1. Phylogeny  
   STKLD1, also known by its alternative names SgK071, C9orf96, and SGK071, is classified as a serine/threonine kinase‐like domain‐containing pseudokinase within the human kinome. Sequence‐based phylogenetic analyses indicate that STKLD1 shares the common overall bilobal kinase fold with other eukaryotic serine/threonine kinases, yet it exhibits notable deviations in the conserved catalytic motifs. In particular, studies of human kinase complements have identified a subset of kinase‐like proteins that lack one or more residues essential for conventional catalysis; STKLD1 falls into this evolutionary group where divergence in the VAIK, HRD, and DFG motifs is observed. Comparative analysis based on the seminal work of Manning and colleagues shows that while classical serine/threonine kinases are grouped principally among the AGC, CAMK, CMGC, TK, and TKL families, STKLD1 is positioned within the group of pseudokinases that are evolutionarily derived from an ancestral serine/threonine kinase gene. This subset of kinases is found throughout mammalian species, and similar kinase‐like proteins have been reported across diverse taxa, illustrating an early divergence in kinase evolution that has resulted in proteins with a conserved structural framework but variable catalytic potential. The divergence in sequence and degradation of key catalytic residues—elements that are typically conserved in active kinases—has led to the inclusion of STKLD1 in catalogs of human pseudokinases, supporting its assignment within groups defined by such motif deficiencies. This phylogenetic context underscores that while STKLD1 maintains the overall structural scaffold of the kinase fold, its evolutionary history is more aligned with proteins that have transitioned toward non‐catalytic regulatory roles (eyers2013dawnofthe pages 1-2, murphy2014arobustmethodology pages 9-11, boudeau2006emergingrolesof pages 2-4).
2. Reaction Catalyzed  
   The canonical reaction catalyzed by serine/threonine kinases involves the transfer of the γ‐phosphate from ATP to the hydroxyl group of a serine or threonine residue in substrate proteins. In biochemical terms, the reaction is represented as follows:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction requires the binding of ATP in a correctly configured kinase active site and is typically coupled with substrate recognition through specific amino acid motifs. For STKLD1, which contains a kinase‐like domain, the canonical reaction would follow this overall mechanism; however, its classification as a pseudokinase implies that the intrinsic catalytic activity may be compromised or absent due to degradation of essential catalytic residues (bailey2014biochemicalanalysisof pages 23-26).
3. Cofactor Requirements  
   In active serine/threonine kinases, magnesium ions (Mg²⁺) serve as a critical cofactor by coordinating the phosphoryl groups of ATP and thereby facilitating the phosphate transfer reaction. Accordingly, the reaction mechanism traditionally requires Mg²⁺ to stabilize the negative charges that develop during phosphoryl transfer. As with many members of the serine/threonine kinase superfamily, the catalytic mechanism of STKLD1 is expected to be Mg²⁺‐dependent, even though the exact cofactor requirement for STKLD1 has not been exhaustively characterized in functional assays. Based on the conserved properties of kinases and pseudokinases with similar structural features, the presence of Mg²⁺ is assumed to be integral to any nucleotide‐binding activity that may occur (bailey2014biochemicalanalysisof pages 23-26).
4. Substrate Specificity  
   Serine/threonine kinases characteristically recognize specific consensus motifs in their substrates. For example, many such enzymes display a substrate preference for sequences that contain basic residues surrounding the target serine or threonine or demonstrate a particular spatial arrangement of amino acids that is accommodated by the substrate‐binding groove. The substrate specificity of conventional kinases is determined by conserved elements within the kinase domain, including the glycine‐rich loop, the β3 strand lysine, and the catalytic loop that contains the HRD motif, as well as the DFG motif that coordinates magnesium ions. In contrast, for STKLD1 the substrate specificity remains experimentally uncharacterized. Although the overall kinase fold is conserved, available biochemical data indicate that STKLD1 lacks clearly defined catalytic activity and the conventional nucleotide‐binding and substrate‐alignment motifs appear degraded. This suggests that the canonical substrate recognition mechanism observed in active kinases may not apply in a straightforward manner to STKLD1. Instead, insights gathered from studies of related pseudokinases imply that the substrate‐binding region may either be repurposed for non‐catalytic functions or may participate in protein–protein interactions that do not involve phosphorylation of classic serine/threonine consensus motifs. No consensus substrate motif has been confidently established for STKLD1 in published sources, and it remains to be determined whether STKLD1 can bind substrates in a manner similar to active serine/threonine kinases or if it functions primarily as a scaffolding or regulatory molecule (foulkes2018biochemicalanalysisof pages 65-66, murphy2014arobustmethodology pages 26-26, bailey2014biochemicalanalysisof pages 23-26).
5. Structure  
   The domain organization of STKLD1 is characterized by a central kinase‐like domain that adopts the canonical bilobal structure seen in protein kinases. The N-terminal lobe of the kinase fold is primarily comprised of a small β-sheet structure, and it is connected via a hinge region to a larger C-terminal lobe that is predominantly helical. Detailed analyses indicate that the glycine-rich loop, which is typically involved in ATP binding, is notably degraded in STKLD1; for instance, alterations to the canonical GxGxxG sequence are observed (scheeff2009structureofthe pages 4-5). In addition, critical residues within the VAIK motif, which normally stabilize ATP binding through the formation of salt bridges, are modified or substituted, and the aspartate present in the HRD motif required for catalytic activity is absent or replaced by non-canonical residues. The DFG motif, essential for coordinating Mg²⁺ and determining active–inactive conformations in canonical kinases, also exhibits deviations from its standard sequence.  
   Structural prediction models, including those generated by AlphaFold, indicate that despite these divergence events, the overall kinase fold remains intact. The predicted three-dimensional structure of STKLD1 reveals a preserved bilobal architecture with a distinct ATP‐binding cleft, albeit with potential modifications in the substrate and ligand interaction regions. The C-helix, an important regulatory element in kinases that typically forms a salt bridge with a lysine residue from the β3 strand, is present but may adopt an altered conformation relative to catalytically active kinases. Structural features such as the hydrophobic spines, which are critical for maintaining the active conformation in conventional kinases, are only partially conserved in STKLD1. Moreover, the overall organization of secondary structure elements—including the β-sheets and α-helices within both lobes—is consistent with a kinase-type fold, reinforcing the classification of STKLD1 as a kinase‐like pseudokinase.  
   Additional 3D structural modeling data from subcellular localization studies indicate that STKLD1 is predominantly cytosolic, with its kinase‐like domain exposed to the cytoplasm where it may potentially interact with other proteins or regulatory factors. These structural observations support a role for STKLD1 that is distinct from those of active catalytic kinases, in that the preserved overall fold may function as a protein-protein interaction module or conformational switch within cellular signaling complexes. The loss or alteration of key catalytic amino acids underscores the possibility that STKLD1 operates primarily through scaffolding or allosteric regulatory mechanisms rather than traditional phosphotransferase activity (scheeff2009structureofthe pages 4-5, murphy2014arobustmethodology pages 6-8, zhang2021asubcellularmap pages 4-6).
6. Regulation  
   Regulatory mechanisms for serine/threonine kinases typically involve post-translational modifications, such as phosphorylation, and interactions with regulatory proteins that modulate the conformation of the kinase domain. For STKLD1, current data indicate that detailed regulatory processes have not been fully elucidated. However, examination of related pseudokinases provides a framework for understanding possible regulatory modes. In active kinases, the phosphorylation of activation loop residues, as well as autoinhibitory mechanisms that involve residues in the N-terminal or C-terminal regions, play central roles in modulating catalytic competence. In the case of STKLD1, the degradation of key catalytic motifs suggests that it does not undergo regulation via classical mechanisms that control phosphotransfer activity. Instead, studies on other pseudokinases have demonstrated that proteins with similar divergence in the nucleotide-binding pocket may be subject to allosteric regulation through protein-protein interactions or conformational changes induced by ligand binding. In particular, some pseudokinases function as molecular scaffolds and may be regulated by transient interactions that do not involve direct catalytic turnover.  
   A report by Bayliss et al. has listed STKLD1 as having potential regulatory input through mechanisms such as Tyr-down autoinhibition, in which a tyrosine residue within or near the regulatory spine participates in conformational stabilization of an inactive state. Although the precise phosphorylation sites and the identity of regulatory enzymes (such as upstream kinases or phosphatases) have not been specifically mapped for STKLD1, the possibility exists that STKLD1 may be modified post-translationally to influence its conformation and interaction capabilities. Additionally, thermal shift assays employed in pseudokinase subclassification studies have indicated that STKLD1 is capable of binding both nucleotides and divalent cations, which suggests that binding-induced stabilization of the kinase domain may contribute to its regulatory state. These biochemical assays imply that STKLD1, like other members of the pseudokinase family, may undergo subtle conformational transitions upon ligand association that impact its interaction with potential binding partners. Despite these insights, specific regulatory modifications such as phosphorylation, ubiquitination, or interactions with dedicated modulatory subunits remain to be definitively reported through experimental investigation. Consequently, the regulatory profile of STKLD1 is currently described in terms of potential allosteric and conformational regulation mechanisms rather than through detailed mappings of post-translational modification events (bayliss2015theysand pages 14-20, foulkes2018biochemicalanalysisof pages 65-66).
7. Function  
   The biological function of STKLD1 remains largely uncharacterized, with available literature indicating that its role within cellular signaling pathways has not been definitively established. Expression studies in kinome profiling experiments have incorporated STKLD1 into broader analyses of human kinases, and in such contexts it is listed among the human pseudokinases. For instance, kinome analyses performed in human lung epithelial cells (alveolar type II and basal cells) have identified STKLD1 as part of the overall kinase repertoire, with its expression profile included in reprogramming studies associated with lung cancer, although no specific functional role has been assigned to it based on these data (leach2019thekinomeof pages 42-42). In addition, in silico analyses of protein–protein interaction networks, particularly those related to sperm markers involved in oocyte activation, have predicted that STKLD1 may interact with key regulatory kinases such as SRC and protein kinase C isoforms. Such predictive interaction networks place STKLD1 among a subset of kinase-like proteins that are potentially involved in the regulation of fertilization-related signaling cascades. Moreover, review articles on pseudokinases highlight that although many such proteins lack catalytic activity, they frequently serve as scaffolds or molecular switches within multiprotein complexes, thereby indirectly influencing downstream signaling pathways through non-catalytic mechanisms.  
   STKLD1 is predominantly localized in the cytosol, as indicated by recent high-resolution subcellular localization studies, and this compartmentalization is consistent with a role in mediating cytosolic signaling events or in forming complexes with other regulatory proteins. Despite the prediction of nucleotide and cation binding properties—features that are maintained even in pseudokinase domains—the precise substrates or interacting proteins of STKLD1 have not been conclusively determined through direct biochemical assays. In addition, while some kinases in the pseudokinase group have been implicated in developmental and cellular stress‐response pathways, STKLD1 itself has not been firmly associated with any specific signaling cascade or cellular process. Overall, the functional profile of STKLD1 is defined, based on current evidence, by its inclusion in the pseudokinase family with a conserved structural fold, its predicted cytosolic localization, and its potential involvement in non‐catalytic regulatory roles that might include scaffolding interactions with other signal transduction proteins (eyers2013dawnofthe pages 1-2, foulkes2018biochemicalanalysisof pages 65-66, virgili…Unknownyearpredictionofsperm pages 157-159, leach2019thekinomeof pages 42-42).
8. Other Comments  
   Additional information regarding STKLD1 highlights that the protein is frequently identified in kinome profiling studies despite the absence of well-defined catalytic activity. Its inclusion in the human kinome as a pseudokinase emphasizes its potential as a regulatory entity, and the various alternative nomenclatures (SgK071, C9orf96, SGK071) point to its recognition by multiple research groups using different databases and annotations. Although no selective inhibitors have been reported that specifically target STKLD1, its presence in therapeutic cysteinome analyses suggests that the accessibility of cysteine residues within its kinase domain may be considered in the context of drug discovery targeting kinase-like regulatory proteins. Furthermore, while disease associations for many pseudokinases have been documented, STKLD1 has not been conclusively linked to any particular pathological condition or genetic disorder in the current literature. Its potential role in sperm signaling and oocyte activation, as predicted by in silico protein–protein interaction studies, raises the possibility that it might have tissue-specific functions that are yet to be validated experimentally. In addition, structural and biochemical methodologies such as thermal shift assays have been used to classify STKLD1 among pseudokinases that retain some nucleotide- and cation-binding capabilities, a feature that indicates the possibility of ligand-induced conformational regulation. Overall, further experimental work is required to determine whether STKLD1 exerts its cellular function solely as a scaffolding protein or if it participates in regulatory signaling through alternative, non-catalytic mechanisms (bayliss2015theysand pages 14-20, murphy2014arobustmethodology pages 6-8, li2022therapeutictargetingthe pages 29-29).
9. References
10. Eyers, P. A. and Murphy, J. M. (2013). Dawn of the dead: protein pseudokinases signal new adventures in cell biology. Biochemical Society Transactions, 41(4):969-74 (eyers2013dawnofthe pages 1-2).
11. Eyers, P. A. and Murphy, J. M. (2013). Dawn of the dead: protein pseudokinases signal new adventures in cell biology. Biochemical Society Transactions, 41(4):969-74 (eyers2013dawnofthe pages 4-5).
12. Murphy, J. M., Zhang, Q., Young, S. N., Reese, M. L., Bailey, F. P., Eyers, P. A., et al. (2014). A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical Journal, 457(2):323-34 (murphy2014arobustmethodology pages 9-11, murphy2014arobustmethodology pages 6-8, murphy2014arobustmethodology pages 26-26, murphy2014arobustmethodology pages 14-15, murphy2014arobustmethodology pages 4-6, murphy2014arobustmethodology pages 8-9).
13. Boudeau, J., Miranda-Saavedra, D., Barton, G. J., and Alessi, D. R. (2006). Emerging roles of pseudokinases. Trends in Cell Biology, 16:443-452 (boudeau2006emergingrolesof pages 2-4).
14. Scheeff, E. D., Eswaran, J., Bunkoczi, G., Knapp, S., and Manning, G. (2009). Structure of the pseudokinase VRK3 reveals a degraded catalytic site, a highly conserved kinase fold, and a putative regulatory binding site. Structure, 17:128-138 (scheeff2009structureofthe pages 4-5).
15. Leach, S. M., Finigan, J., Vasu, V. T., Mishra, R., Ghosh, M., Foster, D., Mason, R., Kosmider, B., Hesson, E. F., and Kern, J. A. (2019). The kinome of human alveolar type II and basal cells, and its reprogramming in lung cancer. American Journal of Respiratory Cell and Molecular Biology, 61:481-491 (leach2019thekinomeof pages 36-40, leach2019thekinomeof pages 42-42).
16. Thiriet, M. (2013). Cytoplasmic protein serine/threonine kinases. In Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 1-4, 76-78 (thiriet2013cytoplasmicproteinserinethreonine pages 1-4, thiriet2013cytoplasmicproteinserinethreonine pages 76-78).
17. Zhang, H., Cao, X., Tang, M., Zhong, G., Si, Y., Li, H., Zhu, F., Liao, Q., Li, L., Zhao, J., Feng, J., Li, S., Wang, C., Kaulich, M., Wang, F., Chen, L., Li, L., Xia, Z., Liang, T., Lu, H., Feng, X.-H., and Zhao, B. (2021). A subcellular map of the human kinome. eLife, 10:e64943 (zhang2021asubcellularmap pages 4-6).
18. Bayliss, R., Haq, T., and Yeoh, S. (2015). The ys and wherefores of protein kinase autoinhibition. Biochimica et Biophysica Acta (BBA) - Proteins and Proteomics, 1854:1586-1594 (bayliss2015theysand pages 14-20).
19. Li, L., Meyer, C. J., Zhou, Z.-W., Elmezayen, A. D., and Westover, K. (2022). Therapeutic targeting the allosteric cysteinome of Ras and kinase families. Journal of Molecular Biology, 434:167626 (li2022therapeutictargetingthe pages 29-29).
20. Epelboin, Y., Quintric, L., Guévélou, E., Boudry, P., Pichereau, V., and Corporeau, C. (2016). The kinome of Pacific oyster Crassostrea gigas, its expression during development and in response to environmental factors. PLOS ONE, 11:e0155435 (epelboin2016thekinomeof pages 2-4, epelboin2016thekinomeof pages 7-8, epelboin2016thekinomeof pages 8-11, epelboin2016thekinomeof pages 11-12).

References

1. (foulkes2018biochemicalanalysisof pages 65-66): DM Foulkes. Biochemical analysis of tribbles 2 pseudokinase using repurposed kinase inhibitors. Unknown journal, 2018.
2. (murphy2014arobustmethodology pages 9-11): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.
3. (bailey2014biochemicalanalysisof pages 23-26): F Bailey. Biochemical analysis of human cancer-associated pseudokinases. Unknown journal, 2014.
4. (eyers2013dawnofthe pages 1-2): Patrick A. Eyers and James M. Murphy. Dawn of the dead: protein pseudokinases signal new adventures in cell biology. Biochemical Society transactions, 41 4:969-74, Aug 2013. URL: https://doi.org/10.1042/bst20130115, doi:10.1042/bst20130115. This article has 109 citations and is from a peer-reviewed journal.
5. (eyers2013dawnofthe pages 4-5): Patrick A. Eyers and James M. Murphy. Dawn of the dead: protein pseudokinases signal new adventures in cell biology. Biochemical Society transactions, 41 4:969-74, Aug 2013. URL: https://doi.org/10.1042/bst20130115, doi:10.1042/bst20130115. This article has 109 citations and is from a peer-reviewed journal.
6. (leach2019thekinomeof pages 36-40): Sonia M. Leach, Jay Finigan, Vihas T. Vasu, Rangnath Mishra, Moumita Ghosh, Daniel Foster, Robert Mason, Beata Kosmider, Eveline Farias Hesson, and Jeffrey A. Kern. The kinome of human alveolar type ii and basal cells, and its reprogramming in lung cancer. American Journal of Respiratory Cell and Molecular Biology, 61:481-491, Oct 2019. URL: https://doi.org/10.1165/rcmb.2018-0283oc, doi:10.1165/rcmb.2018-0283oc. This article has 2 citations and is from a peer-reviewed journal.
7. (murphy2014arobustmethodology pages 26-26): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.
8. (murphy2014arobustmethodology pages 6-8): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.
9. (scheeff2009structureofthe pages 4-5): Eric D. Scheeff, Jeyanthy Eswaran, Gabor Bunkoczi, Stefan Knapp, and Gerard Manning. Structure of the pseudokinase vrk3 reveals a degraded catalytic site, a highly conserved kinase fold, and a putative regulatory binding site. Structure(London, England:1993), 17:128-138, Jan 2009. URL: https://doi.org/10.1016/j.str.2008.10.018, doi:10.1016/j.str.2008.10.018. This article has 229 citations.
10. (thiriet2013cytoplasmicproteinserinethreonine pages 1-4): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
11. (thiriet2013cytoplasmicproteinserinethreonine pages 76-78): M Thiriet M Thiriet. Cytoplasmic protein serine/threonine kinases. Biomathematical and Biomechanical Modeling of the Circulatory and Ventilatory Systems, pages 175-310, Jul 2013. URL: https://doi.org/10.1007/978-1-4614-4370-4\_5, doi:10.1007/978-1-4614-4370-4\_5. This article has 11 citations.
12. (virgili…Unknownyearpredictionofsperm pages 157-159): RO Virgili… MT Massana, MB Monasterio. Prediction of sperm markers involved in oocyte activation by in silico analysis of protein-protein interactions. Unknown journal, Unknown year.
13. (zhang2021asubcellularmap pages 4-6): Haitao Zhang, Xiaolei Cao, Mei Tang, Guoxuan Zhong, Yuan Si, Haidong Li, Feifeng Zhu, Qinghua Liao, Liuju Li, Jianhui Zhao, Jia Feng, Shuaifeng Li, Chenliang Wang, Manuel Kaulich, Fangwei Wang, Liangyi Chen, Li Li, Zongping Xia, Tingbo Liang, Huasong Lu, Xin-Hua Feng, and Bin Zhao. A subcellular map of the human kinome. eLife, May 2021. URL: https://doi.org/10.7554/elife.64943, doi:10.7554/elife.64943. This article has 80 citations and is from a domain leading peer-reviewed journal.
14. (bayliss2015theysand pages 14-20): Richard Bayliss, Tamanna Haq, and Sharon Yeoh. The ys and wherefores of protein kinase autoinhibition. Biochimica et Biophysica Acta (BBA) - Proteins and Proteomics, 1854:1586-1594, Oct 2015. URL: https://doi.org/10.1016/j.bbapap.2015.04.025, doi:10.1016/j.bbapap.2015.04.025. This article has 21 citations.
15. (boudeau2006emergingrolesof pages 2-4): Jérôme Boudeau, Diego Miranda-Saavedra, Geoffrey J. Barton, and Dario R. Alessi. Emerging roles of pseudokinases. Trends in Cell Biology, 16:443-452, Sep 2006. URL: https://doi.org/10.1016/j.tcb.2006.07.003, doi:10.1016/j.tcb.2006.07.003. This article has 647 citations and is from a domain leading peer-reviewed journal.
16. (epelboin2016thekinomeof pages 11-12): Yanouk Epelboin, Laure Quintric, Eric Guévélou, Pierre Boudry, Vianney Pichereau, and Charlotte Corporeau. The kinome of pacific oyster crassostrea gigas, its expression during development and in response to environmental factors. PLOS ONE, 11:e0155435, May 2016. URL: https://doi.org/10.1371/journal.pone.0155435, doi:10.1371/journal.pone.0155435. This article has 20 citations and is from a peer-reviewed journal.
17. (epelboin2016thekinomeof pages 2-4): Yanouk Epelboin, Laure Quintric, Eric Guévélou, Pierre Boudry, Vianney Pichereau, and Charlotte Corporeau. The kinome of pacific oyster crassostrea gigas, its expression during development and in response to environmental factors. PLOS ONE, 11:e0155435, May 2016. URL: https://doi.org/10.1371/journal.pone.0155435, doi:10.1371/journal.pone.0155435. This article has 20 citations and is from a peer-reviewed journal.
18. (li2022therapeutictargetingthe pages 29-29): Lianbo Li, Cynthia J. Meyer, Zhi-Wei Zhou, Ammar D. Elmezayen, and K. Westover. Therapeutic targeting the allosteric cysteinome of ras and kinase families. Journal of Molecular Biology, 434:167626, Sep 2022. URL: https://doi.org/10.1016/j.jmb.2022.167626, doi:10.1016/j.jmb.2022.167626. This article has 11 citations and is from a domain leading peer-reviewed journal.
19. (epelboin2016thekinomeof pages 7-8): Yanouk Epelboin, Laure Quintric, Eric Guévélou, Pierre Boudry, Vianney Pichereau, and Charlotte Corporeau. The kinome of pacific oyster crassostrea gigas, its expression during development and in response to environmental factors. PLOS ONE, 11:e0155435, May 2016. URL: https://doi.org/10.1371/journal.pone.0155435, doi:10.1371/journal.pone.0155435. This article has 20 citations and is from a peer-reviewed journal.
20. (epelboin2016thekinomeof pages 8-11): Yanouk Epelboin, Laure Quintric, Eric Guévélou, Pierre Boudry, Vianney Pichereau, and Charlotte Corporeau. The kinome of pacific oyster crassostrea gigas, its expression during development and in response to environmental factors. PLOS ONE, 11:e0155435, May 2016. URL: https://doi.org/10.1371/journal.pone.0155435, doi:10.1371/journal.pone.0155435. This article has 20 citations and is from a peer-reviewed journal.
21. (leach2019thekinomeof pages 42-42): Sonia M. Leach, Jay Finigan, Vihas T. Vasu, Rangnath Mishra, Moumita Ghosh, Daniel Foster, Robert Mason, Beata Kosmider, Eveline Farias Hesson, and Jeffrey A. Kern. The kinome of human alveolar type ii and basal cells, and its reprogramming in lung cancer. American Journal of Respiratory Cell and Molecular Biology, 61:481-491, Oct 2019. URL: https://doi.org/10.1165/rcmb.2018-0283oc, doi:10.1165/rcmb.2018-0283oc. This article has 2 citations and is from a peer-reviewed journal.
22. (murphy2014arobustmethodology pages 14-15): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.
23. (murphy2014arobustmethodology pages 4-6): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.
24. (murphy2014arobustmethodology pages 8-9): James M. Murphy, Qingwei Zhang, Samuel N. Young, Michael L. Reese, Fiona P. Bailey, Patrick A. Eyers, Daniela Ungureanu, Henrik Hammaren, Olli Silvennoinen, Leila N. Varghese, Kelan Chen, Anne Tripaydonis, Natalia Jura, Koichi Fukuda, Jun Qin, Zachary Nimchuk, Mary Beth Mudgett, Sabine Elowe, Christine L. Gee, Ling Liu, Roger J. Daly, Gerard Manning, Jeffrey J. Babon, and Isabelle S. Lucet. A robust methodology to subclassify pseudokinases based on their nucleotide-binding properties. The Biochemical journal, 457 2:323-34, Jan 2014. URL: https://doi.org/10.1042/bj20131174, doi:10.1042/bj20131174. This article has 295 citations.