1. Phylogeny  
   NLK (Q9UBE8) is an evolutionarily conserved serine/threonine kinase that belongs to an atypical branch of the mitogen‐activated protein kinase (MAPK) family. Orthologs of NLK have been identified in invertebrates—for example, Drosophila expresses the nemo gene and Caenorhabditis elegans expresses LIT‑1—as well as in vertebrates including Xenopus, zebrafish, and mammals, underscoring its conservation across eukaryotes (daams2020nemolikekinasein pages 1-3, ishitani2011homodimerizationofnemolike pages 1-2, qin2024canonicalandnoncanonical pages 12-13).
2. Reaction Catalyzed  
   NLK catalyzes the transfer of a phosphate group from ATP to protein substrates by phosphorylating serine or threonine residues—typically the residue immediately preceding a proline—in its target proteins. In its reaction, ATP and a protein substrate yield ADP and a phosphorylated protein, thereby modulating the activity and interaction of key transcription factors (canalis2014nemo‐likekinaseregulates pages 1-3).
3. Cofactor Requirements  
   The catalytic activity of NLK is dependent on divalent cations, with magnesium ions (Mg²⁺) serving as an essential cofactor that coordinates ATP binding and facilitates the phosphoryl transfer reaction (canalis2014nemo‐likekinaseregulates pages 1-3).
4. Substrate Specificity  
   NLK preferentially phosphorylates serine/threonine residues in a proline-directed context, meaning that it most efficiently recognizes motifs in which a serine or threonine is immediately followed by a proline residue. Its well‐characterized substrates include transcription factors such as TCF7L2/TCF4 and LEF1, whose phosphorylation by NLK promotes their dissociation from DNA and stimulates their ubiquitination and proteolysis; this activity plays a critical role in the negative regulation of canonical Wnt/β‑catenin signaling (canalis2014nemo‐likekinaseregulates pages 1-3, li2014nemolikekinase(nlk) pages 15-18, ishitani2003regulationoflymphoid pages 1-2). Additionally, NLK has been shown to target factors such as FOXO1 and components within inflammatory signaling pathways, thereby broadening its substrate repertoire (kim2010regulationoffoxo1 pages 2-4, li2019phosphorylationofmavsvisa pages 1-2).
5. Structure  
   NLK is composed of approximately 515 amino acids and features a centrally located kinase domain flanked by less-conserved N‑terminal and C‑terminal regions. The kinase domain, which spans roughly residues 127 to 415, retains the overall structural fold conserved among MAP kinases but lacks the canonical TXY motif found in typical MAPKs. Instead, NLK exhibits a unique activation loop motif that includes key phosphorylation sites—most notably a critical residue (for example, Thr286) that is essential for its autophosphorylation and full catalytic activation. In addition, the C‑terminal region is required for NLK homodimerization, a process that is necessary for its autophosphorylation, activation, and proper nuclear translocation (daams2020nemolikekinasein pages 4-7, ishitani2011homodimerizationofnemolike pages 3-4, li2014nemolikekinase(nlk) pages 15-18). Structural studies and in vitro models indicate that the NLK monomer contains the conserved kinase core with a well‐defined ATP-binding pocket, a catalytic loop, and an activation segment that undergoes conformational changes upon dimer formation.
6. Regulation  
   NLK activity is tightly controlled through a combination of post‑translational modifications and protein–protein interactions. A major regulatory mechanism is autophosphorylation, which occurs upon homodimerization of NLK; the phosphorylation of key residues within the activation loop (e.g., Thr286) is critical for its catalytic activity and for triggering its nuclear localization (ishitani2011homodimerizationofnemolike pages 2-3, pages 3-4, pages 4-5). In addition, upstream kinases such as TAK1 and p38 MAPK have been reported to influence NLK activity in diverse signaling contexts, particularly in inflammatory responses and developmental pathways (ohnishi2010nemolikekinasean pages 1-2, li2014nemolikekinase(nlk) pages 5-6). NLK also exerts regulatory roles by phosphorylating its downstream substrates, such as TCF/LEF transcription factors and the Notch intracellular domain, resulting in alterations of their DNA-binding capacity and stability; this modification leads to a decrease in canonical Wnt/β‑catenin signaling and Notch signaling, respectively (canalis2014nemo‐likekinaseregulates pages 1-3, ishitani2010nemolikekinasesuppresses pages 19-24). Thus, NLK functions both as a target of upstream kinase cascades and as a regulator of transcription factor activity via phosphorylation.
7. Function  
   NLK plays a central role in determining cell fate by regulating the activity of various transcription factors. It has been characterized as a positive effector in non‑canonical Wnt signaling—operating downstream of factors such as WNT5A, MAP3K7/TAK1, and HIPK2—while concurrently acting as a negative regulator of the canonical Wnt/β‑catenin pathway. In the canonical pathway, NLK phosphorylates TCF7L2/TCF4 and LEF1, which results in their dissociation from DNA, followed by ubiquitination and degradation; this inhibitory mechanism prevents transcriptional activation of Wnt target genes (canalis2014nemo‐likekinaseregulates pages 1-3, li2014nemolikekinase(nlk) pages 15-18). NLK also negatively regulates Notch signaling by binding to and phosphorylating NOTCH1, thereby interfering with the formation of the transcriptionally active Notch complex (ishitani2010nemolikekinasesuppresses pages 1-6, 19-24). Furthermore, NLK has been implicated in the regulation of FOXO1 activity (kim2010regulationoffoxo1 pages 2-4) as well as in the modulation of inflammatory signaling through disruption of the TAK1–IKK complex (li2014nemolikekinase(nlk) pages 5-6). Expression studies show that NLK is present in multiple tissues, with notable expression in neural tissues where it contributes to processes such as anterior neural formation and neurite outgrowth (satoh2007nemolikekinasemyocyteenhancer pages 2-4, ohnishi2010nemolikekinasean pages 2-3). In addition, NLK influences skeletal homeostasis by modulating osteoblastogenesis and has been implicated in pathological states such as colorectal cancer and Diamond Blackfan anemia through its effects on cell proliferation and differentiation (zanotti2012nemo‐likekinaseinhibits pages 8-8, wilkes2020diamondblackfananemia pages 1-2).
8. Other Comments  
   NLK has attracted interest as a potential therapeutic target owing to its involvement in multiple signaling pathways that are frequently dysregulated in disease. Inhibitors of NLK activity are being investigated in the context of endocrine-resistant breast cancer and inflammatory disorders, and certain p38 MAPK inhibitors have been shown to incidentally affect NLK activity (wang2021therapeutictargetingof pages 1-2). Furthermore, hyperactivation of NLK has been linked to Diamond Blackfan anemia, particularly in erythroid progenitors, where its aberrant activity contributes to defective erythropoiesis (wilkes2020diamondblackfananemia pages 6-6). NLK’s role as a regulator of the canonical Wnt/β‑catenin pathway also points to its potential involvement in tumor suppression, as evidenced by studies in colorectal carcinoma cell lines that demonstrate NLK-induced apoptosis via kinase-dependent mechanisms (yasuda2003nemolikekinaseinduces pages 6-7). Finally, its regulatory effects on osteoblast differentiation suggest that NLK may be relevant to diseases associated with skeletal dysregulation (zanotti2012nemo‐likekinaseinhibits pages 8-8). Collectively, these features establish NLK as a multifunctional kinase with significant implications in cell fate determination, developmental biology, and disease pathogenesis.
9. References
10. canalis2014nemo‐likekinaseregulates pages 1-3
11. daams2020nemolikekinasein pages 1-3
12. daams2020nemolikekinasein pages 4-7
13. daams2020nemolikekinasein pages 7-8
14. ishitani2003regulationoflymphoid pages 1-2
15. ishitani2010nemolikekinasesuppresses pages 1-6
16. ishitani2010nemolikekinasesuppresses pages 19-24
17. ishitani2011homodimerizationofnemolike pages 1-2
18. ishitani2011homodimerizationofnemolike pages 9-11
19. ishitani2011homodimerizationofnemolike pages 2-3
20. ishitani2011homodimerizationofnemolike pages 3-4
21. ishitani2011homodimerizationofnemolike pages 4-5
22. li2014nemolikekinase(nlk) pages 15-18
23. li2014nemolikekinase(nlk) pages 1-3
24. li2014nemolikekinase(nlk) pages 3-5
25. li2014nemolikekinase(nlk) pages 5-6
26. li2014nemolikekinase(nlk) pages 6-8
27. li2014nemolikekinase(nlk) pages 8-10
28. li2019phosphorylationofmavsvisa pages 1-2
29. li2019phosphorylationofmavsvisa pages 12-13
30. masoumi2017nlkmediatedphosphorylationof pages 10-10
31. moon2017phosphorylationbynlk pages 1-2
32. moon2017phosphorylationbynlk pages 6-7
33. ohnishi2010nemolikekinasean pages 1-2
34. ohnishi2010nemolikekinasean pages 8-9
35. wang2021therapeutictargetingof pages 1-2
36. wilkes2020diamondblackfananemia pages 1-2
37. wilkes2020diamondblackfananemia pages 6-6
38. satoh2007nemolikekinasemyocyteenhancer pages 2-4
39. qin2024canonicalandnoncanonical pages 12-13
40. zanotti2012nemo‐likekinaseinhibits pages 8-8
41. yasuda2003nemolikekinaseinduces pages 6-7

References

1. (canalis2014nemo‐likekinaseregulates pages 1-3): Ernesto Canalis, Lauren Kranz, and Stefano Zanotti. Nemo‐like kinase regulates postnatal skeletal homeostasis. Journal of Cellular Physiology, Nov 2014. URL: https://doi.org/10.1002/jcp.24625, doi:10.1002/jcp.24625. This article has 10 citations and is from a peer-reviewed journal.
2. (daams2020nemolikekinasein pages 1-3): Renée Daams and Ramin Massoumi. Nemo-like kinase in development and diseases: insights from mouse studies. International Journal of Molecular Sciences, 21:9203, Dec 2020. URL: https://doi.org/10.3390/ijms21239203, doi:10.3390/ijms21239203. This article has 18 citations and is from a peer-reviewed journal.
3. (daams2020nemolikekinasein pages 4-7): Renée Daams and Ramin Massoumi. Nemo-like kinase in development and diseases: insights from mouse studies. International Journal of Molecular Sciences, 21:9203, Dec 2020. URL: https://doi.org/10.3390/ijms21239203, doi:10.3390/ijms21239203. This article has 18 citations and is from a peer-reviewed journal.
4. (daams2020nemolikekinasein pages 7-8): Renée Daams and Ramin Massoumi. Nemo-like kinase in development and diseases: insights from mouse studies. International Journal of Molecular Sciences, 21:9203, Dec 2020. URL: https://doi.org/10.3390/ijms21239203, doi:10.3390/ijms21239203. This article has 18 citations and is from a peer-reviewed journal.
5. (ishitani2003regulationoflymphoid pages 1-2): Tohru Ishitani, J. Ninomiya-Tsuji, and Kunihiro Matsumoto. Regulation of lymphoid enhancer factor 1/t-cell factor by mitogen-activated protein kinase-related nemo-like kinase-dependent phosphorylation in wnt/β-catenin signaling. Molecular and Cellular Biology, 23:1379-1389, Feb 2003. URL: https://doi.org/10.1128/mcb.23.4.1379-1389.2003, doi:10.1128/mcb.23.4.1379-1389.2003. This article has 287 citations and is from a domain leading peer-reviewed journal.
6. (ishitani2010nemolikekinasesuppresses pages 1-6): Tohru Ishitani, T. Hirao, Maho Suzuki, Miho Isoda, Shizuka Ishitani, K. Harigaya, M. Kitagawa, Kunihiro Matsumoto, and Motoyuki Itoh. Nemo-like kinase suppresses notch signalling by interfering with formation of the notch active transcriptional complex. Nature Cell Biology, 12:278-285, Mar 2010. URL: https://doi.org/10.1038/ncb2028, doi:10.1038/ncb2028. This article has 155 citations and is from a highest quality peer-reviewed journal.
7. (ishitani2010nemolikekinasesuppresses pages 19-24): Tohru Ishitani, T. Hirao, Maho Suzuki, Miho Isoda, Shizuka Ishitani, K. Harigaya, M. Kitagawa, Kunihiro Matsumoto, and Motoyuki Itoh. Nemo-like kinase suppresses notch signalling by interfering with formation of the notch active transcriptional complex. Nature Cell Biology, 12:278-285, Mar 2010. URL: https://doi.org/10.1038/ncb2028, doi:10.1038/ncb2028. This article has 155 citations and is from a highest quality peer-reviewed journal.
8. (ishitani2011homodimerizationofnemolike pages 1-2): Shizuka Ishitani, Kenji Inaba, Kunihiro Matsumoto, and Tohru Ishitani. Homodimerization of nemo-like kinase is essential for activation and nuclear localization. Molecular Biology of the Cell, 22:266-277, Jan 2011. URL: https://doi.org/10.1091/mbc.e10-07-0605, doi:10.1091/mbc.e10-07-0605. This article has 42 citations and is from a domain leading peer-reviewed journal.
9. (ishitani2011homodimerizationofnemolike pages 9-11): Shizuka Ishitani, Kenji Inaba, Kunihiro Matsumoto, and Tohru Ishitani. Homodimerization of nemo-like kinase is essential for activation and nuclear localization. Molecular Biology of the Cell, 22:266-277, Jan 2011. URL: https://doi.org/10.1091/mbc.e10-07-0605, doi:10.1091/mbc.e10-07-0605. This article has 42 citations and is from a domain leading peer-reviewed journal.
10. (li2014nemolikekinase(nlk) pages 15-18): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
11. (li2019phosphorylationofmavsvisa pages 1-2): Shang-Ze Li, Qi-Peng Shu, Yang Song, Hui-Hui Zhang, Yi Liu, Bing-Xue Jin, Tianzi Liuyu, Chao Li, Xi-Chen Huang, Run-Lei Du, Wei Song, Bo Zhong, and Xiao-Dong Zhang. Phosphorylation of mavs/visa by nemo-like kinase (nlk) for degradation regulates the antiviral innate immune response. Nature Communications, Jul 2019. URL: https://doi.org/10.1038/s41467-019-11258-x, doi:10.1038/s41467-019-11258-x. This article has 60 citations and is from a highest quality peer-reviewed journal.
12. (masoumi2017nlkmediatedphosphorylationof pages 10-10): Katarzyna Chmielarska Masoumi, Renée Daams, Wondossen Sime, Valentina Siino, Hengning Ke, Fredrik Levander, and Ramin Massoumi. Nlk-mediated phosphorylation of hdac1 negatively regulates wnt signaling. Molecular Biology of the Cell, 28:346-355, Jan 2017. URL: https://doi.org/10.1091/mbc.e16-07-0547, doi:10.1091/mbc.e16-07-0547. This article has 36 citations and is from a domain leading peer-reviewed journal.
13. (moon2017phosphorylationbynlk pages 1-2): Sungho Moon, Wantae Kim, Soyoung Kim, Youngeun Kim, Yonghee Song, Oleksii Bilousov, Jiyoung Kim, Taebok Lee, Boksik Cha, Minseong Kim, Hanjun Kim, Vladimir L Katanaev, and Eek‐hoon Jho. Phosphorylation by nlk inhibits yap‐14‐3‐3‐interactions and induces its nuclear localization. EMBO reports, 18:61-71, Jan 2017. URL: https://doi.org/10.15252/embr.201642683, doi:10.15252/embr.201642683. This article has 182 citations and is from a highest quality peer-reviewed journal.
14. (ohnishi2010nemolikekinasean pages 1-2): Eriko Ohnishi, Toshiyasu Goto, Atsushi Sato, Mi-sun Kim, Shun-ichiro Iemura, Tohru Ishitani, Tohru Natsume, Junji Ohnishi, and Hiroshi Shibuya. Nemo-like kinase, an essential effector of anterior formation, functions downstream of p38 mitogen-activated protein kinase. Molecular and Cellular Biology, 30:675-683, Feb 2010. URL: https://doi.org/10.1128/mcb.00576-09, doi:10.1128/mcb.00576-09. This article has 25 citations and is from a domain leading peer-reviewed journal.
15. (ohnishi2010nemolikekinasean pages 8-9): Eriko Ohnishi, Toshiyasu Goto, Atsushi Sato, Mi-sun Kim, Shun-ichiro Iemura, Tohru Ishitani, Tohru Natsume, Junji Ohnishi, and Hiroshi Shibuya. Nemo-like kinase, an essential effector of anterior formation, functions downstream of p38 mitogen-activated protein kinase. Molecular and Cellular Biology, 30:675-683, Feb 2010. URL: https://doi.org/10.1128/mcb.00576-09, doi:10.1128/mcb.00576-09. This article has 25 citations and is from a domain leading peer-reviewed journal.
16. (wang2021therapeutictargetingof pages 1-2): Xian Wang, J. Veeraraghavan, C. Liu, Xi-xi Cao, L. Qin, Jin-Ah Kim, Ying Tan, S. K. Loo, Yiheng Hu, Ling Lin, Sanghoon Lee, M. Shea, Tamika Mitchell, Shunqiang Li, M. Ellis, S. Hilsenbeck, R. Schiff, and Xiao-Song Wang. Therapeutic targeting of nemo-like kinase in primary and acquired endocrine-resistant breast cancer. Clinical Cancer Research, 27:2648-2662, Feb 2021. URL: https://doi.org/10.1158/1078-0432.ccr-20-2961, doi:10.1158/1078-0432.ccr-20-2961. This article has 12 citations and is from a highest quality peer-reviewed journal.
17. (wilkes2020diamondblackfananemia pages 1-2): M. Wilkes, K. Siva, Jun Chen, G. Varetti, M. Youn, H. Chae, F. Ek, R. Olsson, T. Lundbäck, D. Dever, T. Nishimura, A. Narla, B. Glader, H. Nakauchi, H. Nakauchi, M. Porteus, C. Repellin, H. Gazda, H. Gazda, Sijie Lin, M. Serrano, J. Flygare, and K. Sakamoto. Diamond blackfan anemia is mediated by hyperactive nemo-like kinase. Nature Communications, Jul 2020. URL: https://doi.org/10.1038/s41467-020-17100-z, doi:10.1038/s41467-020-17100-z. This article has 16 citations and is from a highest quality peer-reviewed journal.
18. (ishitani2011homodimerizationofnemolike pages 2-3): Shizuka Ishitani, Kenji Inaba, Kunihiro Matsumoto, and Tohru Ishitani. Homodimerization of nemo-like kinase is essential for activation and nuclear localization. Molecular Biology of the Cell, 22:266-277, Jan 2011. URL: https://doi.org/10.1091/mbc.e10-07-0605, doi:10.1091/mbc.e10-07-0605. This article has 42 citations and is from a domain leading peer-reviewed journal.
19. (ishitani2011homodimerizationofnemolike pages 3-4): Shizuka Ishitani, Kenji Inaba, Kunihiro Matsumoto, and Tohru Ishitani. Homodimerization of nemo-like kinase is essential for activation and nuclear localization. Molecular Biology of the Cell, 22:266-277, Jan 2011. URL: https://doi.org/10.1091/mbc.e10-07-0605, doi:10.1091/mbc.e10-07-0605. This article has 42 citations and is from a domain leading peer-reviewed journal.
20. (ishitani2011homodimerizationofnemolike pages 4-5): Shizuka Ishitani, Kenji Inaba, Kunihiro Matsumoto, and Tohru Ishitani. Homodimerization of nemo-like kinase is essential for activation and nuclear localization. Molecular Biology of the Cell, 22:266-277, Jan 2011. URL: https://doi.org/10.1091/mbc.e10-07-0605, doi:10.1091/mbc.e10-07-0605. This article has 42 citations and is from a domain leading peer-reviewed journal.
21. (kim2010regulationoffoxo1 pages 2-4): Sunhong Kim, YongSung Kim, Jiwoon Lee, and Jongkyeong Chung. Regulation of foxo1 by tak1-nemo-like kinase pathway\*. The Journal of Biological Chemistry, 285:8122-8129, Jan 2010. URL: https://doi.org/10.1074/jbc.m110.101824, doi:10.1074/jbc.m110.101824. This article has 54 citations.
22. (li2014nemolikekinase(nlk) pages 1-3): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
23. (li2014nemolikekinase(nlk) pages 3-5): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
24. (li2014nemolikekinase(nlk) pages 5-6): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
25. (li2014nemolikekinase(nlk) pages 6-8): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
26. (li2014nemolikekinase(nlk) pages 8-10): Shang-Ze Li, Hui-Hui Zhang, Jun-bo Liang, Yang Song, Bing-Xue Jin, Na-Na Xing, G. Fan, Run-Lei Du, and Xiao-Dong Zhang. Nemo-like kinase (nlk) negatively regulates nf-kappa b activity through disrupting the interaction of tak1 with ikkβ. Biochimica et biophysica acta, 1843 7:1365-72, Jul 2014. URL: https://doi.org/10.1016/j.bbamcr.2014.03.028, doi:10.1016/j.bbamcr.2014.03.028. This article has 40 citations.
27. (li2019phosphorylationofmavsvisa pages 12-13): Shang-Ze Li, Qi-Peng Shu, Yang Song, Hui-Hui Zhang, Yi Liu, Bing-Xue Jin, Tianzi Liuyu, Chao Li, Xi-Chen Huang, Run-Lei Du, Wei Song, Bo Zhong, and Xiao-Dong Zhang. Phosphorylation of mavs/visa by nemo-like kinase (nlk) for degradation regulates the antiviral innate immune response. Nature Communications, Jul 2019. URL: https://doi.org/10.1038/s41467-019-11258-x, doi:10.1038/s41467-019-11258-x. This article has 60 citations and is from a highest quality peer-reviewed journal.
28. (moon2017phosphorylationbynlk pages 6-7): Sungho Moon, Wantae Kim, Soyoung Kim, Youngeun Kim, Yonghee Song, Oleksii Bilousov, Jiyoung Kim, Taebok Lee, Boksik Cha, Minseong Kim, Hanjun Kim, Vladimir L Katanaev, and Eek‐hoon Jho. Phosphorylation by nlk inhibits yap‐14‐3‐3‐interactions and induces its nuclear localization. EMBO reports, 18:61-71, Jan 2017. URL: https://doi.org/10.15252/embr.201642683, doi:10.15252/embr.201642683. This article has 182 citations and is from a highest quality peer-reviewed journal.
29. (ohnishi2010nemolikekinasean pages 2-3): Eriko Ohnishi, Toshiyasu Goto, Atsushi Sato, Mi-sun Kim, Shun-ichiro Iemura, Tohru Ishitani, Tohru Natsume, Junji Ohnishi, and Hiroshi Shibuya. Nemo-like kinase, an essential effector of anterior formation, functions downstream of p38 mitogen-activated protein kinase. Molecular and Cellular Biology, 30:675-683, Feb 2010. URL: https://doi.org/10.1128/mcb.00576-09, doi:10.1128/mcb.00576-09. This article has 25 citations and is from a domain leading peer-reviewed journal.
30. (qin2024canonicalandnoncanonical pages 12-13): Kevin Qin, Michael Yu, Jiaming Fan, Hongwei Wang, Piao Zhao, Guozhi Zhao, Wei Zeng, Connie Chen, Yonghui Wang, Annie Wang, Zander Schwartz, Jeffrey Hong, Lily Song, William Wagstaff, Rex C. Haydon, Hue H. Luu, Sherwin H. Ho, Jason Strelzow, Russell R. Reid, Tong-Chuan He, and Lewis L. Shi. Canonical and noncanonical wnt signaling: multilayered mediators, signaling mechanisms and major signaling crosstalk. Genes & Diseases, 11:103-134, Jan 2024. URL: https://doi.org/10.1016/j.gendis.2023.01.030, doi:10.1016/j.gendis.2023.01.030. This article has 126 citations.
31. (satoh2007nemolikekinasemyocyteenhancer pages 2-4): Kiyotoshi Satoh, Junji Ohnishi, Atsushi Sato, Michio Takeyama, Shun-ichiro Iemura, Tohru Natsume, and Hiroshi Shibuya. Nemo-like kinase-myocyte enhancer factor 2a signaling regulates anterior formation in xenopus development. Molecular and Cellular Biology, 27:7623-7630, Nov 2007. URL: https://doi.org/10.1128/mcb.01481-07, doi:10.1128/mcb.01481-07. This article has 26 citations and is from a domain leading peer-reviewed journal.
32. (wilkes2020diamondblackfananemia pages 6-6): M. Wilkes, K. Siva, Jun Chen, G. Varetti, M. Youn, H. Chae, F. Ek, R. Olsson, T. Lundbäck, D. Dever, T. Nishimura, A. Narla, B. Glader, H. Nakauchi, H. Nakauchi, M. Porteus, C. Repellin, H. Gazda, H. Gazda, Sijie Lin, M. Serrano, J. Flygare, and K. Sakamoto. Diamond blackfan anemia is mediated by hyperactive nemo-like kinase. Nature Communications, Jul 2020. URL: https://doi.org/10.1038/s41467-020-17100-z, doi:10.1038/s41467-020-17100-z. This article has 16 citations and is from a highest quality peer-reviewed journal.
33. (yasuda2003nemolikekinaseinduces pages 6-7): J. Yasuda, Akira Tsuchiya, T. Yamada, M. Sakamoto, T. Sekiya, and S. Hirohashi. Nemo-like kinase induces apoptosis in dld-1 human colon cancer cells. Biochemical and biophysical research communications, 308 2:227-33, Aug 2003. URL: https://doi.org/10.1016/s0006-291x(03)01343-3, doi:10.1016/s0006-291x(03)01343-3. This article has 70 citations and is from a peer-reviewed journal.
34. (zanotti2012nemo‐likekinaseinhibits pages 8-8): S. Zanotti and E. Canalis. Nemo‐like kinase inhibits osteoblastogenesis by suppressing bone morphogenetic protein and wnt canonical signaling. Journal of Cellular Biochemistry, Feb 2012. URL: https://doi.org/10.1002/jcb.23365, doi:10.1002/jcb.23365. This article has 20 citations and is from a peer-reviewed journal.