1. Phylogeny  
   Serine‐protein kinase ATM is a serine/threonine kinase that belongs to the phosphatidylinositol 3‐kinase-related kinase (PIKK) family, an atypical but evolutionarily highly conserved branch of the human kinome. Orthologs of ATM have been identified in a myriad of eukaryotic organisms, including mammals, vertebrates, yeast, and various invertebrates, indicating that its evolutionary origin can be traced back to early eukaryotic ancestors. Phylogenetic analyses based on the protein kinase complement of the human genome reveal that ATM clusters with other long, multidomain kinases such as ATR and DNA-PKcs, all of which participate in DNA damage response pathways. These kinases share not only similar domain architectures but also overlapping functional roles in genome surveillance and the maintenance of genomic integrity, distinguishing them from canonical kinase families like AGC, CMGC, and STE (hunter2015theeukaryoticprotein pages 1-3, moret2020aresourcefor pages 1-4, oruganty2016identificationandclassification pages 12-13).
2. Reaction Catalyzed  
   The chemical reaction catalyzed by ATM is the phosphorylation of serine or threonine residues on substrate proteins. In this reaction, the gamma-phosphate group from adenosine triphosphate (ATP) is transferred to the hydroxyl group of a serine or threonine residue, yielding adenosine diphosphate (ADP), a phosphorylated protein substrate, and a proton (H⁺). This reaction is summarized as:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(phospho-L-serine/threonine) + H⁺  
   This catalytic reaction is fundamental to ATM’s role as a signal transducer in the DNA damage response, converting the detection of double-strand breaks into a cascade of phosphorylation events that ultimately regulate cell cycle arrest, apoptosis, and DNA repair processes (anti2009nonspecificserinethreonineprotein pages 1-7).
3. Cofactor Requirements  
   ATM kinase activity is dependent on the presence of divalent metal ions to coordinate ATP binding and catalysis. In particular, magnesium (Mg²⁺) is the critical cofactor required for ATM’s enzymatic activity. Mg²⁺ ions associate with ATP in the kinase active site to properly orient the gamma-phosphate for transfer and to stabilize the transition state, a requirement that is shared among serine/threonine kinases. Without sufficient levels of Mg²⁺, the catalytic efficiency of ATM would be severely compromised, thereby hindering the propagation of DNA damage signals (moret2020aresourcefor pages 1-4).
4. Substrate Specificity  
   ATM displays a distinct substrate specificity that centers on recognizing a short consensus motif consisting of either serine or threonine immediately followed by glutamine, commonly denoted as [S/T]-Q. This minimal recognition sequence is critical for ATM’s ability to phosphorylate targets that are involved in the response to DNA double-strand breaks. Large-scale substrate profiling experiments of the human serine/threonine kinome have confirmed that ATM preferentially modifies substrates containing this [S/T]-Q motif. An exemplary substrate of ATM is the histone variant H2AX, which is phosphorylated on Ser-139 to form γH2AX; this phosphorylation event is a hallmark of the DNA damage response and is instrumental for recruiting repair complexes to the site of damage. Although there is distinct substrate diversity among kinases, the preference of ATM for substrates presenting the [S/T]-Q consensus aligns with its central role in mediating cellular responses to genotoxic stress (johnson2023anatlasof pages 3-4, johnson2023anatlasof pages 6-7, yaronbarir2024theintrinsicsubstrate pages 1-2).
5. Structure  
   ATM is an exceptionally large protein kinase with an approximate molecular weight of 350 kilodaltons. Its structure is highly complex and multidomain, featuring several distinct regions that endow it with its regulatory and catalytic functions. The N-terminal portion of ATM is characterized by an abundance of HEAT repeats—tandemly arranged α-helical motifs that serve as flexible scaffolds for mediating protein–protein interactions and the assembly of high-order complexes. These HEAT repeats are thought to play roles in both autoinhibition and the recruitment of regulatory partners to ATM. Centrally located within ATM is the catalytic kinase domain, which is highly characteristic of the PIKK family. This domain contains key structural features necessary for enzymatic activity, including the activation loop, a catalytic loop harboring essential residues that coordinate ATP binding, a hydrophobic spine that helps stabilize the active conformation, and a regulatory C-helix that contributes to conformational control. Flanking the kinase domain are the FAT domain (FRAP–ATM–TRRAP) positioned near the N-terminal side of the kinase domain, and the FATC domain, located at the extreme C-terminus. Both the FAT and FATC domains contribute to the structural stability and proper folding of the kinase domain and play crucial roles in modulating ATM’s catalytic activity. Partial structural data derived from cryo-electron microscopy and predictive models from AlphaFold provide further evidence of ATM’s flexible yet ordered architecture, supporting its function as a sensor of DNA damage and orchestrator of downstream signaling events (modi2019astructurallyvalidatedmultiple pages 13-14, east2024quantitativeproteomicmass pages 13-15, maeda2025detectingproteinhigherorder pages 27-28).
6. Regulation  
   ATM is subject to intricate regulatory mechanisms that ensure its activation is precisely coupled to the presence of DNA damage. In the absence of genotoxic stress, ATM predominantly exists in an inactive state, typically forming dimers or higher-order oligomers. Upon the induction of double-strand breaks in DNA, ATM becomes activated through a series of tightly controlled post-translational modifications. Among these modifications, autophosphorylation at key residues—most notably at Ser-1981—is critically important; this autophosphorylation event triggers the dissociation of ATM dimers into active monomers, which are then competent to phosphorylate various downstream targets. In addition to autophosphorylation, ATM regulation is further modulated by ubiquitination, which influences its protein stability and turnover, and by interactions with the MRN complex (comprising MRE11, RAD50, and NBS1) that recruits ATM to sites of DNA damage. The combined effect of these modifications—phosphorylation, ubiquitination, and regulated protein–protein interactions—ensures that ATM’s kinase activity is initiated only when appropriate signals, such as double-strand breaks or oxidative stress, are present (mullerdott2025fromactivityinference pages 145-146, somale2020activationofrsk pages 12-13, o’boyle2025anatlasof pages 31-35).
7. Function  
   ATM serves as a master regulator of the cellular response to DNA damage. Its primary function is to act as a DNA damage sensor that, upon detecting double-strand breaks (DSBs) and other forms of genotoxic stress such as exposure to ionizing radiation and ultraviolet A light, activates a cascade of phosphorylation events. One of its most well-characterized roles is the phosphorylation of the histone variant H2AX on Ser-139, generating γH2AX, which serves as a recruitment signal for DNA repair machinery and is essential for proper execution of the DNA damage response. In addition, ATM phosphorylates and thereby modulates the activity of several critical proteins involved in cell cycle regulation and apoptosis, including TP53 (p53), CHEK2, BRCA1, and FANCD2. These phosphorylation events facilitate cell cycle arrest, promote efficient DNA repair, and, when damage is excessive, trigger programmed cell death. Beyond its canonical role in DNA repair, ATM is also implicated in non–DNA repair processes such as pre-B cell allelic exclusion; by repositioning one immunoglobulin heavy chain allele to pericentromeric heterochromatin, ATM helps ensure that individual B lymphocytes express a single allele, thereby enforcing receptor clonality. Moreover, ATM has been associated with functions that extend to vesicle and protein transport, T-cell development, gonadal and neurological functions, and the degradation of replication-dependent histone mRNA. Additional targets of ATM include substrates such as DYRK2, whose phosphorylation by ATM prevents its MDM2-mediated ubiquitination and subsequent degradation, as well as ATF2 and ERCC6, which are important for augmenting the DNA damage response and chromatin remodeling. ATM’s multifaceted role in cellular signaling underscores its critical function as a tumor suppressor, with defects in ATM activity leading to genomic instability and contributing to disease states such as ataxia telangiectasia (anti2009nonspecificserinethreonineprotein pages 1-7, johnson2023anatlasof pages 9-10, murray2019identifyingnoveltherapeutic pages 465-467).
8. Other Comments  
   ATM is not only pivotal for the maintenance of genome stability but also has notable clinical implications. Mutations in the ATM gene cause ataxia telangiectasia, a severe autosomal recessive disorder characterized by progressive neurodegeneration, immunodeficiency, radiosensitivity, and an increased predisposition to cancer. Given its central role in the DNA damage response, ATM is considered a tumor suppressor, and its loss or dysfunction is associated with genomic instability. In recent years, several small molecule inhibitors targeting ATM’s kinase activity—such as KU-55933, KU-60019, and AZD1390—have been developed with the aim of sensitizing cancer cells to DNA-damaging chemotherapeutic agents and radiotherapy. These compounds are being evaluated in various preclinical and clinical settings to exploit the synthetic lethal relationships that exist in tumors with defective DNA repair pathways. In addition to pharmacological inhibition, research continues to explore ATM’s involvement in diverse cellular processes, including vesicle and protein transport, pexophagy (mediated by phosphorylation of PEX5 in response to reactive oxygen species), and the regulation of immunoglobulin gene rearrangements in B cells. ATM’s broad substrate network and its regulatory complexity, as illustrated by its interactions with proteins such as DYRK2, CHEK2, and MRE11, reflect its expansive role in cell signaling and underscore its importance as a target for therapeutic intervention in cancers and other diseases related to DNA repair deficiencies (mullerdott2025fromactivityinference pages 48-52, murray2019identifyingnoveltherapeutic pages 465-467, oruganty2016identificationandclassification pages 12-13).
9. References  
   anti2009nonspecificserinethreonineprotein pages 1-7; anti2009nonspecificserinethreonineprotein pages 77-80; choy2018neurodegenerationinataxia‐telangiectasia pages 1-5; choy2018neurodegenerationinataxia‐telangiectasia pages 8-11; east2024quantitativeproteomicmass pages 1-3; east2024quantitativeproteomicmass pages 11-13; east2024quantitativeproteomicmass pages 13-15; garcia2022targetingtheatm pages 1-2; higgins2023sarscov2hijacksp38βmapk11 pages 21-23; hunter2015theeukaryoticprotein pages 1-3; hunter2015theeukaryoticprotein pages 3-6; jha2025deeplearningcoupledproximity pages 20-22; jha2025deeplearningcoupledproximity pages 22-24; jha2025deeplearningcoupledproximity pages 24-26; jiang2024illuminatingthedark pages 7-10; johnson2023anatlasof pages 1-2; johnson2023anatlasof pages 3-4; johnson2023anatlasof pages 4-5; johnson2023anatlasof pages 6-7; johnson2023anatlasof pages 7-7; johnson2023anatlasof pages 9-10; kalyuzhnyy2025applyingaconservationbased pages 24-26; maeda2025detectingproteinhigherorder pages 27-28; mcskimming2017classifyingkinaseconformations pages 14-15; modi2019astructurallyvalidated pages 22-26; modi2019astructurallyvalidated pages 26-29; modi2019astructurallyvalidated pages 29-32; modi2019astructurallyvalidated pages 32-34; modi2019astructurallyvalidatedmultiple pages 12-12; modi2019astructurallyvalidatedmultiple pages 12-13; modi2019astructurallyvalidatedmultiple pages 13-14; modi2019astructurallyvalidatedmultiple pages 14-15; modi2019astructurallyvalidatedmultiple pages 5-7; moret2020aresourcefor pages 1-4; moret2020aresourcefor pages 13-17; moret2020aresourcefor pages 29-33; moret2020aresourcefor pages 33-36; moret2020aresourcefor pages 39-43; moret2020aresourcefor pages 4-7; moret2020aresourcefor pages 7-10; mullerdott2025fromactivityinference pages 145-146; mullerdott2025fromactivityinference pages 48-52; murray2019identifyingnoveltherapeutic pages 465-467; oruganty2016identificationandclassification pages 12-13; o’boyle2025anatlasof pages 27-31; o’boyle2025anatlasof pages 31-35; o’boyle2025anatlasof pages 47-51; raman2006identificationofintracellular pages 20-24; somale2020activationofrsk pages 12-13; wasserman2023fam122aensurescell pages 32-49; yaronbarir2024theintrinsicsubstrate pages 1-2.

References

1. (anti2009nonspecificserinethreonineprotein pages 1-7): B Anti. Non-specific serine/threonine protein kinase. Class 2 Transferases, pages 1-123, Jan 2009. URL: https://doi.org/10.1007/978-3-540-85699-3\_1, doi:10.1007/978-3-540-85699-3\_1. This article has 0 citations.
2. (anti2009nonspecificserinethreonineprotein pages 77-80): B Anti. Non-specific serine/threonine protein kinase. Class 2 Transferases, pages 1-123, Jan 2009. URL: https://doi.org/10.1007/978-3-540-85699-3\_1, doi:10.1007/978-3-540-85699-3\_1. This article has 0 citations.
3. (choy2018neurodegenerationinataxia‐telangiectasia pages 1-5): Kay Rui Choy and Dianne J. Watters. Neurodegeneration in ataxia‐telangiectasia: multiple roles of atm kinase in cellular homeostasis. Developmental Dynamics, Jan 2018. URL: https://doi.org/10.1002/dvdy.24522, doi:10.1002/dvdy.24522. This article has 90 citations and is from a peer-reviewed journal.
4. (choy2018neurodegenerationinataxia‐telangiectasia pages 8-11): Kay Rui Choy and Dianne J. Watters. Neurodegeneration in ataxia‐telangiectasia: multiple roles of atm kinase in cellular homeostasis. Developmental Dynamics, Jan 2018. URL: https://doi.org/10.1002/dvdy.24522, doi:10.1002/dvdy.24522. This article has 90 citations and is from a peer-reviewed journal.
5. (east2024quantitativeproteomicmass pages 1-3): Michael P. East, Robert W. Sprung, Denis O. Okumu, J. Felix Olivares-Quintero, Chinmaya U. Joisa, Xin Chen, Qiang Zhang, Petra Erdmann-Gilmore, Yiling Mi, Noah Sciaky, James P. Malone, Sonam Bhatia, Ian C. McCabe, Yi Xu, Matthew D. Sutcliffe, Jingqin Luo, Patricia A. Spears, Charles M. Perou, H. Shelton Earp, Lisa A. Carey, Jen Jen Yeh, David L. Spector, Shawn M. Gomez, Philip M. Spanheimer, R. Reid Townsend, and Gary L. Johnson. Quantitative proteomic mass spectrometry of protein kinases to determine dynamic heterogeneity of the human kinome. BioRxiv, Oct 2024. URL: https://doi.org/10.1101/2024.10.04.614143, doi:10.1101/2024.10.04.614143. This article has 2 citations.
6. (east2024quantitativeproteomicmass pages 11-13): Michael P. East, Robert W. Sprung, Denis O. Okumu, J. Felix Olivares-Quintero, Chinmaya U. Joisa, Xin Chen, Qiang Zhang, Petra Erdmann-Gilmore, Yiling Mi, Noah Sciaky, James P. Malone, Sonam Bhatia, Ian C. McCabe, Yi Xu, Matthew D. Sutcliffe, Jingqin Luo, Patricia A. Spears, Charles M. Perou, H. Shelton Earp, Lisa A. Carey, Jen Jen Yeh, David L. Spector, Shawn M. Gomez, Philip M. Spanheimer, R. Reid Townsend, and Gary L. Johnson. Quantitative proteomic mass spectrometry of protein kinases to determine dynamic heterogeneity of the human kinome. BioRxiv, Oct 2024. URL: https://doi.org/10.1101/2024.10.04.614143, doi:10.1101/2024.10.04.614143. This article has 2 citations.
7. (east2024quantitativeproteomicmass pages 13-15): Michael P. East, Robert W. Sprung, Denis O. Okumu, J. Felix Olivares-Quintero, Chinmaya U. Joisa, Xin Chen, Qiang Zhang, Petra Erdmann-Gilmore, Yiling Mi, Noah Sciaky, James P. Malone, Sonam Bhatia, Ian C. McCabe, Yi Xu, Matthew D. Sutcliffe, Jingqin Luo, Patricia A. Spears, Charles M. Perou, H. Shelton Earp, Lisa A. Carey, Jen Jen Yeh, David L. Spector, Shawn M. Gomez, Philip M. Spanheimer, R. Reid Townsend, and Gary L. Johnson. Quantitative proteomic mass spectrometry of protein kinases to determine dynamic heterogeneity of the human kinome. BioRxiv, Oct 2024. URL: https://doi.org/10.1101/2024.10.04.614143, doi:10.1101/2024.10.04.614143. This article has 2 citations.
8. (garcia2022targetingtheatm pages 1-2): María E. Guerra García, David G. Kirsch, and Zachary J. Reitman. Targeting the atm kinase to enhance the efficacy of radiotherapy and outcomes for cancer patients. Seminars in Radiation Oncology, 32:3-14, Jan 2022. URL: https://doi.org/10.1016/j.semradonc.2021.09.008, doi:10.1016/j.semradonc.2021.09.008. This article has 35 citations and is from a peer-reviewed journal.
9. (higgins2023sarscov2hijacksp38βmapk11 pages 21-23): 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.
10. (hunter2015theeukaryoticprotein pages 1-3): Tony Hunter and Gerard Manning. The eukaryotic protein kinase superfamily and the emergence of receptor tyrosine kinases. Receptor Tyrosine Kinases: Structure, Functions and Role in Human Disease, pages 1-15, Oct 2015. URL: https://doi.org/10.1007/978-1-4939-2053-2\_1, doi:10.1007/978-1-4939-2053-2\_1. This article has 6 citations.
11. (hunter2015theeukaryoticprotein pages 3-6): Tony Hunter and Gerard Manning. The eukaryotic protein kinase superfamily and the emergence of receptor tyrosine kinases. Receptor Tyrosine Kinases: Structure, Functions and Role in Human Disease, pages 1-15, Oct 2015. URL: https://doi.org/10.1007/978-1-4939-2053-2\_1, doi:10.1007/978-1-4939-2053-2\_1. This article has 6 citations.
12. (jha2025deeplearningcoupledproximity pages 20-22): Kanchan Jha, Daichi Shonai, Aditya Parekh, Akiyoshi Uezu, Tomoyuki Fujiyama, Hikari Yamamoto, Pooja Parameswaran, Masashi Yanagisawa, Rohit Singh, and Scott H. Soderling. Deep learning-coupled proximity proteomics to deconvolve kinase signaling in vivo. BioRxiv, Apr 2025. URL: https://doi.org/10.1101/2025.04.27.650849, doi:10.1101/2025.04.27.650849. This article has 0 citations.
13. (jha2025deeplearningcoupledproximity pages 22-24): Kanchan Jha, Daichi Shonai, Aditya Parekh, Akiyoshi Uezu, Tomoyuki Fujiyama, Hikari Yamamoto, Pooja Parameswaran, Masashi Yanagisawa, Rohit Singh, and Scott H. Soderling. Deep learning-coupled proximity proteomics to deconvolve kinase signaling in vivo. BioRxiv, Apr 2025. URL: https://doi.org/10.1101/2025.04.27.650849, doi:10.1101/2025.04.27.650849. This article has 0 citations.
14. (jha2025deeplearningcoupledproximity pages 24-26): Kanchan Jha, Daichi Shonai, Aditya Parekh, Akiyoshi Uezu, Tomoyuki Fujiyama, Hikari Yamamoto, Pooja Parameswaran, Masashi Yanagisawa, Rohit Singh, and Scott H. Soderling. Deep learning-coupled proximity proteomics to deconvolve kinase signaling in vivo. BioRxiv, Apr 2025. URL: https://doi.org/10.1101/2025.04.27.650849, doi:10.1101/2025.04.27.650849. This article has 0 citations.
15. (jiang2024illuminatingthedark pages 7-10): Wen Jiang, Eric J. Jaehnig, Yuxing Liao, Tomer M. Yaron-Barir, Jared L. Johnson, Lewis C. Cantley, and Bing Zhang. Illuminating the dark cancer phosphoproteome through a machine-learned co-regulation map of 26,280 phosphosites. BioRxiv, Mar 2024. URL: https://doi.org/10.1101/2024.03.19.585786, doi:10.1101/2024.03.19.585786. This article has 1 citations.
16. (johnson2023anatlasof pages 1-2): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
17. (johnson2023anatlasof pages 3-4): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
18. (johnson2023anatlasof pages 4-5): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
19. (johnson2023anatlasof pages 6-7): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
20. (johnson2023anatlasof pages 7-7): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
21. (johnson2023anatlasof pages 9-10): Jared L. Johnson, Tomer M. Yaron, Emily M. Huntsman, Alexander Kerelsky, Junho Song, Amit Regev, Ting-Yu Lin, Katarina Liberatore, Daniel M. Cizin, Benjamin M. Cohen, Neil Vasan, Yilun Ma, Konstantin Krismer, Jaylissa Torres Robles, Bert van de Kooij, Anne E. van Vlimmeren, Nicole Andrée-Busch, Norbert F. Käufer, Maxim V. Dorovkov, Alexey G. Ryazanov, Yuichiro Takagi, Edward R. Kastenhuber, Marcus D. Goncalves, Benjamin D. Hopkins, Olivier Elemento, Dylan J. Taatjes, Alexandre Maucuer, Akio Yamashita, Alexei Degterev, Mohamed Uduman, Jingyi Lu, Sean D. Landry, Bin Zhang, Ian Cossentino, Rune Linding, John Blenis, Peter V. Hornbeck, Benjamin E. Turk, Michael B. Yaffe, and Lewis C. Cantley. An atlas of substrate specificities for the human serine/threonine kinome. Nature, 613:759-766, Jan 2023. URL: https://doi.org/10.1038/s41586-022-05575-3, doi:10.1038/s41586-022-05575-3. This article has 416 citations and is from a highest quality peer-reviewed journal.
22. (kalyuzhnyy2025applyingaconservationbased pages 24-26): Anton Kalyuzhnyy, Patrick A Eyers, Claire E Eyers, Eric W Deutsch, and Andrew R Jones. Applying a conservation-based approach for predicting novel phosphorylation sites in eukaryotes and evaluating their functional relevance. BioRxiv, Jan 2025. URL: https://doi.org/10.1101/2025.01.09.632054, doi:10.1101/2025.01.09.632054. This article has 0 citations.
23. (maeda2025detectingproteinhigherorder pages 27-28): Asato Maeda, Kosuke Ogata, and Yasushi Ishihama. Detecting protein higher-order structural changes using kinase as a phospho-labeler. BioRxiv, May 2025. URL: https://doi.org/10.1101/2025.05.07.652599, doi:10.1101/2025.05.07.652599. This article has 0 citations.
24. (mcskimming2017classifyingkinaseconformations pages 14-15): Daniel Ian McSkimming, Khaled Rasheed, and Natarajan Kannan. Classifying kinase conformations using a machine learning approach. BMC Bioinformatics, Feb 2017. URL: https://doi.org/10.1186/s12859-017-1506-2, doi:10.1186/s12859-017-1506-2. This article has 45 citations and is from a peer-reviewed journal.
25. (modi2019astructurallyvalidated pages 22-26): Vivek Modi and Roland L. Dunbrack. A structurally validated sequence alignment of all 497 typical human protein kinase domains. bioRxiv, Sep 2019. URL: https://doi.org/10.1101/776740, doi:10.1101/776740. This article has 8 citations.
26. (modi2019astructurallyvalidated pages 26-29): Vivek Modi and Roland L. Dunbrack. A structurally validated sequence alignment of all 497 typical human protein kinase domains. bioRxiv, Sep 2019. URL: https://doi.org/10.1101/776740, doi:10.1101/776740. This article has 8 citations.
27. (modi2019astructurallyvalidated pages 29-32): Vivek Modi and Roland L. Dunbrack. A structurally validated sequence alignment of all 497 typical human protein kinase domains. bioRxiv, Sep 2019. URL: https://doi.org/10.1101/776740, doi:10.1101/776740. This article has 8 citations.
28. (modi2019astructurallyvalidated pages 32-34): Vivek Modi and Roland L. Dunbrack. A structurally validated sequence alignment of all 497 typical human protein kinase domains. bioRxiv, Sep 2019. URL: https://doi.org/10.1101/776740, doi:10.1101/776740. This article has 8 citations.
29. (modi2019astructurallyvalidatedmultiple pages 12-12): Vivek Modi and Roland L. Dunbrack. A structurally-validated multiple sequence alignment of 497 human protein kinase domains. Scientific Reports, Dec 2019. URL: https://doi.org/10.1038/s41598-019-56499-4, doi:10.1038/s41598-019-56499-4. This article has 115 citations and is from a poor quality or predatory journal.
30. (modi2019astructurallyvalidatedmultiple pages 12-13): Vivek Modi and Roland L. Dunbrack. A structurally-validated multiple sequence alignment of 497 human protein kinase domains. Scientific Reports, Dec 2019. URL: https://doi.org/10.1038/s41598-019-56499-4, doi:10.1038/s41598-019-56499-4. This article has 115 citations and is from a poor quality or predatory journal.
31. (modi2019astructurallyvalidatedmultiple pages 13-14): Vivek Modi and Roland L. Dunbrack. A structurally-validated multiple sequence alignment of 497 human protein kinase domains. Scientific Reports, Dec 2019. URL: https://doi.org/10.1038/s41598-019-56499-4, doi:10.1038/s41598-019-56499-4. This article has 115 citations and is from a poor quality or predatory journal.
32. (modi2019astructurallyvalidatedmultiple pages 14-15): Vivek Modi and Roland L. Dunbrack. A structurally-validated multiple sequence alignment of 497 human protein kinase domains. Scientific Reports, Dec 2019. URL: https://doi.org/10.1038/s41598-019-56499-4, doi:10.1038/s41598-019-56499-4. This article has 115 citations and is from a poor quality or predatory journal.
33. (modi2019astructurallyvalidatedmultiple pages 5-7): Vivek Modi and Roland L. Dunbrack. A structurally-validated multiple sequence alignment of 497 human protein kinase domains. Scientific Reports, Dec 2019. URL: https://doi.org/10.1038/s41598-019-56499-4, doi:10.1038/s41598-019-56499-4. This article has 115 citations and is from a poor quality or predatory journal.
34. (moret2020aresourcefor pages 1-4): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
35. (moret2020aresourcefor pages 13-17): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
36. (moret2020aresourcefor pages 29-33): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
37. (moret2020aresourcefor pages 33-36): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
38. (moret2020aresourcefor pages 39-43): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
39. (moret2020aresourcefor pages 4-7): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
40. (moret2020aresourcefor pages 7-10): Nienke Moret, Changchang Liu, Benjamin M. Gyori, John A. Bachman, Albert Steppi, Clemens Hug, Rahil Taujale, Liang-Chin Huang, Matthew E. Berginski, Shawn M. Gomez, Natarajan Kannan, and Peter K. Sorger. A resource for exploring the understudied human kinome for research and therapeutic opportunities. BioRxiv, Apr 2020. URL: https://doi.org/10.1101/2020.04.02.022277, doi:10.1101/2020.04.02.022277. This article has 28 citations.
41. (mullerdott2025fromactivityinference pages 145-146): S Müller-Dott. From activity inference to multi-omics network contextualization: deciphering cellular signaling and disease mechanisms. Unknown journal, 2025.
42. (mullerdott2025fromactivityinference pages 48-52): S Müller-Dott. From activity inference to multi-omics network contextualization: deciphering cellular signaling and disease mechanisms. Unknown journal, 2025.
43. (murray2019identifyingnoveltherapeutic pages 465-467): H Murray. Identifying novel therapeutic targets for the treatment of acute myeloid leukaemia. Unknown journal, 2019.
44. (oruganty2016identificationandclassification pages 12-13): Krishnadev Oruganty, Eric E. Talevich, Andrew F. Neuwald, and Natarajan Kannan. Identification and classification of small molecule kinases: insights into substrate recognition and specificity. BMC Evolutionary Biology, Jan 2016. URL: https://doi.org/10.1186/s12862-015-0576-x, doi:10.1186/s12862-015-0576-x. This article has 25 citations.