 175-178, yan2024theroleof pages 22-23, zeke2016jnksignalingregulation pages 31-32).
9. References  
   ha2019phosphorylationdynamicsof pages 13-15;  
   ha2019phosphorylationdynamicsof pages 11-13;  
   ha2019phosphorylationdynamicsof pages 16-18;  
   ha2019phosphorylationdynamicsof pages 3-6;  
   ha2019phosphorylationdynamicsof pages 7-9;  
   orand2023revealingthemechanism pages 33-38;  
   orand2023revealingthemechanism pages 25-29;  
   orand2023revealingthemechanism pages 41-45;  
   rehfeldt2020cjunnterminalkinase pages 9-11;  
   rehfeldt2020cjunnterminalkinase pages 27-29;  
   sun2016themitogenactivatedprotein pages 1-2;  
   sun2016themitogenactivatedprotein pages 2-4;  
   yan2024theroleof pages 1-2;  
   yan2024theroleof pages 13-14;  
   yan2024theroleof pages 2-4;  
   yan2024theroleof pages 23-25;  
   yan2024theroleof pages 25-26;  
   yan2024theroleof pages 4-7;  
   yan2024theroleof pages 7-8;  
   yan2024theroleof pages 8-10;  
   zeke2016jnksignalingregulation pages 1-1;  
   zeke2016jnksignalingregulation pages 6-7;  
   zeke2016jnksignalingregulation pages 8-10;  
   zeke2016jnksignalingregulation pages 10-10;  
   zeke2016jnksignalingregulation pages 10-13;  
   zeke2016jnksignalingregulation pages 13-14;  
   zeke2016jnksignalingregulation pages 2-3;  
   zeke2016jnksignalingregulation pages 3-5;  
   zeke2016jnksignalingregulation pages 31-31;  
   zeke2016jnksignalingregulation pages 31-32;  
   zeke2016jnksignalingregulation pages 42-43;  
   zeke2016jnksignalingregulation pages 43-43;  
   ansideri2018structuraloptimizationof pages 23-23;  
   honzejkova2024structuralstudiesof pages 20-24;  
   honzejkova2024structuralstudiesof pages 106-108;  
   honzejkova2024structuralstudiesof pages 15-20;  
   shillingford2022insightsintothe pages 89-95.

References

1. (ha2019phosphorylationdynamicsof pages 13-15): Jain Ha, Eunjeong Kang, Jihye Seo, and Sayeon Cho. Phosphorylation dynamics of jnk signaling: effects of dual-specificity phosphatases (dusps) on the jnk pathway. International Journal of Molecular Sciences, 20:6157, Dec 2019. URL: https://doi.org/10.3390/ijms20246157, doi:10.3390/ijms20246157. This article has 74 citations and is from a peer-reviewed journal.
2. (orand2023revealingthemechanism pages 175-178): T Orand. Revealing the mechanism of action of intrinsically disordered proteins in mapk cell signalling. Unknown journal, 2023.
3. (orand2023revealingthemechanism pages 298-299): T Orand. Revealing the mechanism of action of intrinsically disordered proteins in mapk cell signalling. Unknown journal, 2023.
4. (orand2023revealingthemechanism pages 33-38): T Orand. Revealing the mechanism of action of intrinsically disordered proteins in mapk cell signalling. Unknown journal, 2023.
5. (rehfeldt2020cjunnterminalkinase pages 11-13): Stephanie Cristine Hepp Rehfeldt, Fernanda Majolo, Márcia Inês Goettert, and Stefan Laufer. C-jun n-terminal kinase inhibitors as potential leads for new therapeutics for alzheimer’s diseases. International Journal of Molecular Sciences, 21:9677, Dec 2020. URL: https://doi.org/10.3390/ijms21249677, doi:10.3390/ijms21249677. This article has 45 citations and is from a peer-reviewed journal.
6. (rehfeldt2020cjunnterminalkinase pages 27-29): Stephanie Cristine Hepp Rehfeldt, Fernanda Majolo, Márcia Inês Goettert, and Stefan Laufer. C-jun n-terminal kinase inhibitors as potential leads for new therapeutics for alzheimer’s diseases. International Journal of Molecular Sciences, 21:9677, Dec 2020. URL: https://doi.org/10.3390/ijms21249677, doi:10.3390/ijms21249677. This article has 45 citations and is from a peer-reviewed journal.
7. (yan2024theroleof pages 1-2): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
8. (yan2024theroleof pages 13-14): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
9. (yan2024theroleof pages 2-4): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
10. (yan2024theroleof pages 23-25): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
11. (yan2024theroleof pages 25-26): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
12. (yan2024theroleof pages 4-7): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
13. (yan2024theroleof pages 7-8): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
14. (yan2024theroleof pages 8-10): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
15. (zeke2016jnksignalingregulation pages 1-1): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
16. (ha2019phosphorylationdynamicsof pages 11-13): Jain Ha, Eunjeong Kang, Jihye Seo, and Sayeon Cho. Phosphorylation dynamics of jnk signaling: effects of dual-specificity phosphatases (dusps) on the jnk pathway. International Journal of Molecular Sciences, 20:6157, Dec 2019. URL: https://doi.org/10.3390/ijms20246157, doi:10.3390/ijms20246157. This article has 74 citations and is from a peer-reviewed journal.
17. (ha2019phosphorylationdynamicsof pages 16-18): Jain Ha, Eunjeong Kang, Jihye Seo, and Sayeon Cho. Phosphorylation dynamics of jnk signaling: effects of dual-specificity phosphatases (dusps) on the jnk pathway. International Journal of Molecular Sciences, 20:6157, Dec 2019. URL: https://doi.org/10.3390/ijms20246157, doi:10.3390/ijms20246157. This article has 74 citations and is from a peer-reviewed journal.
18. (ha2019phosphorylationdynamicsof pages 3-6): Jain Ha, Eunjeong Kang, Jihye Seo, and Sayeon Cho. Phosphorylation dynamics of jnk signaling: effects of dual-specificity phosphatases (dusps) on the jnk pathway. International Journal of Molecular Sciences, 20:6157, Dec 2019. URL: https://doi.org/10.3390/ijms20246157, doi:10.3390/ijms20246157. This article has 74 citations and is from a peer-reviewed journal.
19. (ha2019phosphorylationdynamicsof pages 7-9): Jain Ha, Eunjeong Kang, Jihye Seo, and Sayeon Cho. Phosphorylation dynamics of jnk signaling: effects of dual-specificity phosphatases (dusps) on the jnk pathway. International Journal of Molecular Sciences, 20:6157, Dec 2019. URL: https://doi.org/10.3390/ijms20246157, doi:10.3390/ijms20246157. This article has 74 citations and is from a peer-reviewed journal.
20. (honzejkova2024structuralstudiesof pages 20-24): K Honzejková. Structural studies of selected protein complexes involved in signal transduction. Unknown journal, 2024.
21. (orand2023revealingthemechanism pages 25-29): T Orand. Revealing the mechanism of action of intrinsically disordered proteins in mapk cell signalling. Unknown journal, 2023.
22. (orand2023revealingthemechanism pages 41-45): T Orand. Revealing the mechanism of action of intrinsically disordered proteins in mapk cell signalling. Unknown journal, 2023.
23. (rehfeldt2020cjunnterminalkinase pages 9-11): Stephanie Cristine Hepp Rehfeldt, Fernanda Majolo, Márcia Inês Goettert, and Stefan Laufer. C-jun n-terminal kinase inhibitors as potential leads for new therapeutics for alzheimer’s diseases. International Journal of Molecular Sciences, 21:9677, Dec 2020. URL: https://doi.org/10.3390/ijms21249677, doi:10.3390/ijms21249677. This article has 45 citations and is from a peer-reviewed journal.
24. (shillingford2022insightsintothe pages 89-95): SR Shillingford. Insights into the allosteric regulation and exploitation of the mapk phosphatases as therapeutic targets. Unknown journal, 2022.
25. (shillingford2023mitogenactivatedproteinkinase pages 20-23): Shanelle R. Shillingford and Anton M. Bennett. Mitogen-activated protein kinase phosphatases: no longer undruggable? Annual Review of Pharmacology and Toxicology, 63:617-636, Jan 2023. URL: https://doi.org/10.1146/annurev-pharmtox-051921-121923, doi:10.1146/annurev-pharmtox-051921-121923. This article has 20 citations and is from a highest quality peer-reviewed journal.
26. (sun2016themitogenactivatedprotein pages 1-2): Jing Sun and Guangxian Nan. The mitogen-activated protein kinase (mapk) signaling pathway as a discovery target in stroke. Journal of Molecular Neuroscience, 59:90-98, Feb 2016. URL: https://doi.org/10.1007/s12031-016-0717-8, doi:10.1007/s12031-016-0717-8. This article has 229 citations and is from a peer-reviewed journal.
27. (sun2016themitogenactivatedprotein pages 2-4): Jing Sun and Guangxian Nan. The mitogen-activated protein kinase (mapk) signaling pathway as a discovery target in stroke. Journal of Molecular Neuroscience, 59:90-98, Feb 2016. URL: https://doi.org/10.1007/s12031-016-0717-8, doi:10.1007/s12031-016-0717-8. This article has 229 citations and is from a peer-reviewed journal.
28. (yan2024theroleof pages 19-20): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
29. (yan2024theroleof pages 22-23): Huaying Yan, Lanfang He, De Lv, Jun Yang, and Zhu Yuan. The role of the dysregulated jnk signaling pathway in the pathogenesis of human diseases and its potential therapeutic strategies: a comprehensive review. Biomolecules, 14:243, Feb 2024. URL: https://doi.org/10.3390/biom14020243, doi:10.3390/biom14020243. This article has 44 citations and is from a peer-reviewed journal.
30. (zeke2016jnksignalingregulation pages 10-10): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
31. (zeke2016jnksignalingregulation pages 10-13): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
32. (zeke2016jnksignalingregulation pages 13-14): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
33. (zeke2016jnksignalingregulation pages 2-3): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
34. (zeke2016jnksignalingregulation pages 3-5): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
35. (zeke2016jnksignalingregulation pages 31-31): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
36. (zeke2016jnksignalingregulation pages 31-32): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
37. (zeke2016jnksignalingregulation pages 42-43): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
38. (zeke2016jnksignalingregulation pages 43-43): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
39. (zeke2016jnksignalingregulation pages 6-7): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
40. (zeke2016jnksignalingregulation pages 8-10): András Zeke, Mariya Misheva, Attila Reményi, and Marie A. Bogoyevitch. Jnk signaling: regulation and functions based on complex protein-protein partnerships. Microbiology and Molecular Biology Reviews, 80:793-835, Sep 2016. URL: https://doi.org/10.1128/mmbr.00043-14, doi:10.1128/mmbr.00043-14. This article has 562 citations and is from a domain leading peer-reviewed journal.
41. (ansideri2018structuraloptimizationof pages 23-23): Francesco Ansideri, Joana T. Macedo, Michael Eitel, Ahmed El-Gokha, Dhafer S. Zinad, Camilla Scarpellini, Mark Kudolo, Dieter Schollmeyer, Frank M. Boeckler, Bärbel S. Blaum, Stefan A. Laufer, and Pierre Koch. Structural optimization of a pyridinylimidazole scaffold: shifting the selectivity from p38α mitogen-activated protein kinase to c-jun n-terminal kinase 3. ACS Omega, 3:7809-7831, Jul 2018. URL: https://doi.org/10.1021/acsomega.8b00668, doi:10.1021/acsomega.8b00668. This article has 32 citations and is from a peer-reviewed journal.
42. (honzejkova2024structuralstudiesof pages 106-108): K Honzejková. Structural studies of selected protein complexes involved in signal transduction. Unknown journal, 2024.
43. (honzejkova2024structuralstudiesof pages 15-20): K Honzejková. Structural studies of selected protein complexes involved in signal transduction. Unknown journal, 2024.