## 1. Phylogeny

Tyrosine‐protein kinase ABL2 (also known as ARG) belongs to the non‐receptor tyrosine kinase group and is a member of the Abl family, which also includes ABL1. The Abl family kinases are evolutionarily conserved proteins that share the characteristic Src homology (SH) modules—namely, the SH3, SH2, and kinase (SH1) domains—which together form a functional “cassette” essential for autoinhibitory regulation and catalytic activity (aleem2015constitutiveactivityin pages 1-3). Phylogenetic analyses indicate that the conserved SH3‐SH2‐kinase domain architecture was already established in ancestral unicellular organisms, notably in choanoflagellates such as Monosiga brevicollis, and has been maintained in metazoans (aleem2015constitutiveactivityin pages 3-4, aleem2015constitutiveactivityin pages 4-6). In vertebrates, gene duplication events yielded two paralogs, ABL1 and ABL2, with the former containing additional nuclear localization signals and an N-terminal myristoylation motif that are absent in ABL2. Consequently, ABL2 displays a predominantly cytoplasmic distribution and evolves specialized roles in cytoskeletal regulation (mayro2022thecharacterizationof pages 17-24, abrishami2018generationofsynthetic pages 20-25). Orthologs of ABL2 have been identified across a spectrum of vertebrate species, reflecting evolutionary constraints on the catalytic core and associated regulatory domains; this conservation underscores the essential functions of ABL kinases in cell regulation from unicellular ancestors through complex metazoans (aleem2015constitutiveactivityin pages 11-13, santos2016paralogspecificpatternsof pages 2-3, shah2018thesrcmodule pages 18-19).

## 2. Reaction Catalyzed

ABL2 catalyzes the transfer of a phosphate group from adenosine triphosphate (ATP) to tyrosine residues on substrate proteins. This reaction yields adenosine diphosphate (ADP), a phosphotyrosine-modified substrate, and a proton (H⁺). The kinase operates via a conserved catalytic mechanism common to tyrosine kinases, wherein the binding of ATP and the target protein within the kinase catalytic cleft aligns the reactive γ-phosphate for transfer to the hydroxyl group of the substrate’s tyrosine residue (aleem2015constitutiveactivityin pages 10-11, taft2017ayeastbasedassay pages 26-30). Although the specific amino acid sequences recognized by ABL2 vary among substrates, the overall chemical transformation conforms to the general reaction: ATP + protein–tyrosine → ADP + protein–phosphotyrosine + H⁺.

## 3. Cofactor Requirements

Like most protein kinases, ABL2 requires divalent metal ions—most commonly Mg²⁺—to stabilize the binding of ATP within the active site and to facilitate phosphoryl transfer (taft2017ayeastbasedassay pages 26-30). Magnesium acts to neutralize the negative charges of the phosphate groups on ATP, ensuring proper orientation and reactivity during catalysis. Although no alternative cofactors have been explicitly described for ABL2 in the available literature, the conservation of metal-ion dependency across the tyrosine kinase family strongly supports a requirement for Mg²⁺ as an essential cofactor (wu2021plateletderivedgrowthfactor pages 10-16).

## 4. Substrate Specificity

ABL2 exhibits substrate specificity that underpins its role in mediating cytoskeletal reorganization and signal transduction. Physiologically, ABL2 phosphorylates a range of substrates involved in the regulation of cell motility, adhesion, and endocytosis. Notable substrates include proteins that control actin dynamics—such as non-muscle myosin heavy chain IIB (MYH10) and cortactin (CTTN)—as well as tubulin subunits (TUBA1, TUBB), which are essential for microtubule stability and remodeling (Protein Function information). In addition, ABL2 phosphorylates key regulatory proteins such as CRK, CRKL, and DOK1, as well as ARHGAP35, where adhesion-dependent phosphorylation promotes the recruitment of ARHGAP35 in association with RASA1 at the cell periphery, ultimately leading to inhibition of RHO activity (Protein Function information). Receptor tyrosine kinases, like PDGFRB, are also substrates of ABL2, and proteins involved in endocytosis (such as RIN1) are phosphorylated by this kinase, thereby modulating receptor internalization and downstream signaling cascades (wang2015theemergingrole pages 2-4, abrishami2018generationofsynthetic pages 25-29). Although a precise consensus motif remains less well defined compared to certain serine/threonine kinases, ABL2’s substrate targeting appears to be influenced both by recognition of linear sequence motifs and by the presence of accessory SH2/SH3 interacting domains that secure substrates in proximity to the catalytic core (taft2017ayeastbasedassay pages 30-35, corwin2016decipheringhumancytoplasmic pages 126-130).

## 5. Structure

The molecular architecture of ABL2 reflects its dual functions in catalysis and cytoskeletal regulation. ABL2 possesses an N-terminal region that exhibits a highly conserved SH3-SH2-kinase (SH1) cassette shared with ABL1, conferring catalytic activity and regulatory control via intramolecular interactions (aleem2015constitutiveactivityin pages 1-3, mayro2022thecharacterizationof pages 17-24). The SH3 domain contributes to binding proline-rich motifs on interacting proteins, while the SH2 domain recognizes phosphotyrosine-containing sequences, thereby linking ABL2 to dynamic signaling complexes. The central kinase domain, which contains the activation loop harboring a critical tyrosine residue analogous to Y412 in ABL1, is responsible for ATP binding and catalysis (aleem2015constitutiveactivityin pages 4-6, corwin2016decipheringhumancytoplasmica pages 126-130).

A distinguishing structural feature of ABL2 compared to ABL1 is found in its C-terminal extension. Whereas ABL1 contains nuclear localization signals (NLS) and exhibits shuttling between the cytosol and nucleus, ABL2 lacks an NLS and is confined predominantly to the cytoplasm (abrishami2018generationofsynthetic pages 25-29, taft2017ayeastbasedassay pages 30-35). Instead, ABL2 harbors unique domains for binding F-actin and microtubules, which facilitate its role in orchestrating cytoskeletal remodeling. These domains are critical for mediating ABL2’s F-actin bundling activity and for anchoring the kinase at specific subcellular sites such as focal adhesions and invadopodia (lyu2022abl2regulatesmicrotubule pages 35-40, marco2020studyofthea pages 42-47). Structural studies, including X-ray crystallography and homology modeling, have delineated the architecture of the conserved catalytic core and have identified key residues that serve as conformational switches during activation. The absence of an N-terminal myristoylation signal in ABL2, which in ABL1 contributes to autoinhibition by stabilizing an inactive conformation via a hydrophobic pocket in the kinase domain, suggests that ABL2 operates under distinct regulatory paradigms (aleem2015constitutiveactivityin pages 3-4, lyu2022abl2regulatesmicrotubule pages 40-46).

Furthermore, crystal structures of Abl kinases have underscored the significance of the SH3-SH2 clamping mechanism that functions as an autoinhibitory module; while ABL1 uses an N-terminal myristoyl cap to stabilize this inactive state, ABL2 relies on alternative regulatory interactions—likely involving its C-terminal cytoskeletal binding regions—to modulate kinase activity (hoj2020thecharacterizationofa pages 31-37, jones2020allostericinhibitionof pages 1-5).

## 6. Regulation

The activity of ABL2 is tightly controlled by a combination of intramolecular and intermolecular regulatory mechanisms. Autoinhibition in ABL family kinases is mediated by intramolecular interactions involving the SH3 and SH2 domains along with the kinase domain; in ABL1, the N-terminal myristoyl modification further reinforces this inhibition, whereas ABL2, lacking the myristoylated N-terminal cap, is thought to exhibit a higher basal activity and distinct regulatory dynamics (aleem2015constitutiveactivityin pages 10-11, mayro2022thecharacterizationofa pages 17-24).

Activation of ABL2 is achieved through conformational changes induced by phosphorylation. Auto-phosphorylation within the activation loop—at a residue analogous to ABL1 Tyr412—serves as a key switch that transitions the enzyme from an autoinhibited (‘closed’) to an active (‘open’) conformation, thereby allowing substrate access (aleem2015constitutiveactivityin pages 4-6, shah2018thesrcmodule pages 19-20). In addition, trans-phosphorylation by upstream kinases, including members of the Src family, can further enhance ABL2 activity. Regulatory phosphorylation events may also occur on residues outside the activation loop, contributing to changes in localization or interactions with adaptor proteins (luttman2021roleofthe pages 1-2, abrishami2018generationofsynthetic pages 25-29).

A notable aspect of ABL2 regulation is its ability to autophosphorylate and concurrently modulate its own inhibitory mechanisms. For instance, ABL2 has been shown to phosphorylate its inhibitor ABI1, thereby providing a feedback loop that affects its own kinase activity (aleem2015constitutiveactivityin pages 10-11, wu2021plateletderivedgrowthfactor pages 102-105). This autoregulatory capacity, together with interactions mediated by its SH2 and SH3 domains, enables ABL2 to integrate diverse extracellular stimuli—such as growth factors, adhesion signals, oxidative stress, and pathogen-derived cues—into precise changes in kinase activity and downstream signaling output (mayro2022thecharacterizationof pages 24-29, taft2017ayeastbasedassay pages 30-35).

## 7. Function

ABL2 is a multifunctional non-receptor tyrosine kinase critically involved in regulating cytoskeletal dynamics, cellular adhesion, motility, and receptor endocytosis. Functionally, ABL2 phosphorylates key components of the cell’s cytoskeletal network, thereby coordinating actin filament bundling and microtubule stabilization. For example, phosphorylation of MYH10 modulates actomyosin contractility, while modification of CTTN influences signaling cascades related to cell migration (Protein Function information, wu2021plateletderivedgrowthfactor pages 102-105).

In the context of adhesive signaling, ABL2 phosphorylates adaptor proteins such as CRK and CRKL, as well as regulatory molecules like DOK1 and ARHGAP35. Specifically, adhesion-dependent phosphorylation of ARHGAP35 promotes its interaction with RASA1, facilitating its recruitment to the cell periphery where it negatively regulates RHO activity—a critical step in the modulation of focal adhesion turnover and cell migration (Protein Function information, mayro2022thecharacterizationofa pages 17-24). These events underscore the role of ABL2 in the dynamic regulation of cell adhesion and motility, processes that are essential not only for normal cellular function but also for pathological states such as cancer metastasis (hoj2020thecharacterizationof pages 31-37, marco2020studyofthea pages 42-47).

Beyond its roles in cytoskeletal remodeling, ABL2 contributes to receptor endocytosis by phosphorylating receptor tyrosine kinases (e.g., PDGFRB) and endocytic regulators such as RIN1. This regulation integrates extracellular signals with the internalization and trafficking of receptors, thereby modulating downstream signal transduction pathways (wang2015theemergingrole pages 2-4, abrishami2018generationofsynthetic pages 25-29). In the nervous system, ABL2 is implicated in the regulation of neurotransmission through phosphorylation of synaptic proteins, thus affecting synaptic plasticity and neuronal communication (wu2021plateletderivedgrowthfactor pages 10-16).

Additionally, ABL2 is hijacked by various pathogens during infection, as it plays a role in reorganizing the host cell’s actin cytoskeleton to facilitate intracellular movement and cell exit. This function highlights its integration into multiple signaling networks that govern immune cell migration and pathogen-host interactions (mayro2022thecharacterizationof pages 136-140, moharram2021roleofflt3 pages 90-95). Moreover, through its substrate interactions and self-regulatory autophosphorylation, ABL2 overlaps functionally with ABL1 in pathways that regulate cell growth and survival, although each paralog also exhibits specific roles based on their distinct domain structures and subcellular localizations (aleem2015constitutiveactivityin pages 8-10, luttman2021roleofthe pages 10-11).

## 8. Other Comments

ABL2 is currently an active target of research due to its involvement in cell migration, cytoskeletal remodeling, and oncogenic signaling. Its role in modulating chemokine-mediated T-cell migration, polarization, and homing to lymphoid tissues further underscores its importance in immune cell signaling (Protein Function information). In the context of cancer, aberrant activation of ABL2 has been linked to aggressive tumor invasiveness and metastasis, particularly in solid tumors such as breast and renal carcinomas (marco2020studyofthea pages 42-47, moharram2021roleofflt3 pages 90-95). Inhibitors designed against ABL kinases—originally discovered in the context of BCR-ABL fusion proteins in chronic myelogenous leukemia—have spurred interest in developing drugs that can selectively target ABL2’s unique regulatory and cytoskeletal functions. Experimental inhibitors and drug candidates that block ABL kinase activity are being evaluated for their effectiveness in mitigating invasive cell behavior and drug resistance associated with elevated ABL2 activity (wang2015theemergingrole pages 2-4, wu2021plateletderivedgrowthfactor pages 102-105).

Recent advances in structural studies and biochemical assays continue to elucidate the functional distinctions between ABL1 and ABL2. For instance, alternative splicing of the first exon gives rise to multiple isoforms, which differ in regulatory sequences such as N-terminal myristoylation signals and nuclear localization motifs; while ABL1 isoforms can translocate to the nucleus, ABL2 isoforms are largely confined to the cytoplasm, directing their functional specialization toward actin and microtubule dynamics (abrishami2018generationofsynthetic pages 25-29, taft2017ayeastbasedassay pages 26-30).

Additionally, the interplay between ABL2 and its substrates is a subject of intense investigation, with recent studies focusing on how phosphorylation of cytoskeletal regulators and adaptor proteins translates into tangible changes in cell motility and adhesion (hoj2020thecharacterizationof pages 31-37, lyu2022abl2regulatesmicrotubule pages 35-40). Given its multifunctional roles, ABL2 also functions as a node for integrating signals from growth factor receptors, integrins, and other stimuli that converge on the actin cytoskeleton, making it a promising target for therapeutic intervention in both cancer and infectious diseases (mayro2022thecharacterizationof pages 24-29, ablishami2018generationofsynthetic pages 20-25).

Overall, the ongoing research into ABL2’s regulatory mechanisms, interaction networks, and contributions to pathological conditions continues to expand our understanding of its evolutionary conserved role within the tyrosine kinase superfamily.

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