1. Phylogeny  
   Tyrosine‐protein kinase Tec (gene: TEC, UniProt: P42680) is a member of the Tec family of non‐receptor tyrosine kinases, a subgroup that also includes Bruton’s tyrosine kinase (BTK), interleukin‐2–inducible T‐cell kinase (ITK), BMX/ETK, TXK (also known as RLK) and others. The Tec family is evolutionarily conserved among vertebrates and is present in diverse hematopoietic lineages. Phylogenetically, these kinases can be traced back to early metazoans, and their presence in numerous species suggests they form an ancient branch of the tyrosine kinome. They belong to the cytoplasmic (non‐receptor) tyrosine kinase group, which stands apart from receptor tyrosine kinases that evolved by gene fusion with extracellular ligand–binding domains. Comparative analyses have shown that the domain architecture characteristic of Tec kinases—comprising an N‐terminal pleckstrin homology (PH) domain, a BTK‐type zinc finger region, and tandem Src‐homology (SH3 and SH2) domains preceding the kinase domain—is deeply conserved (siveen2018roleofnon pages 6-8, alexander2015theconciseguide pages 10-13). Orthologs of TEC exist throughout mammals and are also found in lower vertebrates, confirming their ancient origin and essential role in the adaptive immune system (yeung2021evolutionoffunctional pages 9-10).
2. Reaction Catalyzed  
   Tec catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on target proteins. The general reaction can be described as follows:  
     ATP + [Protein]-L-tyrosine → ADP + [Protein]-phospho-L-tyrosine + H^+  
   Physiologically, Tec phosphorylates substrates that are integral to cell signaling. For example, it phosphorylates DOK1 and STAP1 in the context of CD28- and B-cell receptor (BCR)-mediated signaling, respectively, and it is responsible for the phosphorylation of FGF2 on Tyr-215, which is critical for an unconventional secretion pathway. In addition, Tec-mediated phosphorylation of GRB10 has been implicated in the reciprocal regulation of FOS transcription (siveen2018roleofnon pages 6-8). Although the precise kinetic mechanism and transient intermediates remain to be fully elucidated, the enzyme follows the general mechanism of tyrosine kinases where proper alignment of the substrate’s hydroxyl group and coordinated binding of ATP are essential to facilitate efficient phosphoryl transfer (oliveira2016revisitingproteinkinase–substrate pages 1-2).
3. Cofactor Requirements  
   Like other protein kinases, Tec requires ATP as a phosphate donor and typically depends on the presence of divalent metal ions to coordinate ATP binding. In most tyrosine kinases, Mg^2+ is essential for phosphotransfer activity because it stabilizes the negative charges of the ATP phosphate groups and is coordinated by conserved residues in the kinase active site. Although explicit experimental details for Tec are not provided in every report, by analogy with related kinases (e.g., BTK, ITK) and information on the conserved active site geometry drawn from structural studies, it is widely accepted that Tec relies on Mg^2+ for catalytic activity (alexander2015theconciseguide pages 10-13, taft2017ayeastbasedassay pages 13-22).
4. Substrate Specificity  
   Tec’s substrate specificity is determined by both the intrinsic properties of its catalytic domain and interactions mediated by its regulatory domains. Among its known physiological substrates are:  • DOK1, which acts as a substrate during signaling downstream of CD28 in T cells, thereby contributing to adaptive immune regulation (siveen2018roleofnon pages 6-8).  
    • STAP1, a B-cell receptor (BCR)–associated protein that becomes phosphorylated by Tec, thereby playing a role in B-cell activation and development (siveen2018roleofnon pages 6-8).  
    • FGF2, where phosphorylation of Tyr-215 by Tec modulates an unconventional secretion mechanism from the endoplasmic reticulum/Golgi (siveen2018roleofnon pages 6-8).  
    • GRB10, whose phosphorylation by Tec is involved in attenuating FOS transcriptional activation (siveen2018roleofnon pages 6-8).  
   Although a defined consensus substrate motif for Tec has not been as rigorously established as for some serine/threonine kinases, its substrate recognition likely involves coordination of residues adjacent to the target tyrosine by interactions contributed by its kinase domain as well as docking interactions mediated by SH2 and SH3 domains. These domains help in the spatial localization and substrate engagement within receptor complexes (bryan2018kinaseinhibitorsfor pages 8-10, oliveira2016revisitingproteinkinase–substrate pages 1-2).
5. Structure  
   Tec exhibits a modular architecture that is characteristic of the Tec family of non-receptor tyrosine kinases. Its domain organization is as follows:  • An N-terminal pleckstrin homology (PH) domain, which binds phosphoinositides such as PIP3. This binding recruits Tec to cellular membranes where lipid signaling is active.  
    • A BTK-type zinc finger (or BTK motif), which is thought to contribute to protein stability and may also play a role in substrate interactions.  
    • SH3 and SH2 domains that mediate protein–protein interactions. The SH3 domain typically binds proline-rich motifs, whereas the SH2 domain specifically engages phosphotyrosine-containing sequences. Together, these domains are critical for assembling signaling complexes and for allosteric regulation (siveen2018roleofnon pages 6-8, alexander2015theconciseguide pages 10-13).  
    • A C-terminal kinase (catalytic) domain that is bilobal—with the N-terminal lobe largely consisting of β-sheets and the C-terminal lobe rich in α-helices—and contains the active site responsible for ATP binding and phosphotransfer. Key catalytic residues include the invariant aspartate necessary for catalysis and likely a cysteine residue in the ATP-binding pocket that can serve as a site for covalent inhibitor interaction (e.g., in dual JAK3/TEC inhibitors) (xu2019pf06651600adual pages 6-7, lin2024conformationalheterogeneityof pages 27-28).  
   High-resolution structural information obtained by crystallography and computational methods such as AlphaFold modeling has provided insights into the conformational flexibility of the kinase domain and autoinhibitory states mediated via interdomain contacts (chopra2016dynamicallosterymediated pages 1-2, lin2024conformationalheterogeneityof pages 27-28).
6. Regulation  
   Tec is regulated through a combination of post-translational modifications and domain–domain interactions that modulate its catalytic activity. The following regulatory mechanisms have been described:  • Phosphorylation events: Tec itself is subject to tyrosine phosphorylation events that can either promote or inhibit its activity. For instance, autophosphorylation within the activation loop of the catalytic domain is an essential step for full activation, while phosphorylation events on other regulatory residues may modulate its interactions with partner proteins (siveen2018roleofnon pages 6-8, andreotti2010tcellsignalingregulated pages 18-19).  
    • Lipid binding: The PH domain of Tec binds phosphoinositides (especially PIP3), enabling its membrane localization where it is activated by receptor signals. This spatial regulation is critical for initiating downstream signaling cascades (lien2017pi3ksignalingin pages 4-6).  
    • Protein–protein interactions: The SH2 and SH3 domains play key roles in docking to phosphorylated partners and proline-rich sequences, respectively. Such interactions not only target Tec to specific signaling complexes but also contribute to its autoinhibition through intramolecular associations that restrict its kinase activity in the absence of activating signals (chopra2016dynamicallosterymediated pages 17-18, raussendorf2017aswitchin pages 1-2).  
    • Reciprocal phosphorylation: Tec cooperates with other kinases, notably JAK2, through reciprocal phosphorylation events that modulate transcription factor activation (e.g., FOS induction) downstream of cytokine signaling (siveen2018roleofnon pages 6-8, xu2019pf06651600adual pages 7-8).  
   In addition, allosteric regulation involving interdomain contacts and coupled conformational transitions—such as those mediated by dynamic changes in the αC helix and the regulatory spine—is emerging as a critical mechanism controlling Tec kinase activity (chopra2016dynamicallosterymediated pages 1-2, raussendorf2017aswitchin pages 8-9).
7. Function  
   Tec functions as a signal transducer in multiple downstream pathways emanating from a variety of receptor types. Its biological roles include:  • Immune cell signaling: Tec plays a redundant role relative to ITK in T-cell receptor (TCR) signaling. It is required for TCR-dependent IL-2 gene induction and contributes to the differentiation and function of conventional T cells as well as nonconventional natural killer T (NKT) cells (siveen2018roleofnon pages 6-8, andreotti2010tcellsignalingregulated pages 19-19).  
    • B-cell receptor (BCR) signaling: Tec acts redundantly with BTK to facilitate B-cell development and activation. In this context, phosphorylation of the adaptor protein STAP1 by Tec is important for propagating BCR-mediated signals (siveen2018roleofnon pages 6-8).  
    • Mast cell and myeloid cell activation: Tec is required in mast cells for efficient cytokine production and contributes to the growth, differentiation, and functional activation of myeloid cells in response to granulocyte colony-stimulating factor (CSF3) (siveen2018roleofnon pages 6-8).  
    • Platelet signaling: Tec participates in integrin-mediated platelet signaling, thus playing a role in thrombus formation and hemostasis.  
    • Hepatocyte proliferation and liver regeneration: Tec is involved in transducing signals downstream of hepatocyte growth factor (HGF) and mediates ERK signaling, which is critical for liver regeneration (siveen2018roleofnon pages 6-8).  
    • Regulation of unconventional secretion: By phosphorylating FGF2 on Tyr-215, Tec regulates an ER/Golgi-independent secretion mechanism that is important under various physiological conditions.  
    • Modulation of transcription factors: Tec can influence gene expression by, for example, phosphorylating GRB10—a negative regulator of FOS transcription—thereby impacting cytokine-driven growth and differentiation signals (siveen2018roleofnon pages 6-8, xu2019pf06651600adual pages 7-8).  
   Collectively, these functions underline Tec’s broad role in mediating signaling from cytokine receptors, receptor tyrosine kinases, G-protein–coupled receptors, and integrins, with significant impacts on immune regulation, hematopoiesis, liver regeneration, and even osteoclast differentiation (siveen2018roleofnon pages 6-8, OpenTargets Search: -TEC).
8. Other Comments  
   Tec is emerging as an important therapeutic target given its pivotal roles in signaling pathways that contribute to both normal immune function and disease pathogenesis. Dual inhibitors that target both JAK3 and Tec family kinases, such as PF-06651600, have been shown to modulate immune cell functions including T-cell activation and cytolytic responses in CD8^+ T cells and natural killer cells (xu2019pf06651600adual pages 1-2, xu2019pf06651600adual pages 7-8). In addition, abnormal Tec signaling has been implicated in tumorigenesis (for example, via overexpression in liver cancers) and its precise regulation is associated with control of hematologic malignancies and inflammatory diseases. Although specific clinically approved inhibitors directly targeting Tec are not currently well established, inhibitors developed against related Tec family members (such as BTK inhibitors) highlight the translational potential of modulating Tec activity (bryan2018kinaseinhibitorsfor pages 8-10). Recent genetic association studies and database searches (OpenTargets Search: -TEC) further support the notion that TEC signaling plays a role in autoimmune conditions such as rheumatoid arthritis and alopecia areata. From a research perspective, ongoing studies are focused on detailed structural analyses (including crystallographic and AlphaFold-based approaches) and dynamic regulation via allosteric mechanisms to better understand substrate specificity and post-translational modifications that govern Tec activity (chopra2016dynamicallosterymediated pages 1-2, lin2024conformationalheterogeneityof pages 27-28). These insights might ultimately lead to the development of new therapeutic strategies to modulate Tec-dependent signaling in diverse clinical contexts.
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