1. Phylogeny  
   Ephrin type-A receptor 2 (EPHA2) is a member of the Eph receptor tyrosine kinase family, which comprises the largest subgroup of receptor tyrosine kinases in vertebrates. EPHA2 falls within the EphA subclass that preferentially binds glycosylphosphatidylinositol (GPI)‐anchored ephrin-A ligands. Orthologs of EPHA2 have been identified in a broad spectrum of vertebrate species, and its domain organization and sequence conservation indicate that it has been maintained throughout evolution as a critical mediator of contact‐dependent cellular communication (lackmann2008ephaprotein pages 2-3). Comparative analyses reveal that the Eph receptors, including EPHA2, share a conserved extracellular ligand‐binding module and cytoplasmic kinase domain that are also detectable in orthologous proteins from fish, amphibians, birds, and mammals (lackmann2008ephaprotein pages 3-4). In evolutionary terms, EPHA2 is part of an ancient signaling system that likely emerged during early vertebrate evolution, with the diversification of Eph receptors paralleling the increased complexity of tissue organization in multicellular organisms (rasool2024masterregulatorsof pages 1-2). The conservation of key residues in the ligand‐binding and kinase domains underscores the essential function of EPHA2 in guiding processes such as neural map formation and tissue patterning. This evolutionary stability also reflects its significant role in both developmental processes and adult physiological functions, and its orthologs continue to provide valuable models for studying receptor function across species (lackmann2008ephaprotein pages 2-3, rasool2024masterregulatorsof pages 1-2).
2. Reaction Catalyzed  
   EPHA2 functions as a receptor tyrosine kinase that catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins. In this canonical reaction, ATP and a target protein containing an accessible L‐tyrosine residue serve as substrates and are converted into ADP and a phosphorylated protein derivative along with the release of a proton. This phosphorylation event is critical for the propagation of downstream signaling and is initiated through ligand‐induced dimerization and autophosphorylation of EPHA2 (ziemiecki2003ephandephrin pages 1-3). The reaction can be summarized as follows: ATP + protein–L‐tyrosine → ADP + protein–L‐tyrosine‐phosphate + H⁺, a transformation that is foundational for its role in bidirectional signal transduction between adjacent cells (lackmann2008ephaprotein pages 13-14).
3. Cofactor Requirements  
   The kinase activity of EPHA2 is dependent on the presence of ATP as a phosphate donor, and like many kinases, its catalytic function requires divalent metal ions, most notably Mg²⁺. The magnesium ion coordinates with ATP in the nucleotide‐binding site, thereby facilitating the transfer of the γ‐phosphate group to the substrate tyrosine residue. This cofactor requirement is critical for the proper orientation of the substrate and for the stabilization of transition states during catalysis (xu2015identifyingthreedimensionalstructures pages 7-9).
4. Substrate Specificity  
   EPHA2 exhibits substrate specificity characteristic of receptor tyrosine kinases by phosphorylating tyrosine residues in target proteins, including sites within its own cytoplasmic domain through autophosphorylation. Key autophosphorylation sites, such as Tyr588 and Tyr594, are highly conserved across Eph receptors and serve as docking sites for downstream signaling molecules equipped with SH2 domains (xu2015identifyingthreedimensionalstructures pages 7-9). In addition to these canonical autophosphorylation events, EPHA2 has been reported to be phosphorylated on other residues, including Y772 and S897, which participate in modulating its interaction with adaptor proteins and affecting signaling outcomes (singh2018theepha2receptor pages 10-11). Although a strict consensus sequence for EPHA2 substrates is less clearly delineated than that seen for some serine/threonine kinases, the receptor’s propensity to phosphorylate tyrosine residues in motifs flanked by hydrophobic and conserved polar residues has been inferred from structural and mutagenesis studies (ziemiecki2003ephandephrin pages 1-3).
5. Structure  
   EPHA2 is a transmembrane receptor that displays a modular architecture typical of Eph receptor tyrosine kinases. The extracellular portion comprises an N‐terminal ligand‐binding domain that exhibits an immunoglobulin-like fold, followed by a cysteine‐rich region and two fibronectin type III repeats, which together mediate high affinity binding to membrane‐bound ephrin-A ligands (ziemiecki2003ephandephrin pages 1-3). This extracellular assembly is responsible for initiating contact‐dependent cell signaling through its ability to interact with ephrin molecules presented on adjacent cells. A single transmembrane helix spans the lipid bilayer, anchoring the receptor into the plasma membrane. The intracellular region of EPHA2 contains a juxtamembrane segment that becomes phosphorylated upon activation, a well‐conserved tyrosine kinase domain that catalyzes the phosphorylation reaction on tyrosine residues, and C-terminal regulatory modules comprising a sterile alpha motif (SAM) and a PDZ-binding motif that facilitate protein–protein interactions and receptor clustering (lackmann2008ephaprotein pages 13-14, ziemiecki2003ephandephrin pages 1-3).  
   High‐resolution structural studies, including X-ray crystallography of the ligand-binding domain in complex with synthetic agonistic peptides, have elucidated the molecular determinants for ephrin binding. These studies have shown that specific salt bridges and hydrogen bonds—for example, those formed between peptide arginine residues and specific glutamate residues on EPHA2—are critical for high‐affinity interactions, with designed peptides achieving nanomolar affinity (gambini2018structurebaseddesignof pages 7-9, gambini2018structurebaseddesignof pages 9-11). The kinase domain has been similarly characterized by co-crystallization with clinical kinase inhibitors. Structural analyses have defined key motifs, including a Gly-rich loop, the αC helix, a gatekeeper residue (Thr692), the catalytic loop containing the HRD motif, and the activation loop with the DFG motif. Notably, EPHA2 can adopt distinct conformations, such as the DFG-in and DFG-out states, which are of importance during inhibitor binding as identified in structural biology and chemical proteomics studies (heinzlmeir2016chemicalproteomicsand pages 21-25, heinzlmeir2016chemicalproteomicsand pages 34-39, xu2015identifyingthreedimensionalstructures pages 7-9).  
   These detailed structural insights, which combine experimental X-ray data with molecular modeling, establish the basis for the rational design of synthetic agonists or inhibitors that selectively modulate EPHA2 activity. The unique spatial arrangement of its extracellular and intracellular domains facilitates both adhesive interactions at the cell surface and the initiation of robust intracellular phosphorylation cascades (gambini2018structurebaseddesignof pages 7-9, gambini2018structurebaseddesignof pages 22-24).
6. Regulation  
   EPHA2 regulation is mediated by multiple post-translational modifications and spatial control mechanisms that ensure precise modulation of its signaling functions. Activation of EPHA2 occurs upon binding to ephrin-A ligands, primarily ephrin-A1, which induces receptor clustering, leading to autophosphorylation of key tyrosine residues within the juxtamembrane region and the kinase domain. This autophosphorylation event not only relieves autoinhibitory constraints but also creates binding sites for SH2 domain-containing adaptor proteins, thereby propagating downstream signaling (gambini2018structurebaseddesignof pages 1-2, xu2015identifyingthreedimensionalstructures pages 7-9).  
   In addition to ligand-induced autophosphorylation, EPHA2 is subject to regulation by serine phosphorylation. Phosphorylation of serine residues such as S897 has been documented as part of a mechanism that modulates receptor function independently of ligand binding, and such serine phosphorylation events are mediated by serine kinases including RSK and PKA (singh2018theepha2receptor pages 10-11). Furthermore, receptor ubiquitination plays a critical role in determining the fate of EPHA2 after activation. Upon ligand engagement and subsequent clustering, the receptor becomes a substrate for ubiquitin ligases such as c-Cbl. Ubiquitination directs EPHA2 toward endocytic pathways, resulting in its internalization and either recycling or degradation via lysosomes or multivesicular bodies (sabet2015ubiquitinationswitchesepha2 pages 1-2, sabet2015ubiquitinationswitchesepha2 pages 9-9).  
   Moreover, spatial regulation via endosomal trafficking mechanisms further refines EPHA2 signaling outputs. For instance, the receptor may reside transiently in Rab11-positive recycling endosomes, where its phosphorylation status is modulated by associated protein tyrosine phosphatases such as PTP1B. The balance between kinase activity and phosphatase-mediated dephosphorylation provides a dynamic means to switch off the signal after an appropriate duration (heinzlmeir2016chemicalproteomicsand pages 1-5, sabet2015ubiquitinationswitchesepha2 pages 1-2).  
   This network of regulatory events—including ligand-induced clustering and autophosphorylation, serine phosphorylation, and ubiquitination-dependent trafficking—ensures that EPHA2 signaling is both spatially and temporally controlled, and it allows the receptor to mediate distinct biological outcomes depending on the cellular context (gambini2018structurebaseddesignof pages 1-2, singh2018theepha2receptor pages 11-11).
7. Function  
   EPHA2 functions as a receptor tyrosine kinase that plays a central role in mediating contact-dependent bidirectional signaling between adjacent cells. Upon binding to membrane-bound ephrin-A ligands, particularly ephrin-A1, EPHA2 initiates “forward signaling” pathways in the receptor-expressing cell while simultaneously triggering “reverse signaling” cascades in the ligand-bearing cell. Through these mechanisms, EPHA2 regulates multiple cellular processes including cell adhesion, migration, proliferation, and differentiation (gambini2018structurebaseddesignof pages 1-2, singh2018theepha2receptor pages 1-2).  
   Forward signaling initiated by EPHA2 has been shown to influence integrin-mediated adhesion and to modulate the assembly of focal adhesions, thereby affecting cell shape and motility. The interaction of EPHA2 with downstream effectors such as Rho family GTPases (including Rac1, RhoA, and Cdc42) links receptor activation to reorganization of the actin cytoskeleton, which is critical for promoting cell migration and regulating tissue architecture (rasool2024masterregulatorsof pages 7-8, rasool2024masterregulatorsof pages 8-9). In addition, EPHA2 is capable of modulating mitogen-activated protein kinase (MAPK) pathways, in particular, its activation leads to inhibition of the ERK1/ERK2 cascade, which in turn can reduce cell proliferation and promote differentiation in certain cellular contexts (gambini2018structurebaseddesignof pages 1-2).  
   Beyond its roles in cell adhesion and migration, EPHA2 has established functions in developmental processes. It participates in the patterning of fetal tissues, contributes to angiogenesis by influencing endothelial cell behavior, and is implicated in early hindbrain development, where tightly regulated signaling is essential for proper anatomical segmentation (rasool2024masterregulatorsof pages 7-8, rasool2024masterregulatorsof pages 8-9). In the context of cancer, EPHA2 demonstrates a duality in function. Unligated EPHA2 may exhibit pro-oncogenic behavior, promoting chemotactic migration and contributing to drug resistance, whereas engagement by ephrin-A1 or synthetic agonistic peptides can trigger receptor internalization and degradation, thus activating tumor-suppressive signaling pathways (gambini2018structurebaseddesignof pages 1-2, singh2018theepha2receptor pages 1-2).  
   The downstream substrates of EPHA2 include a variety of adaptor and scaffold proteins that, upon binding to phosphorylated tyrosine residues on the receptor, propagate signals that regulate cellular proliferation and differentiation. Interaction with desmoglein-1 (DSG1) has been implicated in modulating cell–cell adhesion and epithelial differentiation, while cross-talk with integrin signaling further orchestrates cytoskeletal dynamics necessary for directed cell migration (gambini2018structurebaseddesignof pages 1-2, singh2018theepha2receptor pages 10-11).  
   Collectively, the multifaceted functions of EPHA2 underscore its critical role in orchestrating cellular responses during both development and disease. Its capacity to drive alterations in cell adhesion, movement, and growth renders it a focal point in studies of tissue morphogenesis as well as in the investigation of cancer progression and metastasis (gambini2018structurebaseddesignof pages 1-2, rasool2024masterregulatorsof pages 7-8).
8. Other Comments  
   A number of chemical biology and proteomics studies have contributed to the understanding of EPHA2 as a drug target and have advanced the development of specific modulators. Synthetic peptide agonists designed using structure-based approaches have demonstrated nanomolar affinity for EPHA2 and are capable of inducing receptor autophosphorylation and subsequent internalization, leading to reduced cell migration and invasive behavior (gambini2018structurebaseddesignof pages 19-22, gambini2018structurebaseddesignof pages 7-9). These agents mimic the natural ligand ephrin-A1 and have been used as tools to interrogate the receptor’s function in various cancer models.  
   Chemical proteomics studies have further revealed that a diverse range of clinical kinase inhibitors exhibit sub-micromolar binding affinities toward EPHA2. Detailed crystallographic analyses of the EPHA2 kinase domain have elucidated interactions within the ATP binding pocket, categorizing residues into key, scaffold, potency, and selectivity groups. Such studies provide a framework for the rational design of selective EPHA2 inhibitors that target both its kinase domain and its ligand-binding domain (heinzlmeir2016chemicalproteomicsand pages 27-29, heinzlmeir2016chemicalproteomicsand pages 29-34).  
   Clinically, aberrant expression and dysregulated signaling of EPHA2 have been linked to unfavorable outcomes in several cancers, including melanoma, breast, lung, and pancreatic carcinomas. Overexpression of EPHA2 is associated with enhanced tumor cell migration, invasion, and the development of chemoresistance, thereby establishing it as a candidate for targeted cancer therapies. In addition, EPHA2 has been implicated in UV radiation-induced apoptosis and may have a ligand-independent stimulatory effect on chemotactic cell migration, further highlighting its complex role in both normal physiology and disease (gambini2018structurebaseddesignof pages 1-2, singh2018theepha2receptor pages 1-2).  
   The identification of specific post-translational modifications, such as the phosphorylation of S897 and the ubiquitination that marks the receptor for internalization, are central to the modulation of EPHA2 signaling. These modifications represent potential intervention points, and several inhibitors that target these regulatory processes are under preclinical evaluation.  
   Finally, the interplay between extracellular ligand binding, receptor dimerization, intracellular kinase activation, and subsequent regulatory modifications makes EPHA2 a paradigmatic example of how receptor tyrosine kinases integrate multiple layers of control to execute precise cellular responses. The robust structural and biochemical characterization of EPHA2 provided by recent studies lays the groundwork for future therapeutic strategies aimed at targeting its oncogenic as well as its developmental functions (sabet2015ubiquitinationswitchesepha2 pages 4-5, singh2018theepha2receptor pages 11-12, lackmann2008ephaprotein pages 10-11, ziemiecki2003ephandephrin pages 6-8).

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