1. Phylogeny  
   Serine/threonine‐protein kinase Greatwall, commonly designated as MASTL (and also known as GWL or THC2), is a member of the AGC kinase family that is evolutionarily conserved across eukaryotes, with well‐characterized orthologs in Drosophila, Xenopus, and mammals, including humans (castro2018greatwallkinaseat pages 1-2). The human ortholog MASTL has been traced back to the common ancestor of eukaryotes and is part of an evolutionarily preserved regulatory network controlling mitotic progression, which places it in a core set of cell cycle regulators alongside other AGC kinases (lorca2013thegreatwallkinase pages 1-3). In Drosophila, the gene was originally identified as greatwall and mutations in this gene result in pronounced mitotic defects, a phenotype that has been recapitulated in vertebrate systems, thus emphasizing the deep evolutionary conservation of its function (williams2004greatwallkinasea pages 1-2). Phylogenetic analyses have revealed that while MASTL shares common features with other AGC family members, it possesses unique structural attributes – such as an unusually large insertion interrupting the kinase domain – that set it apart from its kin (hermida2020molecularbasisof pages 1-2).
2. Reaction Catalyzed  
   MASTL catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine/threonine residues on its substrate proteins, which in the case of its best‐characterized substrates occurs on ARPP19 and ENSA (ammarah2018identificationofnew pages 1-2). In its canonical reaction, ATP and a protein substrate combine to yield ADP and a phosphorylated protein product with a phosphate ester linkage on a serine or threonine residue, concomitantly releasing a proton; this reaction is a hallmark of serine/threonine kinases (hara2012greatwallkinaseand pages 1-2). The phosphorylation of ARPP19 at Ser-62 and ENSA at Ser-67 converts these small proteins into potent inhibitors of the PP2A-B55 phosphatase complex, thereby indirectly modulating the overall phosphorylation state of critical mitotic substrates (castro2018greatwallkinaseat pages 1-2).
3. Cofactor Requirements  
   As is typical for serine/threonine kinases, the catalytic activity of MASTL relies on the presence of divalent cations, with Mg²⁺ serving as the required cofactor to coordinate ATP binding and phosphotransfer (castro2018greatwallkinaseat pages 1-2). The requirement for Mg²⁺ is essential for stabilizing the ATP molecule in the active site, allowing the proper orientation of ATP for efficient phosphoryl transfer (diril2016lossofthe pages 1-2).
4. Substrate Specificity  
   The substrate specificity of MASTL is defined primarily by its ability to phosphorylate the small, intrinsically disordered proteins ARPP19 and ENSA, which play a critical role as downstream effectors in the MASTL signaling cascade (ammarah2018identificationofnew pages 1-2). Phosphorylation occurs at highly conserved serine residues—Ser-62 in ARPP19 and Ser-67 in ENSA—and this modification is necessary to convert these substrates into inhibitors of the PP2A-B55 phosphatase (castro2018greatwallkinaseat pages 1-2, wong2016mastl(greatwall)regulatesdna pages 1-2). Although the precise consensus phosphorylation motif for MASTL beyond these substrates is not fully defined, screening studies have suggested that a basic residue environment may be favored near the phosphorylation sites, a feature common among many AGC kinases; however, detailed motif analysis has not yet conclusively established a broader consensus sequence (marzec2022silackinasescreen pages 2-3).
5. Structure  
   MASTL is characterized as an atypical member of the AGC kinase family; it contains a central catalytic domain that exhibits the classical bilobal structure found in protein kinases, composed of a smaller N-lobe and a larger C-lobe (neil2013investigationofthe pages 121-124). A unique and distinguishing feature of this kinase is the presence of an approximately 500-amino acid insertion within its catalytic domain, often referred to as the non-conserved middle region (NCMR) or large T-loop insertion, which is not observed in typical AGC kinases and is thought to modulate substrate interactions and regulatory mechanisms (ammarah2018identificationofnew pages 1-2, castro2018greatwallkinaseat pages 2-3). Despite lacking a canonical hydrophobic motif that is characteristic of other AGC family members, MASTL retains a hydrophobic pocket that is critical for its regulation and activation (castro2018greatwallkinaseat pages 1-2, lorca2013thegreatwallkinase pages 4-5). The conserved kinase domain also contains key structural features such as a glycine-rich loop that facilitates ATP binding, the DFG motif essential for coordinating divalent cations, and a critical salt bridge formed between a conserved lysine and a glutamic acid within the αC helix, all of which are necessary for catalytic activity (neil2013investigationofthe pages 121-124, hermida2020molecularbasisof pages 17-18). These structural elements work in concert to form an active conformation that, upon phosphorylation, enables MASTL to efficiently phosphorylate its substrates (castro2018greatwallkinaseat pages 3-4).
6. Regulation  
   Regulation of MASTL is predominantly achieved through phosphorylation-dependent mechanisms that control its catalytic activity and subcellular distribution during the cell cycle. During mitosis, MASTL is activated by phosphorylation events mediated by cyclin B1-CDK1 complexes, which prime the kinase for full activation and may be followed by cis-autophosphorylation steps (hara2012greatwallkinaseand pages 1-2, diril2016lossofthe pages 20-22). Once activated, MASTL phosphorylates ARPP19 and ENSA, thus converting them into inhibitors of PP2A-B55; this inhibition is pivotal for maintaining the phosphorylation status of cyclin B1-CDK1 substrates and ensuring robust mitotic progression (ammarah2018identificationofnew pages 1-2, castro2018greatwallkinaseat pages 6-6). In addition, the inactivation of MASTL at mitotic exit is regulated by dephosphorylation reactions mediated by phosphatases such as PP1 and PP2A, thereby allowing the removal of inhibitory phosphates from cyclin B1-CDK1 substrates and facilitating the transition into interphase (lorca2013thegreatwallkinase pages 6-7, diril2016lossofthe pages 2-4). Under conditions of DNA damage, MASTL activity is suppressed to allow proper checkpoint activation and DNA repair, and its subsequent reactivation is necessary for timely recovery from the damage-induced G2 arrest (wong2016mastl(greatwall)regulatesdna pages 1-2, conway2020kinaseindependentfunctionsof pages 1-5). These multilayered regulatory processes ensure that MASTL activity is tightly coupled to the cell cycle, thereby maintaining mitotic fidelity.
7. Function  
   MASTL functions as a master regulator of mitosis by maintaining a high level of cyclin B1-CDK1 activity during the M phase of the cell cycle. It accomplishes this by phosphorylating ARPP19 and ENSA, which, upon phosphorylation at serine-62 and serine-67 respectively, inhibit the phosphatase PP2A-B55; this inhibition prevents premature dephosphorylation of CDK1 substrates, a process that is critical for proper mitotic entry and progression (ammarah2018identificationofnew pages 1-2, castro2018greatwallkinaseat pages 1-2). Moreover, MASTL plays a role in ensuring proper chromosome condensation and spindle assembly checkpoint integrity; its loss or dysfunction has been associated with aberrant chromosome segregation, delayed mitotic progression, and compromised spindle checkpoint function, as demonstrated by studies in mouse embryonic fibroblasts and Drosophila models (diril2016lossofthe pages 9-12, williams2004greatwallkinasea pages 2-3). In the context of DNA damage, MASTL is involved in checkpoint recovery by enabling the reactivation of cyclin B1-CDK1 once repair processes are complete, thereby facilitating a timely entry into mitosis (wong2016mastl(greatwall)regulatesdna pages 1-2, neil2013investigationofthe pages 59-62). There is also evidence suggesting a potential role for MASTL in megakaryocyte differentiation, although the primary body of literature has focused on its function in regulating mitosis through the MASTL/ENSA/PP2A axis (ammarah2018identificationofnew pages 1-2). Lastly, altered expression or hyperactivation of MASTL has been associated with various cancers, including breast, colon, and gastric malignancies, where elevated MASTL levels correlate with increased tumor cell proliferation, metastasis, and chemoresistance (misra2023targetedinhibitionof pages 13-15, fatima2020mastlanovel pages 1-2).
8. Other Comments  
   Several inhibitors targeting MASTL have been identified, including natural compounds such as flavonoid derivatives that demonstrate nanomolar inhibition of its kinase activity; these inhibitors have shown efficacy in inducing cell cycle arrest and apoptosis in cancer cell models (misra2023targetedinhibitionof pages 13-15). In addition, large-scale kinome screening approaches, such as SILAC-based kinase screens, have been employed to identify novel substrates and binding partners of MASTL, expanding the known regulatory network and providing potential avenues for therapeutic intervention (marzec2022silackinasescreen pages 2-3). Overexpression of MASTL has emerged as a prognostic marker in several human cancers, with elevated levels correlating with poor clinical outcomes and resistance to conventional chemotherapies; this association underlines the therapeutic potential of MASTL inhibition in oncology (fatima2020mastlanovel pages 1-2, misra2023targetedinhibitionof pages 13-15). Furthermore, ongoing studies continue to refine our understanding of the molecular determinants of MASTL activation and regulation, including the functional significance of its large insertion domain and the potential for crosstalk with other cell cycle kinases such as PLK1 and Aurora A (lorca2013thegreatwallkinase pages 7-8, conway2020kinaseindependentfunctionsof pages 1-5).
9. References
10. ammarah2018identificationofnew pages 1-2
11. ammarah2018identificationofnew pages 10-11
12. bisteau2020thegreatwallkinase pages 1-2
13. bisteau2020thegreatwallkinase pages 9-11
14. castro2018greatwallkinaseat pages 1-2
15. castro2018greatwallkinaseat pages 2-3
16. castro2018greatwallkinaseat pages 3-4
17. castro2018greatwallkinaseat pages 6-6
18. castro2018greatwallkinaseat pages 6-7
19. conway2020kinaseindependentfunctionsof pages 1-5
20. diril2016lossofthe pages 1-2
21. diril2016lossofthe pages 12-13
22. diril2016lossofthe pages 2-4
23. diril2016lossofthe pages 20-22
24. erguven2020complementationcloningidentifies pages 1-4
25. gouttia2022themastlensapp2ab55axis pages 9-9
26. hara2012greatwallkinaseand pages 1-2
27. hermida2020molecularbasisof pages 1-2
28. hermida2020molecularbasisof pages 11-12
29. hermida2020molecularbasisof pages 18-18
30. lorca2013thegreatwallkinase pages 1-3
31. lorca2013thegreatwallkinase pages 3-4
32. lorca2013thegreatwallkinase pages 4-5
33. lorca2013thegreatwallkinase pages 5-6
34. lorca2013thegreatwallkinase pages 6-7
35. lorca2013thegreatwallkinase pages 7-8
36. neil2013investigationofthe pages 111-114
37. neil2013investigationofthe pages 115-118
38. neil2013investigationofthe pages 118-121
39. neil2013investigationofthe pages 124-127
40. neil2013investigationofthe pages 43-46
41. neil2013investigationofthe pages 46-50
42. neil2013investigationofthe pages 89-93
43. vigneron2016themastergreatwall pages 5-6
44. vigneron2016themastergreatwall pages 8-9
45. williams2004greatwallkinasea pages 3-5
46. wong2016mastl(greatwall)regulatesdna pages 1-2
47. misra2023targetedinhibitionof pages 13-15
48. marzec2022silackinasescreen pages 2-2
49. marzec2022silackinasescreen pages 2-3
50. fatima2020mastlanovel pages 1-2

References

1. (ammarah2018identificationofnew pages 1-2): Ummi Ammarah, Amit Kumar, Rajesh Pal, Naresh C. Bal, and Gauri Misra. Identification of new inhibitors against human great wall kinase using in silico approaches. Scientific Reports, Mar 2018. URL: https://doi.org/10.1038/s41598-018-23246-0, doi:10.1038/s41598-018-23246-0. This article has 33 citations and is from a poor quality or predatory journal.
2. (ammarah2018identificationofnew pages 10-11): Ummi Ammarah, Amit Kumar, Rajesh Pal, Naresh C. Bal, and Gauri Misra. Identification of new inhibitors against human great wall kinase using in silico approaches. Scientific Reports, Mar 2018. URL: https://doi.org/10.1038/s41598-018-23246-0, doi:10.1038/s41598-018-23246-0. This article has 33 citations and is from a poor quality or predatory journal.
3. (bisteau2020thegreatwallkinase pages 1-2): Xavier Bisteau, Joann Lee, Vinayaka Srinivas, Joanna H. S. Lee, Joanna Niska-Blakie, Gifford Tan, Shannon Y. X. Yap, Kevin W. Hom, Cheng Kit Wong, Jeongjun Chae, Loo Chien Wang, Jinho Kim, Giulia Rancati, Radoslaw M. Sobota, Chris S. H. Tan, and Philipp Kaldis. The greatwall kinase safeguards the genome integrity by affecting the kinome activity in mitosis. Oncogene, 39:6816-6840, Sep 2020. URL: https://doi.org/10.1038/s41388-020-01470-1, doi:10.1038/s41388-020-01470-1. This article has 17 citations and is from a domain leading peer-reviewed journal.
4. (bisteau2020thegreatwallkinase pages 9-11): Xavier Bisteau, Joann Lee, Vinayaka Srinivas, Joanna H. S. Lee, Joanna Niska-Blakie, Gifford Tan, Shannon Y. X. Yap, Kevin W. Hom, Cheng Kit Wong, Jeongjun Chae, Loo Chien Wang, Jinho Kim, Giulia Rancati, Radoslaw M. Sobota, Chris S. H. Tan, and Philipp Kaldis. The greatwall kinase safeguards the genome integrity by affecting the kinome activity in mitosis. Oncogene, 39:6816-6840, Sep 2020. URL: https://doi.org/10.1038/s41388-020-01470-1, doi:10.1038/s41388-020-01470-1. This article has 17 citations and is from a domain leading peer-reviewed journal.
5. (castro2018greatwallkinaseat pages 1-2): A. Castro and T. Lorca. Greatwall kinase at a glance. Journal of Cell Science, Oct 2018. URL: https://doi.org/10.1242/jcs.222364, doi:10.1242/jcs.222364. This article has 63 citations and is from a domain leading peer-reviewed journal.
6. (castro2018greatwallkinaseat pages 2-3): A. Castro and T. Lorca. Greatwall kinase at a glance. Journal of Cell Science, Oct 2018. URL: https://doi.org/10.1242/jcs.222364, doi:10.1242/jcs.222364. This article has 63 citations and is from a domain leading peer-reviewed journal.
7. (castro2018greatwallkinaseat pages 3-4): A. Castro and T. Lorca. Greatwall kinase at a glance. Journal of Cell Science, Oct 2018. URL: https://doi.org/10.1242/jcs.222364, doi:10.1242/jcs.222364. This article has 63 citations and is from a domain leading peer-reviewed journal.
8. (castro2018greatwallkinaseat pages 6-6): A. Castro and T. Lorca. Greatwall kinase at a glance. Journal of Cell Science, Oct 2018. URL: https://doi.org/10.1242/jcs.222364, doi:10.1242/jcs.222364. This article has 63 citations and is from a domain leading peer-reviewed journal.
9. (castro2018greatwallkinaseat pages 6-7): A. Castro and T. Lorca. Greatwall kinase at a glance. Journal of Cell Science, Oct 2018. URL: https://doi.org/10.1242/jcs.222364, doi:10.1242/jcs.222364. This article has 63 citations and is from a domain leading peer-reviewed journal.
10. (conway2020kinaseindependentfunctionsof pages 1-5): J. Conway, Elisa Närvä, M. E. Taskinen, and J. Ivaska. Kinase-independent functions of mastl in cancer: a new perspective on mastl targeting. Cells, Jul 2020. URL: https://doi.org/10.3390/cells9071624, doi:10.3390/cells9071624. This article has 5 citations and is from a peer-reviewed journal.
11. (diril2016lossofthe pages 1-2): M. K. Diril, Xavier Bisteau, Mayumi Kitagawa, Matias J. Caldez, Matias J. Caldez, Sheena Wee, J. Gunaratne, J. Gunaratne, Sang Hyun Lee, P. Kaldis, and P. Kaldis. Loss of the greatwall kinase weakens the spindle assembly checkpoint. PLoS Genetics, Sep 2016. URL: https://doi.org/10.1371/journal.pgen.1006310, doi:10.1371/journal.pgen.1006310. This article has 47 citations and is from a domain leading peer-reviewed journal.
12. (diril2016lossofthe pages 12-13): M. K. Diril, Xavier Bisteau, Mayumi Kitagawa, Matias J. Caldez, Matias J. Caldez, Sheena Wee, J. Gunaratne, J. Gunaratne, Sang Hyun Lee, P. Kaldis, and P. Kaldis. Loss of the greatwall kinase weakens the spindle assembly checkpoint. PLoS Genetics, Sep 2016. URL: https://doi.org/10.1371/journal.pgen.1006310, doi:10.1371/journal.pgen.1006310. This article has 47 citations and is from a domain leading peer-reviewed journal.
13. (diril2016lossofthe pages 2-4): M. K. Diril, Xavier Bisteau, Mayumi Kitagawa, Matias J. Caldez, Matias J. Caldez, Sheena Wee, J. Gunaratne, J. Gunaratne, Sang Hyun Lee, P. Kaldis, and P. Kaldis. Loss of the greatwall kinase weakens the spindle assembly checkpoint. PLoS Genetics, Sep 2016. URL: https://doi.org/10.1371/journal.pgen.1006310, doi:10.1371/journal.pgen.1006310. This article has 47 citations and is from a domain leading peer-reviewed journal.
14. (diril2016lossofthe pages 20-22): M. K. Diril, Xavier Bisteau, Mayumi Kitagawa, Matias J. Caldez, Matias J. Caldez, Sheena Wee, J. Gunaratne, J. Gunaratne, Sang Hyun Lee, P. Kaldis, and P. Kaldis. Loss of the greatwall kinase weakens the spindle assembly checkpoint. PLoS Genetics, Sep 2016. URL: https://doi.org/10.1371/journal.pgen.1006310, doi:10.1371/journal.pgen.1006310. This article has 47 citations and is from a domain leading peer-reviewed journal.
15. (erguven2020complementationcloningidentifies pages 1-4): Mehmet Erguven, Ezgi Karaca, and M. Kasim Diril. Complementation cloning identifies the essentials of mammalian mastl kinase activation. BioRxiv, Jul 2020. URL: https://doi.org/10.1101/2020.06.30.179580, doi:10.1101/2020.06.30.179580. This article has 1 citations.
16. (gouttia2022themastlensapp2ab55axis pages 9-9): Odjo G. Gouttia, Jing Zhao, Yanqiu Li, Mackenzie J. Zwiener, Ling Wang, Gregory G. Oakley, and Aimin Peng. The mastl-ensa-pp2a/b55 axis modulates cisplatin resistance in oral squamous cell carcinoma. Frontiers in Cell and Developmental Biology, Sep 2022. URL: https://doi.org/10.3389/fcell.2022.904719, doi:10.3389/fcell.2022.904719. This article has 8 citations and is from a peer-reviewed journal.
17. (hara2012greatwallkinaseand pages 1-2): Masatoshi Hara, Yusuke Abe, Toshiaki Tanaka, Takayoshi Yamamoto, Eiichi Okumura, and Takeo Kishimoto. Greatwall kinase and cyclin b-cdk1 are both critical constituents of m-phase-promoting factor. Nature Communications, Sep 2012. URL: https://doi.org/10.1038/ncomms2062, doi:10.1038/ncomms2062. This article has 118 citations and is from a highest quality peer-reviewed journal.
18. (hermida2020molecularbasisof pages 1-2): Dario Hermida, G. Mortuza, A. Pedersen, I. Pozdnyakova, T. T. Nguyen, M. Maroto, M. Williamson, T. Ebersole, Giuseppe Cazzamali, K. Rand, J. Olsen, Marcos Malumbres, and G. Montoya. Molecular basis of the mechanisms controlling mastl\*. Molecular & Cellular Proteomics, 19:326-343, Dec 2020. URL: https://doi.org/10.1074/mcp.ra119.001879, doi:10.1074/mcp.ra119.001879. This article has 12 citations.
19. (hermida2020molecularbasisof pages 11-12): Dario Hermida, G. Mortuza, A. Pedersen, I. Pozdnyakova, T. T. Nguyen, M. Maroto, M. Williamson, T. Ebersole, Giuseppe Cazzamali, K. Rand, J. Olsen, Marcos Malumbres, and G. Montoya. Molecular basis of the mechanisms controlling mastl\*. Molecular & Cellular Proteomics, 19:326-343, Dec 2020. URL: https://doi.org/10.1074/mcp.ra119.001879, doi:10.1074/mcp.ra119.001879. This article has 12 citations.
20. (hermida2020molecularbasisof pages 18-18): Dario Hermida, G. Mortuza, A. Pedersen, I. Pozdnyakova, T. T. Nguyen, M. Maroto, M. Williamson, T. Ebersole, Giuseppe Cazzamali, K. Rand, J. Olsen, Marcos Malumbres, and G. Montoya. Molecular basis of the mechanisms controlling mastl\*. Molecular & Cellular Proteomics, 19:326-343, Dec 2020. URL: https://doi.org/10.1074/mcp.ra119.001879, doi:10.1074/mcp.ra119.001879. This article has 12 citations.
21. (lorca2013thegreatwallkinase pages 1-3): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
22. (lorca2013thegreatwallkinase pages 3-4): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
23. (lorca2013thegreatwallkinase pages 4-5): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
24. (lorca2013thegreatwallkinase pages 5-6): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
25. (lorca2013thegreatwallkinase pages 6-7): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
26. (lorca2013thegreatwallkinase pages 7-8): T Lorca and A Castro. The greatwall kinase: a new pathway in the control of the cell cycle. Oncogene, 32:537-543, Apr 2013. URL: https://doi.org/10.1038/onc.2012.79, doi:10.1038/onc.2012.79. This article has 77 citations and is from a domain leading peer-reviewed journal.
27. (neil2013investigationofthe pages 111-114): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
28. (neil2013investigationofthe pages 115-118): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
29. (neil2013investigationofthe pages 118-121): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
30. (neil2013investigationofthe pages 124-127): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
31. (neil2013investigationofthe pages 43-46): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
32. (neil2013investigationofthe pages 46-50): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
33. (neil2013investigationofthe pages 89-93): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
34. (vigneron2016themastergreatwall pages 5-6): S. Vigneron, Perle Robert, Khaled Hached, Lena Sundermann, S. Charrasse, J. Labbé, A. Castro, and T. Lorca. The master greatwall kinase, a critical regulator of mitosis and meiosis. The International journal of developmental biology, 60 7-8-9:245-254, Oct 2016. URL: https://doi.org/10.1387/ijdb.160155tl, doi:10.1387/ijdb.160155tl. This article has 32 citations.
35. (vigneron2016themastergreatwall pages 8-9): S. Vigneron, Perle Robert, Khaled Hached, Lena Sundermann, S. Charrasse, J. Labbé, A. Castro, and T. Lorca. The master greatwall kinase, a critical regulator of mitosis and meiosis. The International journal of developmental biology, 60 7-8-9:245-254, Oct 2016. URL: https://doi.org/10.1387/ijdb.160155tl, doi:10.1387/ijdb.160155tl. This article has 32 citations.
36. (williams2004greatwallkinasea pages 3-5): B Williams. Greatwall kinase a nuclear protein required for proper chromosome condensation and mitotic progression in drosophila. Unknown journal, 2004.
37. (wong2016mastl(greatwall)regulatesdna pages 1-2): Po Yee Wong, H. Ma, Hyun-Jung Lee, and R. Poon. Mastl(greatwall) regulates dna damage responses by coordinating mitotic entry after checkpoint recovery and apc/c activation. Scientific Reports, Feb 2016. URL: https://doi.org/10.1038/srep22230, doi:10.1038/srep22230. This article has 45 citations and is from a poor quality or predatory journal.
38. (diril2016lossofthe pages 9-12): M. K. Diril, Xavier Bisteau, Mayumi Kitagawa, Matias J. Caldez, Matias J. Caldez, Sheena Wee, J. Gunaratne, J. Gunaratne, Sang Hyun Lee, P. Kaldis, and P. Kaldis. Loss of the greatwall kinase weakens the spindle assembly checkpoint. PLoS Genetics, Sep 2016. URL: https://doi.org/10.1371/journal.pgen.1006310, doi:10.1371/journal.pgen.1006310. This article has 47 citations and is from a domain leading peer-reviewed journal.
39. (fatima2020mastlanovel pages 1-2): Iram Fatima, Amar B. Singh, and Punita Dhawan. Mastl: a novel therapeutic target for cancer malignancy. Cancer Medicine, 9:6322-6329, Jul 2020. URL: https://doi.org/10.1002/cam4.3141, doi:10.1002/cam4.3141. This article has 28 citations and is from a peer-reviewed journal.
40. (hermida2020molecularbasisof pages 17-18): Dario Hermida, G. Mortuza, A. Pedersen, I. Pozdnyakova, T. T. Nguyen, M. Maroto, M. Williamson, T. Ebersole, Giuseppe Cazzamali, K. Rand, J. Olsen, Marcos Malumbres, and G. Montoya. Molecular basis of the mechanisms controlling mastl\*. Molecular & Cellular Proteomics, 19:326-343, Dec 2020. URL: https://doi.org/10.1074/mcp.ra119.001879, doi:10.1074/mcp.ra119.001879. This article has 12 citations.
41. (marzec2022silackinasescreen pages 2-2): K. Marzec, Samuel Rogers, R. McCloy, B. Parker, David E. James, D. Watkins, and Andrew Burgess. Silac kinase screen identifies potential mastl substrates. Scientific Reports, Jun 2022. URL: https://doi.org/10.1038/s41598-022-14933-0, doi:10.1038/s41598-022-14933-0. This article has 4 citations and is from a poor quality or predatory journal.
42. (marzec2022silackinasescreen pages 2-3): K. Marzec, Samuel Rogers, R. McCloy, B. Parker, David E. James, D. Watkins, and Andrew Burgess. Silac kinase screen identifies potential mastl substrates. Scientific Reports, Jun 2022. URL: https://doi.org/10.1038/s41598-022-14933-0, doi:10.1038/s41598-022-14933-0. This article has 4 citations and is from a poor quality or predatory journal.
43. (misra2023targetedinhibitionof pages 13-15): Gauri Misra, J. Rajawat, Rajesh Pal, Jeremy C. Smith, and Amit Kumar. Targeted inhibition of mastl kinase activity induces apoptosis in breast cancer. Life sciences, pages 122250, Nov 2023. URL: https://doi.org/10.1016/j.lfs.2023.122250, doi:10.1016/j.lfs.2023.122250. This article has 2 citations and is from a peer-reviewed journal.
44. (neil2013investigationofthe pages 121-124): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
45. (neil2013investigationofthe pages 59-62): C Neil. Investigation of the mechanisms of the g2/m phase transition in human cells–the role of greatwall kinase. Unknown journal, 2013.
46. (williams2004greatwallkinasea pages 1-2): B Williams. Greatwall kinase a nuclear protein required for proper chromosome condensation and mitotic progression in drosophila. Unknown journal, 2004.
47. (williams2004greatwallkinasea pages 2-3): B Williams. Greatwall kinase a nuclear protein required for proper chromosome condensation and mitotic progression in drosophila. Unknown journal, 2004.