## 1. Phylogeny

Tyrosine‐protein kinase Lyn is a member of the evolutionarily ancient Src family of non‐receptor tyrosine kinases (SFKs). This family is highly conserved across vertebrates and can be traced back to early metazoan ancestors, which indicates that their modular architecture and signaling functions were fundamental for the evolution of multicellularity (bhanumathy2021proteintyrosinekinases pages 1-2). Within the SFK group, Lyn is closely related to other paralogous kinases such as Src, Fyn, Lck, and Yes. Although these kinases share the hallmark SH4, SH3, SH2, and catalytic domains, Lyn has evolved unique regulatory characteristics that reflect its preferential expression in the hematopoietic and immune cell lineages. This specialization has permitted Lyn to perform dual roles—acting either as a positive or negative regulator—in immune receptor signaling cascades. Studies indicate that the catalytic kinase domain of Lyn is highly conserved among mammals, and orthologs of Lyn exist in all major vertebrate species, underlining its fundamental contribution to immune surveillance and cellular homeostasis (weerawarna2023lynkinasestructure pages 1-3, brodie2018lynlupusand pages 2-3). In addition, gene duplication events that expanded the Src family set have led to diversification in substrate recognition and regulation among the family members. The evolution of distinct regulatory phosphorylation sites and domain interfaces within Lyn underscores how subtle variations in sequence and structure have been selected to meet the specific demands of B-cell receptor (BCR) signaling and innate immune pathways (sakkiah2017overviewofthe pages 2-3, santos2016paralogspecificpatternsof pages 18-19). This evolutionary divergence is critical for enabling the fine-tuned balance between activation and inhibition required for precise immunological responses.

## 2. Reaction Catalyzed

Lyn catalyzes a classic phosphotransfer reaction characteristic of protein tyrosine kinases. In this reaction, Lyn facilitates the transfer of the γ-phosphate group from adenosine triphosphate (ATP) to the hydroxyl group of a tyrosine residue on a substrate protein. The overall reaction can be formally written as:  
 ATP + protein–tyrosine → ADP + protein–phosphotyrosine + H⁺  
This reaction occurs within the well-defined catalytic cleft of Lyn’s kinase (SH1) domain. A conserved glycine-rich loop (often referred to as the P-loop) binds ATP and helps to position it such that the γ-phosphate is precisely oriented for nucleophilic attack by the tyrosine hydroxyl group on the substrate (bhanumathy2021proteintyrosinekinases pages 1-2). The phosphorylation event not only induces conformational changes in the modified substrate, thereby altering its enzymatic activity, localization, or protein–protein interactions, but also generates specific binding sites for downstream adapter proteins containing domains such as SH2 or phosphotyrosine-binding (PTB) domains (corwin2016decipheringhumancytoplasmic pages 146-149). Thus, the kinase reaction carried out by Lyn is central to propagating intracellular signaling cascades in response to extracellular cues.

## 3. Cofactor Requirements

The enzymatic activity of Lyn is heavily dependent on the availability of the divalent metal ion magnesium (Mg²⁺). Magnesium ions bind to ATP and facilitate the correct alignment of its phosphate groups within the active site of the kinase, thereby stabilizing the transition state during phosphotransfer (marholz2018insilicodesign pages 5-5). Although in vitro experiments occasionally use manganese (Mn²⁺) as an alternative cofactor, Mg²⁺ is the physiologically relevant cofactor required for efficient catalysis under cellular conditions. Beyond metal ion coordination, Lyn’s activity also critically depends on its membrane association. This is achieved by dual lipid modifications—myristoylation and palmitoylation—on its N-terminal SH4 domain. These post-translational modifications tether Lyn to the plasma membrane, concentrating it in lipid raft regions where immune receptors are localized and thereby enhancing the efficiency of substrate phosphorylation (weerawarna2023lynkinasestructure pages 3-4).

## 4. Substrate Specificity

Lyn exhibits broad substrate specificity, wherein it phosphorylates a diverse spectrum of proteins that are central to immune receptor signaling and hematopoietic regulation. Among its best-characterized substrates are components and associated molecules of the B-cell receptor (BCR) complex, including CD79A and CD79B; these proteins undergo phosphorylation upon antigen engagement, which is essential for the subsequent initiation of signaling cascades in B lymphocytes (corwin2016decipheringhumancytoplasmic pages 146-149, brodie2018lynlupusand pages 2-3). In addition to BCR components, Lyn phosphorylates co‐receptors such as CD5, CD19, and CD22, thereby fine-tuning both the activation and subsequent down-regulation phases of B cell responses.  
Lyn’s substrate repertoire also includes several signaling and adaptor molecules. For instance, it phosphorylates Bruton’s tyrosine kinase (BTK), Spleen tyrosine kinase (SYK), and TEC kinase, wherein such modifications play pivotal roles in modulating cell proliferation, survival, and differentiation (jin2015proteintyrosinephosphorylation pages 124-129, deng2014globalanalysisof pages 10-13). A distinctive functional attribute of Lyn is its role in the negative regulation of immune signaling. This is achieved via the phosphorylation of immunoreceptor tyrosine-based inhibitory motifs (ITIMs) on various receptor proteins. Upon phosphorylation, these ITIMs serve as docking sites for inhibitory phosphatases such as PTPN6 (SHP-1), PTPN11 (SHP-2), and INPP5D (SHIP-1), which in turn attenuate the signaling cascades to prevent hyperactivation of immune responses (arrington2019identificationofthe pages 12-12, gocek2014nonreceptorproteintyrosine pages 1-2).  
Although Lyn does not adhere to a single strict consensus sequence for its substrates, its recognition largely depends on the combined selectivity of its kinase domain, along with targeting provided by its SH2 and SH3 domains. These domains help to guide Lyn to specific signaling complexes where substrates often present phosphorylation-prone motifs enriched for proline residues in adjacent regions or specific phosphotyrosine contexts (jin2015tyrosinephosphorylationof pages 22-25, santos2016paralogspecificpatternsof pages 18-19). Collectively, this broad and multifaceted substrate specificity enables Lyn to integrate signals from a variety of immune receptors and to contribute dynamically to both the propagation and attenuation of signal transduction.

## 5. Structure

Lyn kinase exhibits a modular structure that is a hallmark of the Src family kinases. Its primary structure can be divided into several distinct domains, each conferring specific functional properties.  
At the very N-terminus lies the SH4 domain, which is relatively short (approximately 16 residues) yet critically important for subcellular localization. This domain harbors sequences that direct dual lipid modifications—myristoylation typically on an N-terminal glycine and subsequent palmitoylation on neighboring cysteine residues. These modifications serve to anchor Lyn to the plasma membrane, particularly to the specialized microdomains known as lipid rafts, where many immune receptors and signaling complexes are found (weerawarna2023lynkinasestructure pages 3-4, marholz2018insilicodesign pages 5-5).  
Following the SH4 domain is the unique domain. This region is characterized by a relatively low degree of sequence conservation among SFKs, which allows for paralog-specific functions and interactions. Variations in the length and sequence of the unique domain, as exemplified by the differences between LynA and LynB isoforms, are thought to contribute to diversified signaling roles, including influences on DNA replication and other nuclear functions in some contexts.  
Immediately succeeding the unique domain is the SH3 domain. Typically comprising 60–70 amino acids, the SH3 domain is responsible for binding proline-rich motifs, usually adopting a polyproline type II helix structure when associated with its ligands. This domain is not only involved in mediating interactions with external substrates and adaptor proteins, but also contributes to autoinhibitory intramolecular interactions that maintain Lyn in a low-activity state under resting conditions (weerawarna2023lynkinasestructure pages 12-13, huang2016directedevolutionof pages 2-4).  
Adjacent to the SH3 domain is the SH2 domain, which specifically recognizes phosphorylated tyrosine residues present in certain sequence contexts. The SH2 domain is essential for substrate recruitment; it binds to phosphotyrosine sites either on target proteins or even to phosphorylated residues on Lyn itself, thereby reinforcing conformational states that regulate kinase activity (gocek2014nonreceptorproteintyrosine pages 1-2, weerawarna2023lynkinasestructure pages 12-13).  
Central to Lyn’s function is the catalytic kinase or SH1 domain. This domain exhibits the classic bilobal architecture seen in many protein kinases: a smaller N-terminal lobe composed mainly of beta sheets (including the glycine-rich loop that facilitates ATP binding) and a larger C-terminal lobe that is predominately α-helical. Key structural motifs are present in this domain, such as the DFG (Asp-Phe-Gly) motif at the beginning of the activation loop and the HRD motif, both of which are essential for catalysis. The activation loop itself, upon autophosphorylation at a key tyrosine residue (often analogous to Tyr397 in Lyn), undergoes a conformational change that is necessary for full catalytic activity (corwin2016decipheringhumancytoplasmic pages 13-16, weerawarna2023lynkinasestructure pages 4-7).  
In addition to these domains, the C-terminal tail of Lyn contains regulatory elements, including a conserved tyrosine residue whose phosphorylation is responsible for mediating the autoinhibited conformation. When this tyrosine is phosphorylated, it binds intramolecularly to the SH2 domain, thereby stabilizing an inactive kinase conformation and preventing uncontrolled substrate phosphorylation. This multi-domain configuration, combined with specific post-translational modifications, allows Lyn to tightly regulate its activity in response to signaling events.

## 6. Regulation

The regulation of Lyn kinase is multifaceted, involving several layers of control that ensure its activity is precisely modulated according to cellular context. At the core of Lyn regulation are two key phosphorylation events that function in an antagonistic manner.  
Autophosphorylation within the activation loop of the catalytic domain—for example, phosphorylation at a tyrosine residue analogous to Tyr397—induces a conformational shift that stabilizes the active form of Lyn, thereby enhancing its catalytic efficiency and substrate turnover (weerawarna2023lynkinasestructure pages 14-16). Conversely, a conserved tyrosine residue located in the C-terminal tail undergoes phosphorylation by dedicated kinases such as C-terminal Src kinase (Csk) or its homologs. Phosphorylation at this residue promotes an intramolecular interaction with Lyn’s own SH2 domain, resulting in a closed, autoinhibited conformation that significantly diminishes kinase activity (tsantikos2014roleofthe pages 14-15, corwin2016decipheringhumancytoplasmic pages 16-18).  
Beyond these primary phosphorylation events, Lyn’s regulatory framework is further reinforced by the roles of its SH3 and SH2 domains. Under basal conditions, these domains engage in intramolecular interactions that help maintain Lyn in a repressed state by sequestering structural elements necessary for catalytic activity. For instance, phosphorylation within the SH2 domain itself (such as on Tyr194) can modulate its binding affinity for external phosphotyrosine-containing substrates and affect its participation in regulatory autoinhibitory complexes (jin2015proteintyrosinephosphorylation pages 152-154).  
Another critical aspect of Lyn regulation is its spatial localization. The dual acylation of the N-terminal SH4 domain anchors Lyn to the plasma membrane, particularly to lipid raft microdomains where immune receptors reside. This localization not only ensures the enzyme encounters its intended substrates promptly upon receptor stimulation but also restricts its activity to specific subcellular compartments, thereby minimizing aberrant phosphorylation events (taft2017ayeastbasedassay pages 26-30, tsantikos2014roleofthe pages 16-16).  
Collectively, these regulatory mechanisms establish Lyn as a molecular switch that can rapidly toggle between active and inactive states. This dynamic regulation is essential in immune cells, where precise control over signaling thresholds is necessary to prevent inappropriate activation or suppression, both of which can lead to pathological conditions such as autoimmunity or uncontrolled cell growth.

## 7. Function

Lyn kinase serves as a central integrator in multiple signaling pathways predominantly within hematopoietic and immune cells. One of its key roles is in the regulation of B-cell receptor (BCR) signaling. Upon antigen engagement, Lyn phosphorylates critical components of the BCR complex—including CD79A and CD79B—and co-receptors such as CD5, CD19, and CD22. These phosphorylation events initiate a cascade that leads to B-cell activation, promoting processes such as differentiation, proliferation, and survival while later contributing to the down-regulation of these signals to maintain immune self-tolerance (arrington2019identificationofthe pages 12-12, brodie2018lynlupusand pages 2-3).  
In addition to its role in B cells, Lyn is crucial in modulating the signaling of Fc receptors. By phosphorylating receptors like FCER1, FCGR1A, and FCGR2, Lyn is able to directly affect mast cell degranulation, macrophage activation, and the inflammatory response to bacterial lipopolysaccharide. This regulation is pivotal during immune responses to pathogens, where a rapid yet controlled inflammatory response is necessary (bhanumathy2021proteintyrosinekinases pages 1-2, corwin2016decipheringhumancytoplasmic pages 26-29).  
Furthermore, Lyn influences signaling pathways initiated by Toll-like receptors (TLR2 and TLR4) as well as a variety of cytokine and growth factor receptors, including EPOR, KIT, MPL, CXCR4, and receptors for IL3, IL5, and CSF2. In these settings, Lyn mediates phosphorylation events that trigger kinase cascades such as the PI3K/AKT, MAP kinase, and STAT5 pathways. These cascades are critical for processes such as cellular proliferation, survival, migration, adhesion, and even differentiation in both hematopoietic progenitors and mature immune cells (deng2014globalanalysisof pages 10-13).  
A particularly noteworthy feature of Lyn’s function is its dual capacity to both activate and inhibit signaling pathways. In certain contexts, Lyn phosphorylates immunoreceptor tyrosine-based inhibitory motifs (ITIMs) on receptor proteins to recruit inhibitory phosphatases like SHP-1 (PTPN6), SHP-2 (PTPN11), and SHIP-1 (INPP5D). This negative feedback serves to attenuate signaling events once an immune response has been initiated, ensuring that responses do not become excessive and cause autoimmunity or chronic inflammation (jin2015proteintyrosinephosphorylation pages 124-129, arrington2019identificationofthe pages 12-12).  
In additional contexts, Lyn has been implicated in oncogenic processes; for example, its substrate repertoire includes the BCR-ABL fusion protein, whose phosphorylation by Lyn is associated with leukemogenesis. Overexpression or dysregulation of Lyn has also been observed in various solid tumors, including breast, prostate, and colorectal carcinomas, where it may drive aberrant proliferation and survival signals.  
Thus, Lyn is not only fundamental for the initiation and modulation of adaptive immune responses but also plays a regulatory role in innate immunity, cell adhesion, and even in the malignant transformation of cells. Its balanced activity, ensured by sophisticated regulatory mechanisms, is crucial for maintaining immune homeostasis and normal cellular functions across diverse cell types.

## 8. Other Comments

Due to its critical regulatory roles in both positive and negative signaling pathways, Lyn has emerged as an attractive therapeutic target in a variety of pathological conditions. Aberrant activation or overexpression of Lyn is implicated in hematological malignancies, autoimmune disorders, and inflammatory diseases. Recent efforts in drug discovery have focused on identifying small-molecule inhibitors that selectively modulate Lyn activity. DNA-encoded library screening approaches, for instance, have yielded novel compounds that target either the ATP-binding pocket or distinct allosteric sites on Lyn. Such inhibitors are in preclinical development, with some progressing to clinical evaluation in attempts to mitigate conditions associated with Lyn hyperactivity (sun2022targetingproteinproteininteractions pages 18-24, sun2022targetingproteinproteininteractions pages 52-56).  
Structural studies of Lyn, although somewhat limited due to the preponderance of crystal structures capturing the kinase in its active conformation, continue to shed light on the dynamic nature of its regulatory domain interactions. Current research aims to solve additional structures—particularly of the inactive conformation—to facilitate structure-based drug design. The high degree of conservation within the ATP-binding site across Src family kinases, however, poses a significant challenge, necessitating strategies that exploit subtle differences in domain interfaces or allosteric regions unique to Lyn (taft2017ayeastbasedassay pages 26-30, tsantikos2014roleofthe pages 16-16).  
Moreover, there is an ongoing interest in understanding the contributions of Lyn’s intrinsically disordered regions, such as the unique domain, which may undergo regulatory phosphorylation that influences functions beyond canonical membrane-associated signaling. In some isoforms, phosphorylation events within these regions have been linked to nuclear activities, including the regulation of DNA replication (weerawarna2023lynkinasestructure pages 3-4, huang2016directedevolutionof pages 2-4).  
From a clinical perspective, mutations that either result in inadequate or excessive Lyn activity have been associated with immune dysregulation. For instance, gain-of-function variants may lead to aberrant activation and early-onset autoinflammatory syndromes, while loss-of-function mutations can result in compromised immune responses and increased susceptibility to infections or autoimmunity (tsantikos2014roleofthe pages 14-15, brodie2018lynlupusand pages 2-3). In addition, the participation of Lyn in the phosphorylation of oncogenic proteins—such as the BCR-ABL fusion protein—underscores its potential as a target in leukemia and other cancers.  
Finally, combination therapy strategies that target Lyn in conjunction with other components of the immune signaling machinery are under investigation. Such approaches could enhance therapeutic efficacy while reducing the potential for drug resistance associated with monotherapy. In summary, Lyn kinase is a multifaceted enzyme whose regulation and function make it both a critical player in normal cellular physiology and a promising target for the development of novel therapeutic agents in immune-related and oncogenic diseases.

## 9. References

arrington2019identificationofthe pages 12-12; bhanumathy2021proteintyrosinekinases pages 1-2; brodie2018lynlupusand pages 2-3; corwin2016decipheringhumancytoplasmic pages 13-16; corwin2016decipheringhumancytoplasmic pages 16-18; corwin2016decipheringhumancytoplasmic pages 26-29; deng2014globalanalysisof pages 10-13; gocek2014nonreceptorproteintyrosine pages 1-2; huang2016directedevolutionof pages 2-4; jin2015proteintyrosinephosphorylation pages 124-129; jin2015proteintyrosinephosphorylation pages 152-154; jin2015tyrosinephosphorylationof pages 22-25; marholz2018insilicodesign pages 5-5; sakkiah2017overviewofthe pages 2-3; santos2016paralogspecificpatternsof pages 18-19; sun2022targetingproteinproteininteractions pages 18-24; sun2022targetingproteinproteininteractions pages 52-56; taft2017ayeastbasedassay pages 26-30; tsantikos2014roleofthe pages 14-15; tsantikos2014roleofthe pages 16-16; weerawarna2023lynkinasestructure pages 1-3; weerawarna2023lynkinasestructure pages 12-13; weerawarna2023lynkinasestructure pages 14-16; weerawarna2023lynkinasestructure pages 3-4; weerawarna2023lynkinasestructure pages 4-7.

References

1. (arrington2019identificationofthe pages 12-12): Justine Arrington, Liang Xue, Wen-Horng Wang, Robert L. Geahlen, and W. Andy Tao. Identification of the direct substrates of the abl kinase via kinase assay linked phosphoproteomics with multiple drug treatments. Journal of Proteome Research, 18:1679-1690, Mar 2019. URL: https://doi.org/10.1021/acs.jproteome.8b00942, doi:10.1021/acs.jproteome.8b00942. This article has 14 citations and is from a peer-reviewed journal.
2. (bhanumathy2021proteintyrosinekinases pages 1-2): Kalpana K. Bhanumathy, Amrutha Balagopal, Frederick S. Vizeacoumar, Franco J. Vizeacoumar, Andrew Freywald, and Vincenzo Giambra. Protein tyrosine kinases: their roles and their targeting in leukemia. Cancers, 13:184, Jan 2021. URL: https://doi.org/10.3390/cancers13020184, doi:10.3390/cancers13020184. This article has 73 citations and is from a peer-reviewed journal.
3. (brodie2018lynlupusand pages 2-3): Erica J. Brodie, Simona Infantino, Michael S. Y. Low, and David M. Tarlinton. Lyn, lupus, and (b) lymphocytes, a lesson on the critical balance of kinase signaling in immunity. Frontiers in Immunology, Mar 2018. URL: https://doi.org/10.3389/fimmu.2018.00401, doi:10.3389/fimmu.2018.00401. This article has 63 citations and is from a peer-reviewed journal.
4. (corwin2016decipheringhumancytoplasmic pages 13-16): T Corwin. Deciphering human cytoplasmic protein tyrosine kinase phosphorylation specificity in yeast. Unknown journal, 2016.
5. (corwin2016decipheringhumancytoplasmic pages 146-149): T Corwin. Deciphering human cytoplasmic protein tyrosine kinase phosphorylation specificity in yeast. Unknown journal, 2016.
6. (corwin2016decipheringhumancytoplasmic pages 16-18): T Corwin. Deciphering human cytoplasmic protein tyrosine kinase phosphorylation specificity in yeast. Unknown journal, 2016.
7. (corwin2016decipheringhumancytoplasmic pages 26-29): T Corwin. Deciphering human cytoplasmic protein tyrosine kinase phosphorylation specificity in yeast. Unknown journal, 2016.
8. (deng2014globalanalysisof pages 10-13): Yang Deng, Nilda L. Alicea-Velázquez, Ludovic Bannwarth, Soili I. Lehtonen, Titus J. Boggon, Heung-Chin Cheng, Vesa P. Hytönen, and Benjamin E. Turk. Global analysis of human nonreceptor tyrosine kinase specificity using high-density peptide microarrays. Journal of Proteome Research, 13:4339-4346, Aug 2014. URL: https://doi.org/10.1021/pr500503q, doi:10.1021/pr500503q. This article has 52 citations and is from a peer-reviewed journal.
9. (gocek2014nonreceptorproteintyrosine pages 1-2): Elzbieta Gocek, Anargyros N. Moulas, and George P. Studzinski. Non-receptor protein tyrosine kinases signaling pathways in normal and cancer cells. Critical Reviews in Clinical Laboratory Sciences, 51:125-137, May 2014. URL: https://doi.org/10.3109/10408363.2013.874403, doi:10.3109/10408363.2013.874403. This article has 154 citations and is from a peer-reviewed journal.
10. (huang2016directedevolutionof pages 2-4): Renhua Huang, Pete Fang, Zengping Hao, and Brian K. Kay. Directed evolution of a highly specific fn3 monobody to the sh3 domain of human lyn tyrosine kinase. PLOS ONE, 11:e0145872, Jan 2016. URL: https://doi.org/10.1371/journal.pone.0145872, doi:10.1371/journal.pone.0145872. This article has 12 citations and is from a peer-reviewed journal.
11. (jin2015proteintyrosinephosphorylation pages 124-129): LL Jin. Protein tyrosine phosphorylation in haematopoietic cancers and the functional significance of phospho-lyn sh2 domain. Unknown journal, 2015.
12. (jin2015proteintyrosinephosphorylation pages 152-154): LL Jin. Protein tyrosine phosphorylation in haematopoietic cancers and the functional significance of phospho-lyn sh2 domain. Unknown journal, 2015.
13. (jin2015tyrosinephosphorylationof pages 22-25): Lily L. Jin, Leanne E. Wybenga-Groot, Jiefei Tong, Paul Taylor, Mark D. Minden, Suzanne Trudel, C. Jane McGlade, and Michael F. Moran. Tyrosine phosphorylation of the lyn src homology 2 (sh2) domain modulates its binding affinity and specificity\*. Molecular & Cellular Proteomics, 14:695-706, Mar 2015. URL: https://doi.org/10.1074/mcp.m114.044404, doi:10.1074/mcp.m114.044404. This article has 51 citations.
14. (marholz2018insilicodesign pages 5-5): Laura J. Marholz, Nicholas A. Zeringo, Hua Jane Lou, Benjamin E. Turk, and Laurie L. Parker. in silico design and in vitro characterization of universal tyrosine kinase peptide substrates. Biochemistry, 57:1847-1851, Mar 2018. URL: https://doi.org/10.1021/acs.biochem.8b00044, doi:10.1021/acs.biochem.8b00044. This article has 10 citations and is from a peer-reviewed journal.
15. (sakkiah2017overviewofthe pages 2-3): Sugunadevi Sakkiah, Guang Ping Cao, Staya P. Gupta, and Keun Woo Lee. Overview of the structure and function of protein kinases. Current Enzyme Inhibition, 13:81-88, Jul 2017. URL: https://doi.org/10.2174/1573408013666161226155608, doi:10.2174/1573408013666161226155608. This article has 13 citations and is from a peer-reviewed journal.
16. (santos2016paralogspecificpatternsof pages 18-19): Helena G. Dos Santos and Jessica Siltberg-Liberles. Paralog-specific patterns of structural disorder and phosphorylation in the vertebrate sh3–sh2–tyrosine kinase protein family. Genome Biology and Evolution, 8:2806-2825, Aug 2016. URL: https://doi.org/10.1093/gbe/evw194, doi:10.1093/gbe/evw194. This article has 9 citations and is from a domain leading peer-reviewed journal.
17. (sun2022targetingproteinproteininteractions pages 18-24): Y Sun. Targeting protein-protein interactions in kinase domains with dna-encoded library approaches for therapeutics and diagnostics. Unknown journal, 2022.
18. (sun2022targetingproteinproteininteractions pages 52-56): Y Sun. Targeting protein-protein interactions in kinase domains with dna-encoded library approaches for therapeutics and diagnostics. Unknown journal, 2022.
19. (taft2017ayeastbasedassay pages 26-30): Joseph M. Taft. A yeast-based assay for protein tyrosine kinase substrate specificity and inhibitor resistance. Unknown journal, Dec 2017. URL: https://doi.org/10.26153/tsw/7450, doi:10.26153/tsw/7450. This article has 0 citations.
20. (tsantikos2014roleofthe pages 14-15): E. Tsantikos, Timothy A. Gottschalk, Mhairi J. Maxwell, and M. Hibbs. Role of the lyn tyrosine kinase in the development of autoimmune disease. International Journal of Clinical Rheumatology, 9:519-535, Nov 2014. URL: https://doi.org/10.2217/ijr.14.44, doi:10.2217/ijr.14.44. This article has 13 citations and is from a poor quality or predatory journal.
21. (tsantikos2014roleofthe pages 16-16): E. Tsantikos, Timothy A. Gottschalk, Mhairi J. Maxwell, and M. Hibbs. Role of the lyn tyrosine kinase in the development of autoimmune disease. International Journal of Clinical Rheumatology, 9:519-535, Nov 2014. URL: https://doi.org/10.2217/ijr.14.44, doi:10.2217/ijr.14.44. This article has 13 citations and is from a poor quality or predatory journal.
22. (weerawarna2023lynkinasestructure pages 1-3): Pathum M. Weerawarna and Timothy I. Richardson. Lyn kinase structure, regulation, and involvement in neurodegenerative diseases: a mini review. Kinases and Phosphatases, 1:23-38, Jan 2023. URL: https://doi.org/10.3390/kinasesphosphatases1010004, doi:10.3390/kinasesphosphatases1010004. This article has 8 citations.
23. (weerawarna2023lynkinasestructure pages 12-13): Pathum M. Weerawarna and Timothy I. Richardson. Lyn kinase structure, regulation, and involvement in neurodegenerative diseases: a mini review. Kinases and Phosphatases, 1:23-38, Jan 2023. URL: https://doi.org/10.3390/kinasesphosphatases1010004, doi:10.3390/kinasesphosphatases1010004. This article has 8 citations.
24. (weerawarna2023lynkinasestructure pages 14-16): Pathum M. Weerawarna and Timothy I. Richardson. Lyn kinase structure, regulation, and involvement in neurodegenerative diseases: a mini review. Kinases and Phosphatases, 1:23-38, Jan 2023. URL: https://doi.org/10.3390/kinasesphosphatases1010004, doi:10.3390/kinasesphosphatases1010004. This article has 8 citations.
25. (weerawarna2023lynkinasestructure pages 3-4): Pathum M. Weerawarna and Timothy I. Richardson. Lyn kinase structure, regulation, and involvement in neurodegenerative diseases: a mini review. Kinases and Phosphatases, 1:23-38, Jan 2023. URL: https://doi.org/10.3390/kinasesphosphatases1010004, doi:10.3390/kinasesphosphatases1010004. This article has 8 citations.
26. (weerawarna2023lynkinasestructure pages 4-7): Pathum M. Weerawarna and Timothy I. Richardson. Lyn kinase structure, regulation, and involvement in neurodegenerative diseases: a mini review. Kinases and Phosphatases, 1:23-38, Jan 2023. URL: https://doi.org/10.3390/kinasesphosphatases1010004, doi:10.3390/kinasesphosphatases1010004. This article has 8 citations.