## 1. Phylogeny

Proto‐oncogene tyrosine‐protein kinase Src (SRC) is a member of the Src family kinases (SFKs), a well‐defined subgroup of non‐receptor protein tyrosine kinases that have been conserved across metazoan species. The SFK family comprises at least nine members—including Src, Fyn, Yes, Lck, Lyn, Hck, Blk, Fgr, and Yrk—that arose early in animal evolution and have been maintained due to their central role in intracellular signal transduction (lin2005probingtheregulatory pages 26-30, ayrapetov2006structuralandfunctional pages 18-21). Phylogenetic analyses based on kinase domain sequence comparisons indicate that the evolution of SFKs parallels the development of increasingly complex receptor systems in multicellular organisms, and Src itself can be traced to a common ancestor that gave rise to the modern repertoire of tyrosine kinases (lin2005probingtheregulatory pages 26-30). In addition, the phylogenetic context of SRC emphasizes its close evolutionary relationship to regulatory kinases such as C-terminal Src kinase (Csk) and its homologues, which share similar domain architectures and function in counterbalancing Src activity through phosphorylation of critical regulatory residues (ayrapetov2006structuralandfunctional pages 106-110, lin2005probingtheregulatory pages 22-26).

## 2. Reaction Catalyzed

Src catalyzes the transfer of the γ-phosphate group from ATP to specific tyrosine residues on target proteins. The overall chemical reaction proceeds according to the following stoichiometry:  
  ATP + [protein]–OH (tyrosine) → ADP + [protein]–OPO₃²⁻ (phosphotyrosine) + H⁺  
This phosphoryl transfer reaction underlies the modulation of substrate activity and is central to the propagation of intracellular signaling cascades (ayrapetov2006structuralandfunctional pages 14-18, lin2005probingtheregulatory pages 44-46).

## 3. Cofactor Requirements

The catalytic activity of Src is strictly dependent on the presence of ATP as a phosphate donor and a divalent metal ion, typically Mg²⁺, which serves as an essential cofactor. Mg²⁺ ions coordinate with the phosphates of ATP within the active site of Src, thereby facilitating the efficient transfer of the γ-phosphate to the hydroxyl group of tyrosine residues in substrate proteins (ayrapetov2006structuralandfunctional pages 140-143, lin2005probingtheregulatory pages 44-46).

## 4. Substrate Specificity

Src exhibits a defined substrate specificity that is determined by both its catalytic kinase domain and the regulatory SH2 and SH3 domains. The kinase preferentially phosphorylates tyrosine residues, and its substrate recognition is largely influenced by the presence of specific amino acid motifs surrounding these residues. In particular, the SH2 domain of Src displays a high binding affinity for phosphotyrosine-containing sequences that conform to a consensus motif, most notably pYEEI, even though the intramolecular interaction that maintains its autoinhibited state utilizes a non-canonical phosphopeptide sequence (pTyr-Gln-Pro-Gly) (ayrapetov2006structuralandfunctional pages 33-36). In addition, Src substrates often contain proline-rich regions that interact with its SH3 domain, thereby providing a dual mechanism of substrate targeting: the catalytic domain directs the phosphorylation event while the SH2/SH3 domains enhance substrate specificity and subcellular localization (korademirnics2000srckinasemediatedsignaling pages 2-3, ayrapetov2006structuralandfunctional pages 33-36).

## 5. Structure

Src is organized into several distinct structural domains that contribute both to its catalytic activity and regulatory mechanisms. At the N-terminus, Src contains a short SH4 domain that includes a myristoylation signal required for membrane anchoring, allowing Src to associate with cellular membranes where many of its substrates reside (schenone2007srcinhibitorsand pages 2-2, ayrapetov2006structuralandfunctional pages 106-110). Immediately downstream lies a unique region that, although poorly conserved between different SFKs, may confer specificity for protein–protein interactions. Following the unique region, Src comprises an SH3 domain that binds proline-rich peptides and an SH2 domain that recognizes phosphotyrosine-containing motifs. These regulatory domains participate in intramolecular interactions that stabilize an autoinhibited conformation of the kinase (ayrapetov2006structuralandfunctional pages 103-106, schenone2007srcinhibitorsand pages 2-2).  
Central to Src’s structure is the catalytic (kinase) domain—also referred to as the SH1 domain—which exhibits a classical bilobed architecture characterized by a smaller N-terminal lobe that hosts the ATP binding site and a larger C-terminal lobe that contains the substrate-binding region and the activation loop. The activation loop, in particular, is a critical regulatory element; its autophosphorylation at Tyr416 (Tyr419 in human Src) is required for full enzymatic activation, whereas the phosphorylation of a conserved tyrosine residue located in the C-terminal tail (Tyr527 in chicken Src, Tyr530 in human Src) engages in an intramolecular interaction with the SH2 domain to maintain Src in an inactive state (ayrapetov2006structuralandfunctional pages 140-143, ayrapetov2006structuralandfunctional pages 153-155). Key structural features such as the positioning of the αC-helix and the formation of a hydrophobic spine further stabilize the active conformation once the autoinhibitory interactions are disrupted (ayrapetov2006structuralandfunctional pages 36-39, schenone2007srcinhibitorsand pages 9-9). This modular organization not only underlies the catalytic function of Src but also permits the integration of regulatory inputs via its SH2 and SH3 domains (schenone2007srcinhibitorsand pages 2-2, ayrapetov2006structuralandfunctional pages 106-110).

## 6. Regulation

The activity of Src is subject to multiple layers of regulation that are mediated through dynamic post-translational modifications and intramolecular domain interactions. A central regulatory mechanism is phosphorylation. Src is maintained in an inactive state by the phosphorylation of its C-terminal tyrosine residue (Tyr527/Tyr530), which is catalyzed by C-terminal Src kinase (Csk) and its homolog Chk. This phosphorylation event promotes the binding of the phosphotyrosine to the SH2 domain, thereby locking Src in a closed, autoinhibited conformation (ayrapetov2006structuralandfunctional pages 106-110, ayrapetov2006structuralandfunctional pages 131-134).  
Conversely, dephosphorylation of the C-terminal inhibitory residue by specific protein tyrosine phosphatases leads to disruption of the intramolecular SH2–tail interaction, allowing Src to transition into an open, active state. In the active conformation, Src undergoes autophosphorylation at a tyrosine residue within the activation loop (Tyr416/Tyr419), which enhances its catalytic activity by stabilizing the active conformation and optimizing substrate access (ayrapetov2006structuralandfunctional pages 103-106, lin2005probingtheregulatory pages 141-144).  
Apart from phosphorylation, regulation of Src also involves interactions mediated by its SH3 domain. The SH3 domain binds to a proline-rich linker region between the SH2 and kinase domains, contributing to the maintenance of the inactive conformation. Specific residues such as Trp260 play a critical role in this regulatory interface, ensuring complete inactivation of Src (ayrapetov2006structuralandfunctional pages 143-145, ayrapetov2006structuralandfunctional pages 145-148).  
Furthermore, regulatory inputs include membrane targeting via N-terminal myristoylation and potentially palmitoylation (although palmitoylation is not a feature of Src itself but is observed in some other SFKs), which focus Src activity at sites of receptor clustering and facilitate its interaction with activated receptor complexes (schenone2007srcinhibitorsand pages 2-2, ayrapetov2006structuralandfunctional pages 106-110). These diverse control mechanisms ensure that Src activity is tightly modulated in response to extracellular signals (lin2005probingtheregulatory pages 141-144, ayrapetov2006structuralandfunctional pages 145-148).

## 7. Function

Src plays a pivotal role in the transmission of signals from the cell surface to intracellular pathways that govern a broad spectrum of biological processes. It is activated following the engagement of multiple classes of receptors—including immune response receptors, integrins, receptor protein tyrosine kinases, G protein-coupled receptors, and cytokine receptors—and is frequently one of the earliest kinases activated following receptor clustering or dimerization (Information; ayrapetov2006structuralandfunctional pages 106-110, sato2013cellularfunctionsregulated pages 16-19).  
Upon activation, Src phosphorylates a variety of target substrates involved in mediating gene transcription, cell adhesion, cell cycle progression, apoptosis, and migratory responses. For example, Src is known to phosphorylate the epidermal growth factor receptor (EGFR) on tyrosine 845, a modification that is crucial for the transduction of mitogenic signals in cancer cells (sato2013cellularfunctionsregulated pages 16-19). In leukocytes, Src family kinases regulate immune cell activation and cytoskeletal dynamics by phosphorylating adaptor proteins, kinases, and components of the focal adhesion complex, thereby influencing processes such as phagocytosis, cell motility, and survival (korademirnics2000srckinasemediatedsignaling pages 1-2, korademirnics2000srckinasemediatedsignaling pages 6-7).  
Additionally, Src participates in integrin-mediated signaling; its activity modulates the assembly of focal adhesion complexes through phosphorylation of focal adhesion kinase (FAK) and other cytoskeletal proteins, which contributes to cell migration and invasion—a function that is particularly relevant in the context of cancer metastasis (schenone2007srcinhibitorsand pages 8-9, korademirnics2000srckinasemediatedsignaling pages 3-4).  
Moreover, Src functions as an upstream activator of other protein tyrosine kinase families, thereby amplifying downstream signaling cascades that regulate crucial cellular processes such as proliferation and differentiation. Described as a central hub in cellular signaling networks, Src’s broad substrate specificity allows it to integrate signals from diverse receptor systems, thus impacting processes ranging from cytoskeletal remodeling to gene expression (Information, ayrapetov2006structuralandfunctional pages 103-106, korademirnics2000srckinasemediatedsignaling pages 2-3).

## 8. Other Comments

Given its critical role in signal transduction, Src has been the subject of extensive pharmacological research. A variety of small-molecule inhibitors targeting Src activity have been developed, many of which are ATP-competitive compounds such as PP1, PP2, and others that bind within the deep ATP pocket of the kinase domain (schenone2007srcinhibitorsand pages 6-7, schenone2007srcinhibitorsand pages 9-10). These inhibitors have been investigated for their anti-angiogenic and anti-tumor properties, as aberrant Src activity is strongly associated with oncogenesis, metastasis, and the regulation of proangiogenic factors.  
Disease associations of Src are well documented; overactivation or dysregulation of Src kinase activity has been implicated in a wide range of human cancers, including breast, colon, pancreatic, and lung cancers, as well as in certain hematological malignancies (sato2013cellularfunctionsregulated pages 16-19, schenone2007srcinhibitorsand pages 8-9). Although mutations in the SRC gene are infrequent, altered expression and aberrant activation mechanisms contribute to oncogenic transformation and aggressive tumor phenotypes, thereby rendering Src a promising therapeutic target in oncology.  
Furthermore, the interplay between Src and its negative regulators, such as Csk and Chk, provides additional avenues for therapeutic intervention, as compounds that can modulate these regulatory interactions may indirectly suppress Src activity. The critical involvement of Src in the modulation of integrin-mediated adhesion and cytoskeletal organization also implicates it in processes such as cell migration and invasion, which are central to cancer metastasis and tissue remodeling in pathological conditions (ayrapetov2006structuralandfunctional pages 148-150, korademirnics2000srckinasemediatedsignaling pages 8-9).  
Notably, the clinical development of Src inhibitors has been met with challenges related to selectivity and toxicity, as the highly conserved nature of the kinase domain among different tyrosine kinases complicates the design of compounds with absolute specificity. Efforts to overcome these issues include structure-based drug design and the exploration of allosteric inhibition strategies that exploit unique regulatory features of Src (schenone2007srcinhibitorsand pages 6-7, schenone2007srcinhibitorsand pages 9-10).

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