1. Phylogeny  
   EPHA5 is a receptor tyrosine kinase that belongs to the highly diversified Eph receptor family, which is the largest subgroup of receptor tyrosine kinases in vertebrates (gaitanos2015theephreceptor pages 1-4, lackmann2008ephaprotein pages 1-2). Within this family, receptors are divided into two subclasses—EphA and EphB—with EPHA5 positioned in the EphA subclass that preferentially binds glycosylphosphatidylinositol (GPI)‐anchored ephrin-A ligands (lackmann2008ephaprotein pages 2-3, anderton2021theroleof pages 2-4). Orthologs of EPHA5 have been identified across mammals and other vertebrate species, underscoring its evolutionary conservation and its fundamental role in developmental signal transduction (gaitanos2015theephreceptor pages 1-4, lackmann2008ephaprotein pages 1-2). The diversification of this receptor subfamily parallels the increasing complexity of tissue patterning and cell–cell communication in vertebrates (lackmann2008ephaprotein pages 2-3, anderton2021theroleof pages 2-4).
2. Reaction Catalyzed  
   EPHA5 catalyzes a canonical receptor tyrosine kinase reaction whereby a phosphate group is transferred from ATP to the hydroxyl group of specific tyrosine residues present either within the receptor itself (autophosphorylation) or on downstream effector proteins. The overall chemical reaction can be summarized as:  
     ATP + [protein]–tyrosine → ADP + [protein]–phosphotyrosine + H⁺  
   This phosphorylation event is central to the initiation and propagation of forward signaling cascades that occur after ligand-induced receptor clustering and activation (gaitanos2015theephreceptor pages 45-50, gaitanos2015theephreceptor pages 94-96).
3. Cofactor Requirements  
   The kinase activity of EPHA5 is dependent upon the presence of divalent metal ions, with Mg²⁺ being the critical cofactor required to coordinate ATP binding in the kinase catalytic domain. This magnesium ion assists in the proper positioning of the ATP molecule within the active site, thereby facilitating the phosphate transfer reaction (lackmann2008ephaprotein pages 3-4).
4. Substrate Specificity  
   EPHA5 displays substrate specificity that is characteristic of receptor tyrosine kinases. Its catalytic activity leads to the phosphorylation of tyrosine residues both within its own intracellular regions (autophosphorylation sites located in the juxtamembrane and kinase domains) and on downstream substrates that are involved in signaling pathways associated with cytoskeletal organization, cell adhesion, and migration. Although a definitive consensus phosphorylation motif for EPHA5 has not been fully delineated in the available literature, its substrate recognition is tied to the structural layout of its intracellular domain and resembles that observed in other EphA receptors (gaitanos2015theephreceptor pages 73-75, gaitanos2015theephreceptor pages 94-96, anderton2021theroleof pages 2-4).
5. Structure  
   EPHA5 exhibits the modular domain architecture typical of Eph receptors. Its extracellular region is composed of an N‐terminal ligand-binding domain (LBD) that mediates the interaction with GPI‐anchored ephrin-A ligands—among which EFNA5 is identified as the most likely functional partner—followed by a cysteine-rich region and two fibronectin type III (FN3) domains that contribute to ligand specificity and receptor oligomerization (gaitanos2015theephreceptor pages 73-75, barquilla2015ephreceptorsand pages 1-3). This is succeeded by a single transmembrane helix that anchors the receptor in the plasma membrane. The intracellular region of EPHA5 comprises a juxtamembrane segment containing regulatory tyrosine residues that are critical for initiating receptor autophosphorylation. The central catalytic tyrosine kinase domain follows, which is responsible for the phosphorylation reaction described above. C-terminal to the kinase domain, EPHA5 contains a sterile-α motif (SAM) and a PDZ-binding motif; these domains serve as platforms for protein–protein interactions, facilitating the recruitment of downstream signaling molecules and contributing to the assembly of multimolecular signaling complexes (gaitanos2015theephreceptor pages 75-78, gaitanos2015theephreceptor pages 88-90). Structural investigations—including crystallographic studies of related Eph receptors and complementary AlphaFold model predictions—support a conserved three-dimensional organization featuring essential motifs such as the activation loop, the catalytic (hydrophobic) spine, and a conserved C-helix that are critical for kinase function (nikolov2007crystalstructureof pages 1-2, sahoo2021structuralandfunctional pages 1-2). Posttranslational modifications, notably tyrosine phosphorylation and N-glycosylation, have been documented and are important for modulating receptor stability and function (gaitanos2015theephreceptor pages 73-75).
6. Regulation  
   Regulation of EPHA5 activity is achieved through multiple interrelated mechanisms. Ligand binding to the extracellular domain triggers receptor dimerization and the formation of higher-order clusters, events that promote autophosphorylation of key tyrosine residues in the juxtamembrane and catalytic regions, and thereby activate the intrinsic kinase activity (gaitanos2015theephreceptor pages 45-50, gaitanos2015theephreceptor pages 88-90). In addition, EPHA5 transcription is modulated by second messenger pathways; for example, its expression is regulated via cAMP‐dependent pathways, and epigenetic modifications such as promoter methylation have been observed in certain cancer contexts, leading to downregulation of its mRNA levels (gaitanos2015theephreceptor pages 45-50, anderton2021theroleof pages 5-6, zhang2021theexpressionprofile pages 10-12). Downstream of receptor activation, EPHA5 regulates small GTPase signaling (e.g., activation of Cdc42 via PI3K) which influences cytoskeletal dynamics and cell adhesion (gaitanos2015theephreceptor pages 45-50). In addition, receptor internalization, often mediated by ligand-induced endocytosis and proteolytic cleavage, serves to modulate the duration and intensity of EPHA5 signaling (gaitanos2015theephreceptor pages 70-73, barquilla2015ephreceptorsand pages 29-30).
7. Function  
   EPHA5 functions as a key mediator of contact-dependent cell–cell communication by virtue of its receptor tyrosine kinase activity. In the nervous system, EPHA5 is expressed predominantly in brain tissues—particularly within cortical neurons and cerebellar Purkinje cells—and plays critical roles in axon guidance during embryonic development (gaitanos2015theephreceptor pages 75-78, anderton2021theroleof pages 2-4). It facilitates the establishment of retinotectal, entorhino-hippocampal, and hippocamposeptal connections by modulating forward signaling upon binding to its primary ligand, EFNA5 (gaitanos2015theephreceptor pages 73-75, gaitanos2015theephreceptor pages 1-4). In the adult brain, EPHA5 contributes to synaptic plasticity by regulating synaptogenesis, thereby impacting the formation and remodeling of functional neuronal circuits (gaitanos2015theephreceptor pages 88-90, anderton2021theroleof pages 2-4). Outside the nervous system, EPHA5 is involved in the regulation of pancreatic islet cell function; its interaction with EFNA5 is reported to mediate communication between islet cells, thereby contributing to the regulation of glucose-stimulated insulin secretion (gaitanos2015theephreceptor pages 23-26). Collectively, these functions underscore the dual developmental and homeostatic roles of EPHA5 in both the central nervous system and metabolic regulation (anderton2021theroleof pages 5-6, gaitanos2015theephreceptor pages 31-33).
8. Other Comments  
   At present, no inhibitors that are specific for EPHA5 have been reported; however, several experimental approaches have employed broader Eph receptor inhibitors—ranging from small molecules and peptides to antibody-based agents—to modulate Eph receptor signaling in cancer and neural regeneration contexts (pasquale2010ephreceptorsand pages 2-4, anderton2021theroleof pages 5-6). EPHA5 is subject to epigenetic regulation, and its promoter methylation has been observed in certain breast cancer samples, suggesting a potential tumor-suppressive role in that context (anderton2021theroleof pages 5-6, zhang2021theexpressionprofile pages 12-13). In addition, altered expression and mutation of EPHA5 may have implications in neurodevelopmental disorders, although the precise disease associations remain to be comprehensively defined (anderton2021theroleof pages 9-10, papadakos2023unravelingthesignificance pages 2-4). Furthermore, in pancreatic islet biology, EPHA5-mediated intercellular signaling is considered important for the regulation of insulin secretion, indicating a role in metabolic homeostasis (gaitanos2015theephreceptor pages 23-26).
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