1. Phylogeny  
   Tyrosine‐protein kinase HCK belongs to the Src family of non‐receptor tyrosine kinases, which is a highly conserved family present in all vertebrates and many eukaryotic organisms. HCK is evolutionarily related to other Src family kinases such as c‐Src, Lyn, Lck, Fyn, Fgr, Blk, Yes, and Yrk, and within the Src family it is often assigned to the SrcB subfamily, distinguished by its preferential expression in hematopoietic lineages (sicheri1997structuresofsrcfamily pages 1-2, banavali2007anatomyofa pages 1-2). Orthologs of HCK have been identified across mammalian species, and its conserved modular architecture—including the SH4, unique, SH3, SH2, catalytic kinase domain, and a C‐terminal regulatory tail—is emblematic of the evolutionary trajectory of this kinase family from a common eukaryotic ancestor (xu1997threedimensionalstructureof pages 7-8, yang2016hckisa pages 16-17).
2. Reaction Catalyzed  
   HCK catalyzes the phosphorylation of tyrosine residues on substrate proteins by transferring the γ‐phosphate group from ATP to the hydroxyl group of a tyrosine residue. The overall chemical reaction can be represented as:  
   ATP + Protein–L‑tyrosine → ADP + Protein–O‑phosphotyrosine + H⁺ (alvarado2010crystalstructureof pages 1-2, english1996expressionofthe pages 1-2).
3. Cofactor Requirements  
   The kinase activity of HCK is dependent on the presence of divalent metal cations, most notably Mg²⁺, which are essential for the proper binding of ATP in the catalytic site and for the phosphoryl transfer reaction (johnson2000modulationofthe pages 1-2, pond2020membraneanchoringof pages 1-2).
4. Substrate Specificity  
   HCK phosphorylates tyrosine residues in multiple substrate proteins, a function that is largely determined by both the catalytic kinase domain and the substrate-recruiting roles of its regulatory SH2 and SH3 domains. Its substrate repertoire includes proteins involved in signal transduction cascades in hematopoietic cells such as CBL, ADAM15, BCR, ELMO1, FCGR2A, GAB1, GAB2, RAPGEF1, STAT5B, TP73, VAV1, and WAS (alvarado2010crystalstructureof pages 1-2, corey1999srcrelatedproteintyrosine pages 10-11, yokoyama2005identificationoftyrosine pages 1-2). Although the precise consensus motif recognized by HCK has not been defined in detail within the provided sources, the intrinsic substrate specificity of Src family kinases suggests a preference for sequences that accommodate interactions with the active site as well as secondary contacts with the SH2/SH3 domains (yokoyama2005identificationoftyrosine pages 10-11, banavali2007anatomyofa pages 15-16).
5. Structure  
   HCK displays a modular domain organization that underpins its function and regulation. At the N-terminus, HCK possesses an SH4 domain carrying signals for myristoylation and, in some isoforms, palmitoylation; these lipid modifications anchor the kinase to membranes, thereby facilitating interaction with membrane-bound receptors (pond2020membraneanchoringof pages 1-2). Adjacent to the SH4 domain is a unique region that is less conserved among Src family members yet contributes to isoform-specific functions—HCK is expressed in two major isoforms, commonly referred to as p59-HCK and p61-HCK, which differ by N-terminal residues and lipid modifications (english1996expressionofthe pages 1-2, gibson1993identificationcloningand pages 12-12). Following the unique region are the SH3 and SH2 domains; the SH3 domain binds to proline-rich polyproline type II helices typically found in the SH2-kinase interdomain linker, while the SH2 domain recognizes phosphorylated tyrosine motifs, including those present in the C-terminal tail of HCK (alvarado2010crystalstructureof pages 1-2, lerner2005activationofthe pages 1-2). The catalytic kinase domain, which occupies the central region of the protein, is bilobed and contains the highly conserved ATP-binding pocket, an activation loop that harbors the autophosphorylation site (analogous to Tyr416 in Src), and structural elements such as the glycine-rich loop, a critical C-helix, and a hydrophobic spine that are responsible for the enzyme’s catalytic activity and conformational switching between active and inactive states (selzer2024cocrystallizationofthe pages 1-2, sicheri1997structuresofsrcfamily pages 3-5). The C-terminal tail concludes the kinase and contains a regulatory tyrosine residue (Tyr501 in HCK, analogous to Tyr527 in c-Src) that, when phosphorylated, interacts intramolecularly with the SH2 domain to maintain the kinase in an autoinhibited conformation (alvarado2010crystalstructureof pages 1-2, lerner2005activationofthe pages 1-1, sicheri1997structuresofsrcfamily pages 6-8).
6. Regulation  
   HCK is regulated through a finely tuned interplay of intramolecular domain interactions and phosphorylation events. In the autoinhibited state, the SH2 domain binds to a phosphorylated tyrosine in the C-terminal tail (Tyr501), and the SH3 domain binds to a proline-rich sequence found in the SH2-kinase linker; these interactions stabilize a conformation in which the catalytic domain is rendered inactive by displacement of the αC-helix and misalignment of critical catalytic residues (lerner2005activationofthe pages 1-2, sicheri1997structuresofsrcfamily pages 6-8). Full activation of HCK requires disruption of these inhibitory intramolecular interactions. Activation is achieved by displacement of the SH2 or SH3 contacts—such as through binding of external ligands (for example, the HIV-1 Nef protein is known to interact with the SH3 domain and activate HCK)—or through dephosphorylation of the inhibitory C-terminal tyrosine by phosphatases, resulting in a conformational change that permits autophosphorylation of the activation loop (Tyr416) and alignment of the ATP-binding pocket for catalysis (alvarado2010crystalstructureof pages 1-2, lerner2005activationofthe pages 6-7, johnson2000modulationofthe pages 7-8). In addition to these intramolecular events, regulatory kinases such as Csk and its homolog Chk phosphorylate the C-terminal tail to promote the inactive conformation, while intermolecular interactions and autophosphorylation events further modulate HCK’s catalytic output (advani2017cskhomologouskinase(chk) pages 8-10, johnson2000modulationofthe pages 2-2).
7. Function  
   HCK is predominantly expressed in hematopoietic cells including neutrophils, monocytes, macrophages, and mast cells, where it plays an instrumental role in the regulation of innate immune responses. Acting downstream of cell surface receptors—which include Fcγ receptors (FCGR1A, FCGR2A), cytokine receptors such as those for IFNG, IL2, IL6, and IL8, as well as integrins (ITGB1 and ITGB2)—HCK transduces signals that regulate phagocytosis, cell adhesion, and migration (alvarado2010crystalstructureof pages 1-2, english1996expressionofthe pages 1-2). During phagocytosis, HCK is involved in mobilization of secretory lysosomes, degranulation, and activation of NADPH oxidase, which together contribute to the respiratory burst essential for microbial killing. Moreover, HCK phosphorylates a panel of substrates that includes CBL, ADAM15, BCR, ELMO1, GAB1, GAB2, RAPGEF1, STAT5B, TP73, VAV1, and WAS, thereby modulating downstream signaling pathways that affect cell survival, proliferation, and cytoskeletal reorganization (alvarado2010crystalstructureof pages 1-2, yokoyama2005identificationoftyrosine pages 10-11). In addition, HCK participates in the reorganization of the actin cytoskeleton, facilitating podosome formation and membrane protrusion development, and it has been implicated in the inhibition of TP73-mediated transcription and apoptosis, thereby influencing cell fate decisions (klejman2002thesrcfamily pages 6-7, english1996expressionofthe pages 7-7).
8. Other Comments  
   HCK is known by several alternative names including Hematopoietic cell kinase, p59-HCK/p60-HCK, and p61-HCK, reflecting the existence of multiple isoforms generated by alternative translational initiation (english1996expressionofthe pages 1-2, gibson1993identificationcloningand pages 12-12). Inhibitors that target Src family kinases, such as the ATP-competitive inhibitor A-419259 and derivative compounds like NaPP1 in chemical genetics studies, have been used to dissect the functional role of HCK in various signaling pathways, including its contribution to drug resistance in chronic myelogenous leukemia (penedumitrescu2008aninhibitorresistantmutant pages 2-4, penedumitrescu2010expressionofa pages 9-10). In particular, overexpression of HCK has been implicated in the resistance of CML cells to imatinib therapy by sustaining phosphorylation events that maintain downstream signaling through pathways such as those involving STAT5 and ERK (penedumitrescu2010expressionofa pages 5-6, penedumitrescu2010expressionofa pages 9-9). Furthermore, HCK inhibition is being explored as a therapeutic strategy in the context of certain hematopoietic malignancies and in HIV infection, where viral proteins such as Nef interact with its regulatory domains to dysregulate activity (alvarado2010crystalstructureof pages 1-2, lerner2005activationofthe pages 1-1). The structural basis of HCK’s activation by membrane anchoring and domain displacement, as revealed by crystallographic studies, also suggests unique sites for allosteric inhibition that may improve therapeutic selectivity (selzer2024cocrystallizationofthe pages 7-8).
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