1. Phylogeny – MAPK14, commonly known as p38α, is a member of the p38 MAP kinase subfamily within the larger serine/threonine kinase superfamily. It is evolutionarily conserved across eukaryotes and is present in mammals as well as lower organisms, highlighting its fundamental role in cellular stress and inflammatory signaling. In the human kinome classification, p38α is grouped with the four p38 isoforms (α, β, γ, δ) that share considerable homology in their catalytic domains yet differ in regulatory and tissue-specific expression properties. Its evolutionary origin can be traced back to ancient eukaryotic protein kinases, and phylogenetic analyses—such as those described in the seminal works by Manning and colleagues—show that p38α and its paralogues diverged early to acquire specialized functions in stress response and cytokine signaling (ganguly2023revisitingp38mitogenactivated pages 3-5, juyoux2023architectureofthe pages 1-3).
2. Reaction Catalyzed – MAPK14 functions as a serine/threonine kinase that catalyzes the transfer of a gamma-phosphate from ATP to the hydroxyl group of serine or threonine residues on target proteins. The reaction can be summarized chemically as: ATP + protein–(L-serine or L-threonine) → ADP + protein–(L-serine/threonine)-phosphate + H⁺ (benn2020clinicallyprecedentedprotein pages 16-17).
3. Cofactor Requirements – The catalytic activity of MAPK14 is dependent on the presence of divalent metal ions, specifically Mg²⁺, which facilitates proper ATP binding and catalysis. In some contexts, Mn²⁺ may also support its enzymatic function, consistent with requirements for most protein kinases (phan2023p38kinasein pages 4-5).
4. Substrate Specificity – MAPK14 exhibits broad substrate specificity, phosphorylating an estimated 200–300 substrates which include transcription factors (such as ATF2, CREB, and p53), downstream kinases (for example, MAPKAPK2/MK2 and MAPKAPK3/MK3), and other proteins involved in cellular processes ranging from gene expression to receptor internalization. Substrate recognition is achieved not only by the catalytic cleft but also via docking interactions with linear motifs—short sequence elements enriched in basic residues—that bind to specific docking grooves on MAPK14. For serine/threonine kinases, recent substrate specificity atlases report consensus motifs that may include preferences for nearby arginine or hydrophobic residues in substrates, a finding that is consistent with the broad specificity documented for kinases in this family (robles2023linearmotifspecificity pages 1-2, singh2023molecularinsightsof pages 12-13, federspiel2023p38mitogenactivatedprotein pages 16-17, federspiel2023p38mitogenactivatedprotein pages 17-18). For tyrosine kinases, similar comprehensive analyses have been reported; however, as MAPK14 is a serine/threonine kinase its specificity is best contextualized with the atlas provided by Johnson et al. (2023) and does not follow the consensus motifs typical of the tyrosine kinome (robles2023linearmotifspecificity pages 9-10).
5. Structure – The three-dimensional structure of MAPK14 comprises a typical bilobed kinase fold with an N-terminal lobe mainly responsible for binding ATP and a larger C-terminal lobe that facilitates substrate interactions and regulatory functions. Key structural features include the activation loop harboring a conserved Thr–Gly–Tyr (TGY) motif; dual phosphorylation of the threonine and tyrosine residues in this loop induces conformational changes essential for catalytic activity and substrate binding (juyoux2023architectureofthe pages 15-17). Additional structural elements include the conserved C-helix, which is critical for maintaining the active conformation, and a hydrophobic spine that aligns upon activation. Detailed cryo-electron microscopy and molecular dynamics analyses of the MKK6–p38α complex have highlighted specific interaction interfaces such as the docking interaction between the αG helix of MKK6 and a hydrophobic pocket in the C-lobe of MAPK14; these interactions not only facilitate p38α activation but also contribute to substrate specificity (juyoux2023architectureofthe pages 1-3, juyoux2023architectureofthe pages 17-24). The core catalytic domain is flanked by regions that, although less structured, are important for subcellular localization and the interaction with regulatory proteins.
6. Regulation – MAPK14 is tightly regulated by phosphorylation events, which are critical for its activation and subsequent signal transduction. Activation is typically achieved by upstream MAP kinase kinases, such as MKK3 and MKK6, which phosphorylate the TGY motif in the activation loop, leading to a conformational change that fosters substrate binding and catalytic efficiency (federspiel2023p38mitogenactivatedprotein pages 16-17, ganguly2023revisitingp38mitogenactivated pages 9-11). In addition, MAPK14 can undergo autophosphorylation, sometimes in a TAB1-dependent manner, which further modulates its activity. It is also subject to regulation by protein phosphatases, including dual-specificity phosphatases (DUSPs) such as DUSP1, which can dephosphorylate and inactivate MAPK14 (patysheva2023dualspecificityphosphatasesin pages 7-9). Other regulatory inputs include interactions with scaffold proteins that bring the kinase into proximity with its substrates and upstream activators, and modifications by casein kinase II that enhance autophosphorylation and capability to phosphorylate substrates like p53 (federspiel2023p38mitogenactivatedprotein pages 17-18, ganguly2023revisitingp38mitogenactivated pages 11-12).
7. Function – MAPK14 serves as a central mediator in the cellular response to a variety of stress signals, including pro-inflammatory cytokines, oxidative stress, and physical stressors. Its role encompasses the activation of downstream kinases such as MSK1, MSK2, MK2, and MK3, which in turn propagate the signal by modulating transcription factors and other cellular targets. In the nucleus, MAPK14 phosphorylates transcription factors like ATF1, ATF2, ELK1, and p53, thereby controlling gene expression that governs cell cycle regulation, apoptosis, and differentiation (federspiel2023p38mitogenactivatedprotein pages 16-17, phan2023p38kinasein pages 7-8). MAPK14 is also involved in the regulation of protein turnover in the cytoplasm, contributing to processes such as receptor endocytosis, autophagy, and the ubiquitin–proteasome system; for example, it phosphorylates the metalloprotease ADAM17, which is essential for ectodomain shedding of membrane proteins like TGF-α (federspiel2023p38mitogenactivatedprotein pages 16-17, phan2023p38kinasein pages 8-8). Further, by phosphorylating components such as CDC25B and CDC25C, MAPK14 contributes to cell cycle checkpoints following DNA damage, and through its effects on factors like TIAR and components of the mRNA translational machinery, it influences post-transcriptional gene regulation (ganguly2023revisitingp38mitogenactivated pages 9-11, phan2023p38kinasein pages 6-7). The kinase is ubiquitously expressed with particularly high levels in tissues involved in inflammatory and immune responses, contributing to its recognized role in mediating cellular responses in diseases such as inflammatory arthritis and certain cancers.
8. Other Comments – Several small molecule inhibitors targeting MAPK14 have been developed and are under investigation for clinical applications in diseases characterized by inflammatory and pro-apoptotic dysregulation. Inhibition of MAPK14 has been explored for therapeutic benefit in conditions ranging from rheumatoid arthritis to certain types of cancer and neurodegenerative diseases; clinical candidates such as ralimetinib have entered trials to evaluate their efficacy and safety profiles (federspiel2023p38mitogenactivatedprotein pages 16-17, ganguly2023revisitingp38mitogenactivated pages 5-6). Furthermore, the modulation of MAPK14 activity by regulatory phosphatases such as DUSP1 underscores the therapeutic potential of targeting the signaling network at multiple nodes (patysheva2023dualspecificityphosphatasesin pages 4-6). Notable disease associations of MAPK14 include its involvement in the modulation of inflammatory cytokine production, regulation of cell survival pathways, effects on endothelial cell functions, and the regulation of epithelial-to-mesenchymal transition (EMT), all of which contribute to its role in cancer progression, immune regulation, and tissue repair (federspiel2023p38mitogenactivatedprotein pages 16-17, phan2023p38kinasein pages 7-8, zavvarian2024translationalrelevanceof pages 19-21). Caution is warranted in therapeutic approaches targeting MAPK14 due to its broad substrate specificity and multiple roles in essential cellular processes, which can lead to off-target effects if not precisely controlled.
9. References
10. federspiel2023p38mitogenactivatedprotein pages 16-17
11. federspiel2023p38mitogenactivatedprotein pages 17-18
12. ganguly2023revisitingp38mitogenactivated pages 3-5
13. ganguly2023revisitingp38mitogenactivated pages 9-11
14. ganguly2023revisitingp38mitogenactivated pages 11-12
15. juyoux2023architectureofthe pages 1-3
16. juyoux2023architectureofthe pages 15-17
17. kadosh2023differentialmodulationof pages 1-2
18. kadosh2023differentialmodulationof pages 16-18
19. phan2023p38kinasein pages 4-5
20. phan2023p38kinasein pages 6-7
21. phan2023p38kinasein pages 7-8
22. phan2023p38kinasein pages 8-8
23. robles2023linearmotifspecificity pages 1-2
24. robles2023linearmotifspecificity pages 9-10
25. singh2023molecularinsightsof pages 12-13
26. zavvarian2024translationalrelevanceof pages 19-21
27. benn2020clinicallyprecedentedprotein pages 16-17
28. cao2023aperoxidedoxinp38mapk pages 1-5
29. ganguly2023revisitingp38mitogenactivated pages 1-3
30. ganguly2023revisitingp38mitogenactivated pages 13-13
31. ganguly2023revisitingp38mitogenactivated pages 5-6
32. phan2023p38kinasein pages 1-2
33. phan2023p38kinasein pages 5-6
34. juyoux2023architectureofthe pages 17-24
35. juyoux2023architectureofthe pages 3-4
36. kadosh2023differentialmodulationof pages 13-15
37. patysheva2023dualspecificityphosphatasesin pages 4-6
38. patysheva2023dualspecificityphosphatasesin pages 7-9
39. li2024stressactivatedproteinkinases pages 4-6
40. moustardas2023mapkpathwaysin pages 1-3
41. chowdhury2023cmgckinasesin pages 6-8
42. higgins2023sarscov2hijacksp38βmapk11 pages 2-6

Note: The above references have been directly extracted from the available context to provide a comprehensive nomenclature and functional profile of MAPK14.

References

1. (federspiel2023p38mitogenactivatedprotein pages 16-17): Julia Federspiel, Maria do Carmo Greier, Andrea Ladányi, and Jozsef Dudas. P38 mitogen-activated protein kinase inhibition of mesenchymal transdifferentiated tumor cells in head and neck squamous cell carcinoma. Biomedicines, 11:3301, Dec 2023. URL: https://doi.org/10.3390/biomedicines11123301, doi:10.3390/biomedicines11123301. This article has 1 citations and is from a peer-reviewed journal.
2. (federspiel2023p38mitogenactivatedprotein pages 17-18): Julia Federspiel, Maria do Carmo Greier, Andrea Ladányi, and Jozsef Dudas. P38 mitogen-activated protein kinase inhibition of mesenchymal transdifferentiated tumor cells in head and neck squamous cell carcinoma. Biomedicines, 11:3301, Dec 2023. URL: https://doi.org/10.3390/biomedicines11123301, doi:10.3390/biomedicines11123301. This article has 1 citations and is from a peer-reviewed journal.
3. (ganguly2023revisitingp38mitogenactivated pages 11-12): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
4. (ganguly2023revisitingp38mitogenactivated pages 3-5): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
5. (ganguly2023revisitingp38mitogenactivated pages 9-11): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
6. (juyoux2023architectureofthe pages 1-3): Pauline Juyoux, Ioannis Galdadas, Dorothea Gobbo, Jill von Velsen, Martin Pelosse, Mark Tully, Oscar Vadas, Francesco Luigi Gervasio, Erika Pellegrini, and Matthew W. Bowler. Architecture of the mkk6-p38α complex defines the basis of mapk specificity and activation. Science, 381:1217-1225, Sep 2023. URL: https://doi.org/10.1126/science.add7859, doi:10.1126/science.add7859. This article has 28 citations and is from a highest quality peer-reviewed journal.
7. (juyoux2023architectureofthe pages 15-17): Pauline Juyoux, Ioannis Galdadas, Dorothea Gobbo, Jill von Velsen, Martin Pelosse, Mark Tully, Oscar Vadas, Francesco Luigi Gervasio, Erika Pellegrini, and Matthew W. Bowler. Architecture of the mkk6-p38α complex defines the basis of mapk specificity and activation. Science, 381:1217-1225, Sep 2023. URL: https://doi.org/10.1126/science.add7859, doi:10.1126/science.add7859. This article has 28 citations and is from a highest quality peer-reviewed journal.
8. (kadosh2023differentialmodulationof pages 1-2): Dganit Melamed Kadosh, Jonah Beenstock, David Engelberg, and Arie Admon. Differential modulation of the phosphoproteome by the map kinases isoforms p38α and p38β. International Journal of Molecular Sciences, 24:12442, Aug 2023. URL: https://doi.org/10.3390/ijms241512442, doi:10.3390/ijms241512442. This article has 3 citations and is from a peer-reviewed journal.
9. (kadosh2023differentialmodulationof pages 16-18): Dganit Melamed Kadosh, Jonah Beenstock, David Engelberg, and Arie Admon. Differential modulation of the phosphoproteome by the map kinases isoforms p38α and p38β. International Journal of Molecular Sciences, 24:12442, Aug 2023. URL: https://doi.org/10.3390/ijms241512442, doi:10.3390/ijms241512442. This article has 3 citations and is from a peer-reviewed journal.
10. (phan2023p38kinasein pages 4-5): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
11. (phan2023p38kinasein pages 6-7): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
12. (phan2023p38kinasein pages 7-8): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
13. (phan2023p38kinasein pages 8-8): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
14. (robles2023linearmotifspecificity pages 1-2): Jaylissa Torres Robles, Hua Jane Lou, Guangda Shi, Pauline Lining Pan, and Benjamin E. Turk. Linear motif specificity in signaling through p38α and erk2 mitogen–activated protein kinases. Proceedings of the National Academy of Sciences, Nov 2023. URL: https://doi.org/10.1073/pnas.2316599120, doi:10.1073/pnas.2316599120. This article has 4 citations.
15. (singh2023molecularinsightsof pages 12-13): Sunil Kumar Singh, Ruchi Roy, Sandeep Kumar, Piush Srivastava, Saket Jha, Basabi Rana, and Ajay Rana. Molecular insights of map4k4 signaling in inflammatory and malignant diseases. Cancers, 15:2272, Apr 2023. URL: https://doi.org/10.3390/cancers15082272, doi:10.3390/cancers15082272. This article has 18 citations and is from a peer-reviewed journal.
16. (zavvarian2024translationalrelevanceof pages 19-21): Mohammad-Masoud Zavvarian, Akshat D. Modi, Sarah Sadat, James Hong, and Michael G. Fehlings. Translational relevance of secondary intracellular signaling cascades following traumatic spinal cord injury. International Journal of Molecular Sciences, 25:5708, May 2024. URL: https://doi.org/10.3390/ijms25115708, doi:10.3390/ijms25115708. This article has 3 citations and is from a peer-reviewed journal.
17. (benn2020clinicallyprecedentedprotein pages 16-17): Caroline L. Benn and Lee A. Dawson. Clinically precedented protein kinases: rationale for their use in neurodegenerative disease. Frontiers in Aging Neuroscience, Sep 2020. URL: https://doi.org/10.3389/fnagi.2020.00242, doi:10.3389/fnagi.2020.00242. This article has 48 citations and is from a peer-reviewed journal.
18. (ganguly2023revisitingp38mitogenactivated pages 1-3): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
19. (ganguly2023revisitingp38mitogenactivated pages 13-13): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
20. (ganguly2023revisitingp38mitogenactivated pages 5-6): Payal Ganguly, Tom Macleod, Chi Wong, Mark Harland, and Dennis McGonagle. Revisiting p38 mitogen-activated protein kinases (mapk) in inflammatory arthritis: a narrative of the emergence of mapk-activated protein kinase inhibitors (mk2i). Pharmaceuticals, 16:1286, Sep 2023. URL: https://doi.org/10.3390/ph16091286, doi:10.3390/ph16091286. This article has 29 citations and is from a peer-reviewed journal.
21. (li2024stressactivatedproteinkinases pages 4-6): Lei Li, Guangzhi Zhang, Zhili Yang, and Xuewen Kang. Stress-activated protein kinases in intervertebral disc degeneration: unraveling the impact of jnk and p38 mapk. Biomolecules, 14:393, Mar 2024. URL: https://doi.org/10.3390/biom14040393, doi:10.3390/biom14040393. This article has 7 citations and is from a peer-reviewed journal.
22. (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.
23. (patysheva2023dualspecificityphosphatasesin pages 4-6): Marina R. Patysheva, Elizaveta A. Prostakishina, Arina A. Budnitskaya, Olga D. Bragina, and Julia G. Kzhyshkowska. Dual-specificity phosphatases in regulation of tumor-associated macrophage activity. International Journal of Molecular Sciences, 24:17542, Dec 2023. URL: https://doi.org/10.3390/ijms242417542, doi:10.3390/ijms242417542. This article has 2 citations and is from a peer-reviewed journal.
24. (phan2023p38kinasein pages 1-2): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
25. (phan2023p38kinasein pages 5-6): Thuy Phan, Xu Hannah Zhang, Steven Rosen, and Laleh G. Melstrom. P38 kinase in gastrointestinal cancers. Cancer Gene Therapy, 30:1181-1189, May 2023. URL: https://doi.org/10.1038/s41417-023-00622-1, doi:10.1038/s41417-023-00622-1. This article has 24 citations and is from a peer-reviewed journal.
26. (robles2023linearmotifspecificity pages 9-10): Jaylissa Torres Robles, Hua Jane Lou, Guangda Shi, Pauline Lining Pan, and Benjamin E. Turk. Linear motif specificity in signaling through p38α and erk2 mitogen–activated protein kinases. Proceedings of the National Academy of Sciences, Nov 2023. URL: https://doi.org/10.1073/pnas.2316599120, doi:10.1073/pnas.2316599120. This article has 4 citations.
27. (juyoux2023architectureofthe pages 17-24): Pauline Juyoux, Ioannis Galdadas, Dorothea Gobbo, Jill von Velsen, Martin Pelosse, Mark Tully, Oscar Vadas, Francesco Luigi Gervasio, Erika Pellegrini, and Matthew W. Bowler. Architecture of the mkk6-p38α complex defines the basis of mapk specificity and activation. Science, 381:1217-1225, Sep 2023. URL: https://doi.org/10.1126/science.add7859, doi:10.1126/science.add7859. This article has 28 citations and is from a highest quality peer-reviewed journal.
28. (juyoux2023architectureofthe pages 3-4): Pauline Juyoux, Ioannis Galdadas, Dorothea Gobbo, Jill von Velsen, Martin Pelosse, Mark Tully, Oscar Vadas, Francesco Luigi Gervasio, Erika Pellegrini, and Matthew W. Bowler. Architecture of the mkk6-p38α complex defines the basis of mapk specificity and activation. Science, 381:1217-1225, Sep 2023. URL: https://doi.org/10.1126/science.add7859, doi:10.1126/science.add7859. This article has 28 citations and is from a highest quality peer-reviewed journal.
29. (kadosh2023differentialmodulationof pages 13-15): Dganit Melamed Kadosh, Jonah Beenstock, David Engelberg, and Arie Admon. Differential modulation of the phosphoproteome by the map kinases isoforms p38α and p38β. International Journal of Molecular Sciences, 24:12442, Aug 2023. URL: https://doi.org/10.3390/ijms241512442, doi:10.3390/ijms241512442. This article has 3 citations and is from a peer-reviewed journal.
30. (patysheva2023dualspecificityphosphatasesin pages 7-9): Marina R. Patysheva, Elizaveta A. Prostakishina, Arina A. Budnitskaya, Olga D. Bragina, and Julia G. Kzhyshkowska. Dual-specificity phosphatases in regulation of tumor-associated macrophage activity. International Journal of Molecular Sciences, 24:17542, Dec 2023. URL: https://doi.org/10.3390/ijms242417542, doi:10.3390/ijms242417542. This article has 2 citations and is from a peer-reviewed journal.
31. (chowdhury2023cmgckinasesin pages 6-8): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
32. (higgins2023sarscov2hijacksp38βmapk11 pages 2-6): Christina A. Higgins, Benjamin E. Nilsson-Payant, Boris Bonaventure, Andrew P. Kurland, Chengjin Ye, Tomer M. Yaron, Jared L. Johnson, Prithy Adhikary, Ilona Golynker, Maryline Panis, Oded Danziger, Brad R. Rosenberg, Lewis C. Cantley, Luis Martínez-Sobrido, Benjamin tenOever, and Jeffrey R. Johnson. Sars-cov-2 hijacks p38β/mapk11 to promote virus replication. mBio, Jun 2023. URL: https://doi.org/10.1128/mbio.01007-23, doi:10.1128/mbio.01007-23. This article has 9 citations and is from a domain leading peer-reviewed journal.