1. Phylogeny  
   Tyrosine‑protein kinase ITK (also known as ITK/TSK, EMT, Lyk) is a member of the Tec family of non‑receptor tyrosine kinases, a group that is evolutionarily distinct from the Src family of kinases. ITK is predominantly expressed in T lymphocytes and closely related to other Tec kinases such as Bruton’s tyrosine kinase (BTK), Tec, Rlk (TXK) and BMX. ITK has been identified in a wide range of vertebrates, and its conservation among mammals indicates that its gene originated early in vertebrate evolution, aligning with the evolutionary core set of kinases observed in eukaryotic organisms (ortutay2008phylogenyoftec pages 7-10, yu2009tecfamilykinases pages 23-26). This phylogenetic context places ITK within a family of kinases that share a characteristic domain architecture (including pleckstrin homology [PH], Tec homology [TH], Src homology 3 [SH3], Src homology 2 [SH2], and a kinase catalytic domain), which distinguishes it from other kinase families in the human kinome (boucheron2012theroleof pages 1-3, tarafdar2014interactionsofthe pages 82-87).
2. Reaction Catalyzed  
   ITK catalyzes the transfer of a phosphate group from ATP to a tyrosine residue on substrate proteins, thereby converting ATP into ADP and generating a phospho‐tyrosine on its target substrate. In the context of T‑cell receptor (TCR) signaling, one well‑characterized substrate is phospholipase C‑γ1 (PLCγ1); phosphorylation of PLCγ1 initiates a cascade that results in the hydrolysis of phosphatidylinositol 4,5‑bisphosphate (PIP2) into inositol trisphosphate (IP3) and diacylglycerol (DAG) (aryal2021molecularmechanismof pages 101-104, zhong2014targetinginterleukin2inducibletcell pages 5-6).
3. Cofactor Requirements  
   The kinase activity of ITK is dependent on the presence of divalent cations, with Mg²⁺ functioning as the essential cofactor for the proper binding of ATP and subsequent phosphoryl-transfer reactions. This requirement for Mg²⁺ aligns with the common cofactor dependency observed across protein kinases (kaur2012inhibitorsofinterleukin2 pages 1-2, yu2009tecfamilykinases pages 50-52).
4. Substrate Specificity  
   ITK displays substrate specificity that is consistent with its role in T‑cell receptor signaling. It primarily phosphorylates tyrosine residues on specific substrates involved in immune signaling; among these, PLCγ1 is a principal substrate, with phosphorylation events occurring on key tyrosine sites that are critical for the initiation of calcium flux and downstream signaling cascades (aryal2021molecularmechanismof pages 101-104, zhong2014targetinginterleukin2inducibletcell pages 5-6). Additionally, ITK phosphorylates adaptor proteins such as LAT and LCP2, which are necessary for the assembly of multiprotein signaling complexes following TCR engagement (boucheron2012theroleof pages 4-6, kaur2012inhibitorsofinterleukin2 pages 15-15). Although a detailed consensus motif for ITK substrates has not been explicitly defined in the available literature, the enzyme’s function is closely tied to the selective phosphorylation of tyrosine residues on proteins that orchestrate adaptive immune responses (zhong2014targetinginterleukin2inducibletcell pages 9-11).
5. Structure  
   ITK comprises a multi‑domain architecture that underpins both its catalytic functions and its regulatory interactions. The N‑terminal region includes a pleckstrin homology (PH) domain, which mediates binding to phosphoinositides such as phosphatidylinositol 3,4,5‑trisphosphate (PIP3) and facilitates membrane recruitment—a step that is indispensable for its activation (boucheron2012theroleof pages 1-3, kaur2012inhibitorsofinterleukin2 pages 4-5). Adjacent to the PH domain is the Tec homology (TH) domain that contains a BTK‑type zinc finger motif and a proline‑rich region (PRR). The PRR is functionally significant as it participates in intramolecular interactions that help maintain ITK in an autoinhibited conformation and modulate its activation upon TCR stimulation (aryal2021molecularmechanismof pages 96-101, gurung2023prolineisomerizationfrom pages 9-11). Following the TH domain, ITK harbors an SH3 domain that binds to polyproline motifs and plays a critical role in protein–protein interactions. Notably, a conserved tyrosine residue (Y180) within the SH3 domain is subject to autophosphorylation, which influences ligand binding and kinase activation (aryal2021molecularmechanismof pages 92-96). The SH2 domain, capable of binding phosphotyrosine‑containing motifs in target proteins, exhibits conformational plasticity due to a proline‐dependent cis–trans isomerization event that further regulates ITK activity (aryal2021molecularmechanismof pages 101-104, gurung2023prolineisomerizationfrom pages 9-11). Finally, the C‑terminal kinase domain (SH1) contains the canonical ATP‑binding site and catalytic loop necessary for phosphotransfer activity, with key structural elements such as the activation loop and the C‑helix playing roles in substrate engagement and catalytic efficiency (zhong2014targetinginterleukin2inducibletcell pages 11-13, kaur2012inhibitorsofinterleukin2 pages 15-15). This arrangement of domains—PH, TH, SH3, SH2, and kinase—ensures that ITK is tightly regulated both spatially and temporally within the T cell (boucheron2012theroleof pages 17-19, aryal2021molecularmechanismof pages 96-101).
6. Regulation  
   ITK is subject to complex regulatory mechanisms that involve both post‑translational modifications and dynamic intramolecular interactions. A key regulatory event occurs upon T‑cell receptor engagement when the Src family kinase Lck phosphorylates ITK at a critical tyrosine residue (Y511) in the activation loop; this phosphorylation event primes ITK for full activation through subsequent autophosphorylation events, including on the SH3 domain at Y180 (zhong2014targetinginterleukin2inducibletcell pages 11-13, aryal2021molecularmechanismof pages 92-96). The autoinhibited state of ITK is maintained by intramolecular interactions between its regulatory domains, such as the binding of the proline-rich region of the TH domain to the SH3 domain; disruption of these interactions, for example through binding of phosphoinositides to the PH domain, results in a conformational shift that releases the kinase domain for catalytic action (aryal2021molecularmechanismof pages 104-109, kaur2012inhibitorsofinterleukin2 pages 14-14). In addition, proline isomerization within the SH2 domain—specifically at residues adjacent to the phosphotyrosine binding pocket—has been shown to modulate ITK’s ligand-binding specificity and thereby impact its downstream signaling functions (gurung2023prolineisomerizationfrom pages 9-11). ITK regulation is also influenced by its interactions with adaptor proteins such as SLP‑76 and LAT, which act as scaffolds to assemble the signaling complex required for full T‑cell activation (boucheron2012theroleof pages 4-6, tarafdar2014interactionsofthe pages 82-87). These regulatory events ensure that ITK activity is tightly coupled to T‑cell receptor stimulation, preventing premature or inappropriate signaling (zhong2014targetinginterleukin2inducibletcell pages 6-8, boucheron2012theroleof pages 8-9).
7. Function  
   ITK plays an essential role in the regulation of adaptive immunity. It is highly expressed in conventional T‑cells as well as in non‑conventional natural killer T‑cells (NKT cells), where it is critical for T‑cell receptor (TCR) signaling. Upon antigen recognition by the TCR, a cascade of phosphorylation events leads to the recruitment of ITK to the plasma membrane, where Lck phosphorylates ITK to initiate its activation. Once activated, ITK phosphorylates several key substrates, including phospholipase C‑γ1 (PLCγ1), which in turn catalyzes the formation of second messengers IP3 and DAG, leading to calcium release from the endoplasmic reticulum and the activation of transcription factors such as NFAT that are essential for lymphokine production, T‑cell proliferation, and differentiation (aryal2021molecularmechanismof pages 101-104, zhong2014targetinginterleukin2inducibletcell pages 1-3). In addition, ITK phosphorylates adaptor proteins such as LAT and LCP2 (SLP‑76), which are required for the formation and stabilization of the TCR signalosome that orchestrates downstream signaling pathways (boucheron2012theroleof pages 4-6, kaur2012inhibitorsofinterleukin2 pages 15-15). ITK is also involved in the modulation of transcriptional regulators; for example, by phosphorylating TBX21 (T‑bet) at Tyr‑530, ITK mediates interactions that influence the balance between T‑helper cell subsets, particularly affecting Th2 differentiation and the expression of cytokines such as interleukin‑2 (IL‑2) (aryal2021molecularmechanismof pages 237-240, boucheron2012theroleof pages 9-11). Beyond its role in classical T‑cell signaling, ITK is essential for TCR‑mediated calcium responses in γδ T‑cells and contributes to the transcriptomic landscape of immature γδ T‑cells, further underpinning its importance in immune regulation (blomberg2009geneexpressionanalysis pages 21-25, lechner2020roleofthe pages 1-2). Collectively, ITK functions as a critical link between antigen recognition and a diverse array of intracellular signaling events that govern T‑cell activation, differentiation, and effector functions in adaptive immunity (kaur2012inhibitorsofinterleukin2 pages 2-3, boucheron2012theroleof pages 17-19).
8. Other Comments  
   Several small molecule inhibitors targeting ITK have been identified and characterized, reflecting its potential as a therapeutic target for T‑cell mediated inflammatory disorders. Among these inhibitors are classes of compounds such as benzimidazole derivatives, aminothiazoles, thiazolyl compounds, and dual inhibitors that also target kinases like RLK and PI3K (kaur2012inhibitorsofinterleukin2 pages 13-14, zhong2014targetinginterleukin2inducibletcell pages 5-6). In addition, experimental compounds like rosmarinic acid have been reported to attenuate T‑cell receptor-induced T‑cell activation in an Lck‑dependent manner, further underscoring the therapeutic promise of modulating ITK activity (kaur2012inhibitorsofinterleukin2 pages 12-13). Disease associations implicate ITK in a range of immune‑related conditions, including allergic asthma, atopic dermatitis, and certain T‑cell lymphomas; loss‑of‑function mutations or dysregulated ITK signaling have been linked to immunodeficiencies and aberrant lymphoproliferative disorders, such as fatal Epstein‑Barr virus‑associated lymphoproliferation (aryal2021molecularmechanismof pages 234-237, boucheron2012theroleof pages 16-17). Notably, oncogenic fusion proteins involving ITK, such as ITK‑SYK, have been identified in peripheral T‑cell lymphomas, highlighting a role for ITK in oncogenic signaling pathways (boucheron2012theroleof pages 15-16, zhong2014targetinginterleukin2inducibletcell pages 9-11). These observations have spurred significant interest in the development of ITK inhibitors as clinical candidates; however, despite promising preclinical data, issues such as pharmacokinetic limitations and specificity challenges remain (kaur2012inhibitorsofinterleukin2 pages 14-14, zhong2014targetinginterleukin2inducibletcell pages 8-9).
9. References  
   aryal2021molecularmechanismof pages 92-96; aryal2021molecularmechanismof pages 96-101; aryal2021molecularmechanismof pages 101-104; aryal2021molecularmechanismof pages 104-109; aryal2021molecularmechanismof pages 234-237; blomberg2009geneexpressionanalysis pages 21-25; boucheron2012theroleof pages 1-3; boucheron2012theroleof pages 4-6; boucheron2012theroleof pages 8-9; boucheron2012theroleof pages 9-11; boucheron2012theroleof pages 15-16; boucheron2012theroleof pages 16-17; boucheron2012theroleof pages 17-19; gocek2014nonreceptorproteintyrosine pages 2-3; gurung2023prolineisomerizationfrom pages 9-11; kaur2012inhibitorsofinterleukin2 pages 1-2; kaur2012inhibitorsofinterleukin2 pages 2-3; kaur2012inhibitorsofinterleukin2 pages 3-4; kaur2012inhibitorsofinterleukin2 pages 4-5; kaur2012inhibitorsofinterleukin2 pages 12-13; kaur2012inhibitorsofinterleukin2 pages 13-14; kaur2012inhibitorsofinterleukin2 pages 14-14; kaur2012inhibitorsofinterleukin2 pages 15-15; ortutay2008phylogenyoftec pages 7-10; tarafdar2014interactionsofthe pages 82-87; yu2009tecfamilykinases pages 23-26; yu2009tecfamilykinases pages 50-52; zhong2014targetinginterleukin2inducibletcell pages 1-3; zhong2014targetinginterleukin2inducibletcell pages 3-5; zhong2014targetinginterleukin2inducibletcell pages 5-6; zhong2014targetinginterleukin2inducibletcell pages 6-8; zhong2014targetinginterleukin2inducibletcell pages 8-9; zhong2014targetinginterleukin2inducibletcell pages 9-11.

References

1. (aryal2021molecularmechanismof pages 101-104): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
2. (aryal2021molecularmechanismof pages 237-240): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
3. (blomberg2009geneexpressionanalysis pages 21-25): KEM Blomberg. Gene expression analysis of tec family kinases in b-and t-lymphocytes. Unknown journal, 2009.
4. (boucheron2012theroleof pages 1-3): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
5. (boucheron2012theroleof pages 17-19): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
6. (kaur2012inhibitorsofinterleukin2 pages 1-2): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
7. (kaur2012inhibitorsofinterleukin2 pages 13-14): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
8. (kaur2012inhibitorsofinterleukin2 pages 2-3): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
9. (kaur2012inhibitorsofinterleukin2 pages 3-4): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
10. (kaur2012inhibitorsofinterleukin2 pages 4-5): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
11. (lechner2020roleofthe pages 1-2): Kristina S. Lechner, Markus F. Neurath, and Benno Weigmann. Role of the il-2 inducible tyrosine kinase itk and its inhibitors in disease pathogenesis. Journal of Molecular Medicine, 98:1385-1395, Aug 2020. URL: https://doi.org/10.1007/s00109-020-01958-z, doi:10.1007/s00109-020-01958-z. This article has 54 citations.
12. (ortutay2008phylogenyoftec pages 7-10): Csaba Ortutay, Beston F. Nore, Mauno Vihinen, and C.I. Edvard Smith. Phylogeny of tec family kinases: identification of a premetazoan origin of btk, bmx, itk, tec, txk, and the btk regulator sh3bp5. Advances in Genetics, 64:51-80, Jan 2008. URL: https://doi.org/10.1016/s0065-2660(08)00803-1, doi:10.1016/s0065-2660(08)00803-1. This article has 38 citations and is from a peer-reviewed journal.
13. (tarafdar2014interactionsofthe pages 82-87): S Tarafdar. Interactions of the hiv-1 nef virulence factor with host cell tyrosine kinases of the src and tec families. Unknown journal, 2014.
14. (yu2009tecfamilykinases pages 23-26): L Yu. Tec family kinases: transcriptional and posttranslational regulation. Unknown journal, 2009.
15. (yu2009tecfamilykinases pages 50-52): L Yu. Tec family kinases: transcriptional and posttranslational regulation. Unknown journal, 2009.
16. (zhong2014targetinginterleukin2inducibletcell pages 1-3): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
17. (zhong2014targetinginterleukin2inducibletcell pages 11-13): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
18. (zhong2014targetinginterleukin2inducibletcell pages 3-5): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
19. (zhong2014targetinginterleukin2inducibletcell pages 5-6): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
20. (zhong2014targetinginterleukin2inducibletcell pages 6-8): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
21. (zhong2014targetinginterleukin2inducibletcell pages 8-9): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
22. (zhong2014targetinginterleukin2inducibletcell pages 9-11): Y. Zhong, A. Johnson, J. Byrd, and J. Dubovsky. Targeting interleukin-2-inducible t-cell kinase (itk) in t-cell related diseases. Postdoc journal : a journal of postdoctoral research and postdoctoral affairs, 2 6:1-11, Jun 2014. URL: https://doi.org/10.14304/surya.jpr.v2n6.1, doi:10.14304/surya.jpr.v2n6.1. This article has 29 citations.
23. (aryal2021molecularmechanismof pages 104-109): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
24. (aryal2021molecularmechanismof pages 234-237): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
25. (aryal2021molecularmechanismof pages 92-96): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
26. (aryal2021molecularmechanismof pages 96-101): M Aryal. Molecular mechanism of bruton’s tyrosine kinase activation by the hiv-1 nef virulence factor. Unknown journal, 2021.
27. (boucheron2012theroleof pages 15-16): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
28. (boucheron2012theroleof pages 16-17): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
29. (boucheron2012theroleof pages 4-6): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
30. (boucheron2012theroleof pages 8-9): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
31. (boucheron2012theroleof pages 9-11): Nicole Boucheron and Wilfried Ellmeier. The role of tec family kinases in the regulation of t-helper-cell differentiation. International Reviews of Immunology, 31:133-154, Mar 2012. URL: https://doi.org/10.3109/08830185.2012.664798, doi:10.3109/08830185.2012.664798. This article has 23 citations and is from a peer-reviewed journal.
32. (gocek2014nonreceptorproteintyrosine pages 2-3): 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.
33. (gurung2023prolineisomerizationfrom pages 9-11): Deepti Gurung, Jacob A. Danielson, Afsara Tasnim, Jian-Ting Zhang, Y. Zou, and Jing-Yuan Liu. Proline isomerization: from the chemistry and biology to therapeutic opportunities. Biology, 12:1008, Jul 2023. URL: https://doi.org/10.3390/biology12071008, doi:10.3390/biology12071008. This article has 20 citations and is from a peer-reviewed journal.
34. (kaur2012inhibitorsofinterleukin2 pages 12-13): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
35. (kaur2012inhibitorsofinterleukin2 pages 14-14): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.
36. (kaur2012inhibitorsofinterleukin2 pages 15-15): Maninder Kaur, Malkeet Singh Bahia, and Om Silakari. Inhibitors of interleukin-2 inducible t-cell kinase as potential therapeutic candidates for the treatment of various inflammatory disease conditions. European journal of pharmaceutical sciences : official journal of the European Federation for Pharmaceutical Sciences, 47 3:574-88, Oct 2012. URL: https://doi.org/10.1016/j.ejps.2012.07.013, doi:10.1016/j.ejps.2012.07.013. This article has 28 citations.