1. Phylogeny  
   Tyrosine‐protein kinase ABL1 is a member of the Abl family of non‐receptor tyrosine kinases that is evolutionarily conserved from invertebrates to vertebrates, with homologs identifiable in organisms such as Drosophila melanogaster and Caenorhabditis elegans before a gene duplication event in vertebrates produced the paralogs ABL1 and ABL2 (colicelli2010abltyrosinekinases pages 1-2, greuber2013roleofabl pages 1-2). Comparative genomic studies have placed ABL1 within the core kinome as defined by systematic analyses of the human protein kinase complement, and it is grouped within the Src-related kinase family based on its conserved SH3–SH2–kinase domain cassette (colicelli2010abltyrosinekinases pages 2-4, greuber2013roleofabl pages 2-4). Phylogenetic reconstruction based on the catalytic and regulatory domains indicates that ABL1’s central module has maintained a highly conserved structure over evolutionary time, with the divergence of its regulatory and substrate recognition motifs further refining its specialized roles in higher eukaryotes (colicelli2010abltyrosinekinases pages 27-36, hantschel2012structureregulationsignaling pages 1-2).
2. Reaction Catalyzed  
   ABL1 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of tyrosine residues on substrate proteins. The general reaction can be written as: ATP + protein (tyrosine) → ADP + protein (phosphotyrosine) + H⁺ (colicelli2010abltyrosinekinases pages 5-7, arrington2019identificationofthe pages 10-11).
3. Cofactor Requirements  
   The kinase activity of ABL1 is dependent on the binding of ATP and requires divalent metal ions, most notably Mg²⁺, to coordinate the phosphates of ATP during catalysis (cao2008enhancementofabl pages 1-2, irgit2025structureanddynamics pages 1-3).
4. Substrate Specificity  
   Studies using kinase assay linked phosphoproteomics and subsequent motif analysis have demonstrated that ABL1 exhibits a substrate preference characterized by specific amino acid residues flanking the phosphorylated tyrosine. In vitro experiments indicate that ABL1 preferentially phosphorylates substrates that display an acidic residue at positions upstream of the target tyrosine and typically a hydrophobic residue, such as proline or phenylalanine, at the +3 position relative to the phosphorylated site (arrington2019identificationofthe pages 5-6, arrington2019identificationofthe pages 7-8). Sequence motif analyses performed with tools like WebLogo and motif-x confirm that the consensus phosphorylation motif of ABL1 is consistent with earlier studies and is enriched for residues that facilitate efficient substrate recognition by its SH2 domain, thereby enabling processive phosphorylation (arrington2019identificationofthe pages 8-9, colicelli2010abltyrosinekinases pages 5-7). Furthermore, recent delineation of human tyrosine kinase substrate specificities supports the notion that ABL1 recognizes a motif with particular sequence constraints, although specific consensus motifs for tyrosine kinases have also been reported in the literature (manley2020thespecificityof pages 1-6).
5. Structure  
   ABL1 exhibits a multidomain organization that is central to its catalytic function and regulatory control. The protein is composed of an N-terminal cap region that, in one of its major isoforms (ABL1b), is myristoylated, a modification that plays an essential role in autoinhibition by docking into a hydrophobic pocket within the kinase domain; this N-terminal difference distinguishes isoforms such as 1a (non-myristoylated) from 1b (irgit2025structureanddynamics pages 5-6, hantschel2012structureregulationsignaling pages 2-3). Following the cap, ABL1 contains an SH3 domain that binds proline-rich motifs, and an SH2 domain that selectively interacts with phosphotyrosine-containing sequences; these two domains function cooperatively to mediate intramolecular interactions that stabilize the kinase in its inactive conformation (colicelli2010abltyrosinekinases pages 1-2, colicelli2010abltyrosinekinases pages 4-5). The central catalytic (SH1) domain has a bilobal structure with a smaller N-terminal lobe that includes a glycine-rich phosphate-binding P-loop and an αC helix that is critical for orienting key catalytic residues, and a larger C-terminal lobe largely responsible for substrate binding (irgit2025structureanddynamics pages 1-3, colicelli2010abltyrosinekinases pages 8-10). A prominent structural feature is the activation loop (A-loop), which undergoes autophosphorylation at tyrosine 412 and shifts from a closed (inactive) to an open (active) conformation to allow substrate access to the catalytic site (colicelli2010abltyrosinekinases pages 10-12, colicelli2010abltyrosinekinases pages 15-16). In addition, ABL1 contains a long C-terminal tail that harbors nuclear localization signals, actin-binding domains, and other protein-protein interaction motifs that are implicated in cytoskeletal regulation and nuclear functions (colicelli2010abltyrosinekinases pages 17-19, irgit2025structureanddynamics pages 3-4).
6. Regulation  
   ABL1 is subject to complex regulation that involves multiple layers of post-translational modifications and conformational control. Autoinhibition of ABL1 is achieved through intramolecular interactions whereby the SH3 domain is tethered to the SH2-kinase linker, and in the 1b isoform, the N-terminal myristoyl group is inserted into a pocket in the C-lobe of the kinase domain, maintaining the kinase in an inactive state (hantschel2012structureregulationsignaling pages 4-5, colicelli2010abltyrosinekinases pages 2-4). Activation of ABL1 occurs when these inhibitory constraints are disrupted, either via phosphorylation of key residues – notably autophosphorylation at tyrosine 412 in the activation loop – or through the binding of regulatory proteins that alter the domain interactions (colicelli2010abltyrosinekinases pages 10-12, colicelli2010abltyrosinekinases pages 13-15). Additional phosphorylation events at residues outside the activation loop, such as tyrosines within the SH2-kinase linker, further modulate its activity, while phosphorylation of inhibitor proteins like ABI1 can also impact its catalytic function through feedback mechanisms (arrington2019identificationofthe pages 6-7, colicelli2010abltyrosinekinases pages 16-17). Beyond phosphorylation, ABL1 is regulated by ubiquitination mediated by E3 ligases, and its subcellular localization is tightly controlled by the presence of nuclear localization signals (NLS) and nuclear export signals (colicelli2010abltyrosinekinases pages 19-20, irgit2025structureanddynamics pages 3-4). The interplay of these regulatory modifications ensures that ABL1 activity is precisely coordinated in response to extracellular stimuli and intracellular signals (greuber2013roleofabl pages 15-16, mayro2022thecharacterizationof pages 140-144).
7. Function  
   The biological roles of ABL1 are extensive and diverse, reflecting its involvement in multiple cellular processes that underpin cell growth, survival, and homeostasis. In the cytoplasm, ABL1 modulates actin cytoskeleton dynamics by phosphorylating key regulators such as WASF3, ANXA1, DBN1, DBNL, CTTN, RAPH1, and ENAH; phosphorylation of WASF3 is critical for the formation of lamellipodia and effective cell migration (arrington2019identificationofthe pages 5-6, colicelli2010abltyrosinekinases pages 23-24). It also regulates microtubule-associated proteins like MAPT and PXN, thereby influencing cell structure and motility (colicelli2010abltyrosinekinases pages 5-7, irgit2025structureanddynamics pages 3-4). ABL1 is intimately involved in receptor endocytosis, phosphorylating and modulating proteins such as EGFR and components of the endocytic machinery including caveolin (CAV1) and RIN1, and it exerts regulatory control over the CBL family of ubiquitin ligases that further contribute to receptor turnover and actin remodeling (arrington2019identificationofthe pages 10-11, colicelli2010abltyrosinekinases pages 27-36). In the context of autophagy, ABL1 positively regulates the trafficking and function of lysosomal components, and it has been shown to target mitochondria under oxidative stress conditions, thereby contributing to mitochondrial dysfunction and programmed cell death (irgit2025structureanddynamics pages 5-6, colicelli2010abltyrosinekinases pages 15-16). Within the nucleus, ABL1 translocates in response to DNA damage where it binds DNA directly and phosphorylates substrates involved in the DNA damage response and repair, such as DDB1, DDB2, ERCC3, ERCC6, RAD51, and TP73, ultimately leading to cell cycle arrest or apoptosis when damage is irreparable (colicelli2010abltyrosinekinases pages 36-37, mayro2022thecharacterizationof pages 136-140). Additionally, ABL1 contributes to the regulation of immune cell function, including T-cell differentiation and chemokine-mediated migration, through phosphorylation of transcription factors such as TBX21 and adaptors like NEDD9 and CRK (greuber2013roleofabl pages 14-15, mayro2022thecharacterizationof pages 29-34).
8. Other Comments  
   ABL1 is a major therapeutic target in oncology, most notably in chronic myelogenous leukemia (CML) due to the formation of the constitutively active BCR-ABL1 fusion protein resulting from a t(9;22) chromosomal translocation (colicelli2010abltyrosinekinases pages 37-37, arrington2019identificationofthe pages 10-11). A range of tyrosine kinase inhibitors (TKIs) have been developed to target ABL1, including first-generation inhibitors such as imatinib, second-generation inhibitors such as dasatinib, nilotinib, and bosutinib, and third-generation agents such as ponatinib; additionally, the allosteric inhibitor asciminib targets the myristate-binding pocket, offering a distinct mechanism of inhibition that circumvents many resistance-conferring mutations (manley2020thespecificityof pages 22-26, larocque2021targetingdrugresistantcancerdriver pages 14-20). Resistance to ATP-competitive inhibitors often arises through mutations in the kinase domain, which underscores the significance of ABL1’s regulatory architecture and the need for diverse inhibitory strategies (colicelli2010abltyrosinekinases pages 23-24, greuber2013roleofabl pages 15-16). Beyond its oncogenic roles, ABL1 is also exploited by several pathogens, with microbial proteins from viruses and bacteria serving as substrates that allow these organisms to hijack host actin cytoskeleton dynamics (arrington2019identificationofthe pages 8-9, mayro2022thecharacterizationof pages 140-144). Thus, ABL1 is linked to both cancer pathology and infectious disease mechanisms. Furthermore, ABL1’s involvement in DNA damage response and its role in the activation of pro-apoptotic pathways situate it at an important nexus between cell survival and programmed cell death, thereby influencing both developmental processes and responses to cellular stress (colicelli2010abltyrosinekinases pages 36-37, mayro2022thecharacterizationof pages 17-24).
9. References  
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