1. Phylogeny  
   Ephrin type‐A receptor 3 (EPHA3) is a member of the Eph receptor subfamily of receptor tyrosine kinases (RTKs) that, as a whole, represents one of the largest groups within the human kinome. EPHA3 is classified within the EphA group that preferentially binds glycosylphosphatidylinositol‐anchored ephrin‐A ligands. Orthologs of EPHA3 have been identified in a wide range of species, including mammals (e.g., human and mouse) and other vertebrates, indicating strong evolutionary conservation of its ligand‐binding and signaling modules (forse2015distinctivestructureof pages 1-2, anderton2021theroleof pages 1-2). Comparative analyses of the human and murine kinomes place EPHA3 alongside other EphA receptors such as EphA2 and EphA4, all of which share a conserved extracellular domain architecture and intracellular catalytic core. Evolutionary studies indicate that the Eph receptor family arose early in chordate evolution and has since diversified to coordinate contact‐dependent cell–cell signaling. This evolutionary conservation mirrors the critical developmental roles these receptors play in processes such as neuronal patterning, vascular morphogenesis, and tissue boundary formation (anderton2021theroleof pages 2-4, arora2023ephreceptorsin pages 20-22).
2. Reaction Catalyzed  
   As a receptor tyrosine kinase, EPHA3 catalyzes the transfer of the γ‐phosphate from ATP to specific tyrosine residues on substrate proteins. The general reaction can be represented as:  
     ATP + [protein]–OH → ADP + [protein]–pTyr + H⁺  
   In the case of EPHA3, this phosphorylation reaction includes autophosphorylation of key tyrosine residues in the juxtamembrane and activation loop regions upon ligand (ephrin‐A) binding, as well as phosphorylation of downstream substrates that serve to recruit adapter proteins with SH2 domains (keane2012epha3asa pages 3-4).
3. Cofactor Requirements  
   The catalytic activity of EPHA3, like that of other protein kinases, is dependent on divalent cations. In particular, Mg²⁺ is required to coordinate ATP in the active site, thereby enabling efficient transfer of the phosphate group to the tyrosine residue of target substrates (arora2023ephreceptorsin pages 20-22).
4. Substrate Specificity  
   EPHA3 exhibits substrate specificity typical of receptor tyrosine kinases, phosphorylating tyrosine residues within specific sequence contexts. Autophosphorylation at conserved tyrosine residues in its juxtamembrane region creates docking sites for SH2‐domain–containing proteins. This phosphorylation event not only increases kinase catalytic activity but also defines the specificity toward downstream signaling proteins such as CrkII and Nck1, which possess binding motifs optimized for interactions with phosphorylated tyrosine sites (keane2012epha3asa pages 3-4). Although a well‐defined linear consensus motif for EPHA3 has not been fully delineated in the literature provided, its substrate recognition is thought to be governed in part by spatial presentation and the local amino acid context surrounding the phosphoacceptor tyrosine residue. The recruitment of downstream adaptors that mediate cytoskeletal reorganization and cellular adhesion supports a model in which EPHA3 exhibits selectivity for substrates involved in cell–cell communication and migration (lisabeth2012cancersomaticmutations pages 1-2, keane2012epha3asa pages 2-3).
5. Structure  
   EPHA3 is organized into several distinct domains that cooperate to effect ligand binding, receptor dimerization/oligomerization, catalytic activity, and downstream signaling. The large extracellular region comprises an N‐terminal ligand‐binding domain (LBD) of approximately 200 amino acids, which is responsible for binding promiscuous ephrin‐A ligands with a preferential affinity for ephrin‐A5. This LBD is followed by a cysteine‐rich region that includes a Sushi (complement control protein) domain and an epidermal growth factor (EGF)–like domain; these contribute to additional stabilizing interactions with the ligand and assist in receptor clustering. Adjacent to the extracellular portion, two fibronectin type III repeats further extend the receptor’s ectodomain, contributing to its structural integrity and overall binding surface (arora2023ephreceptorsin pages 4-5, forse2015distinctivestructureof pages 1-2).

Following the single transmembrane segment, EPHA3 contains an intracellular module that is critical for signal transduction. Immediately after the membrane, a short juxtamembrane region contains key regulatory tyrosine residues; phosphorylation of these residues is necessary for the release of autoinhibition and subsequent activation of the kinase domain. The kinase domain itself exhibits the canonical bilobal structure found in tyrosine kinases, with an N‐lobe composed predominantly of β‐sheet structure juxtaposed to a predominantly α-helical C‐lobe. The location of the activation loop within the kinase domain is a critical regulatory feature; its phosphorylation reorients structural elements such as the C-helix and hydrophobic spine, thereby aligning catalytic residues for efficient substrate turnover (keane2012epha3asa pages 3-4, lisabeth2012cancersomaticmutations pages 4-6).

Beyond the kinase domain, EPHA3 contains a sterile alpha motif (SAM) domain, which is implicated in mediating receptor oligomerization. This oligomerization is essential for the formation of higher-order complexes that enhance ligand binding and signal propagation, as observed in structural studies demonstrating a dual mode of ephrin-A5 interaction that induces a pronounced ligand tilt and increases the contact interface (forse2015distinctivestructureof pages 2-4, forse2015distinctivestructureof pages 9-12). A short PDZ-binding motif is located at the extreme C-terminal end of the receptor, allowing interaction with scaffold proteins that bring together multiple signaling components into functional complexes (arora2023ephreceptorsin pages 3-4).

Structural studies employing techniques such as X-ray crystallography and isothermal titration calorimetry have elucidated details of the EPHA3/ephrin-A5 complex. The ephrin-A5 ligand, upon binding to the LBD of EPHA3, exhibits a distinctive tilt relative to the receptor surface; this conformational change is believed to be critical for stabilizing interactions that facilitate receptor tetramerization and subsequent kinase activation. Key residues in the receptor–ligand interface, as identified in high-resolution structures, include those that form hydrogen bonds and salt bridges, contributing to a binding interface that is both extensive and distinct from other Eph/ephrin complexes (forse2015distinctivestructureof pages 1-2, forse2015distinctivestructureof pages 9-12).

Additionally, mutagenesis experiments have underscored the functional importance of these interface regions. Disruption of residues within the ligand-binding pocket or in the regions implicated in tetramer formation leads to significant decreases in ephrin-A5 binding affinity and impaired receptor activation, underscoring the integrated nature of EPHA3’s structural domains in mediating its biological function (forse2015distinctivestructureof pages 15-16, keane2012epha3asa pages 3-4).

1. Regulation  
   The activation and regulation of EPHA3 are mediated critically by ligand-induced receptor clustering and subsequent autophosphorylation. Binding of ephrin-A ligands, particularly ephrin-A5, triggers receptor dimerization and the formation of higher-order oligomers. This ligand-induced clustering relieves autoinhibitory constraints imposed by the unphosphorylated juxtamembrane region. Autophosphorylation of conserved tyrosine residues within this region and the activation loop is essential for full catalytic activation, as these phosphorylation events create docking sites for SH2 domain–containing adaptor proteins (keane2012epha3asa pages 2-3, lisabeth2012cancersomaticmutations pages 7-8).

In addition to autophosphorylation, EPHA3 regulation is modulated by the binding of protein tyrosine phosphatases, which can dephosphorylate key regulatory residues and thereby modulate receptor activity. Such phosphatases act in a feedback loop to reset the receptor’s activation state once appropriate signaling thresholds have been reached (keane2012epha3asa pages 13-14). Furthermore, the SAM and PDZ-binding domains of the receptor facilitate interactions with scaffold proteins, which help to spatially organize multiprotein complexes at the plasma membrane and can influence the amplitude and duration of downstream signaling.

Receptor internalization is another regulatory mechanism. Ligand binding and subsequent phosphorylation events promote endocytosis of the Eph/ephrin complex, a process that not only attenuates signaling at the cell surface but may also allow for signal propagation from endosomal compartments. In certain contexts, monoclonal antibodies that mimic ligand binding (such as those reported in therapeutic studies) can induce receptor internalization and downregulation, offering a means to modulate EPHA3 activity in disease settings (arora2023ephreceptorsin pages 23-24, keane2012epha3asa pages 12-13).

Regulation is further refined by the balance of forward and reverse signaling. While EPHA3 forward signaling in the receptor-bearing cell is mediated by its kinase activity, reverse signaling through the ephrin ligands on adjacent cells contributes additional layers of modulation. The coordinated interplay of these bidirectional signals controls cellular processes such as adhesion and migration without broadly affecting proliferative pathways (brantleysieders2004ephreceptortyrosine pages 2-3, arora2023ephreceptorsin pages 19-20).

1. Function  
   EPHA3 functions primarily as a mediator of contact‐dependent, bidirectional cell–cell signaling. Upon binding to its cognate ephrin-A ligands, the receptor becomes rapidly phosphorylated and activates a cascade of intracellular events that regulate essential cellular processes. In its canonical forward signaling mode, EPHA3 modulates cell–cell adhesion, cytoskeletal organization, and cell migration. These processes are particularly critical during development, where EPHA3 is implicated in the proper formation of tissue boundaries, such as the segregation of motor and sensory axons in neuromuscular circuits and in the retinotectal mapping of neuronal connections (arora2023ephreceptorsin pages 22-23, gu2001theepha8receptor pages 19-19).

In cardiac development, EPHA3 plays a role in guiding the migration and differentiation of cardiac cells, contributing to the formation of the atrioventricular canal and septum; these processes are thought to be mediated through interactions with ephrin-A ligands, notably ephrin-A1, which initiate downstream cascades leading to changes in cellular adhesion and shape (arora2023ephreceptorsin pages 22-23, keane2012epha3asa pages 13-14). In the developing nervous system, EPHA3 is involved in establishing precise topographic maps through its regulation of growth cone dynamics and axon guidance. The ligand-induced repulsive signals generated by EPHA3 activity ensure that neurons extend processes to their correct targets, thereby contributing to the overall architecture of neural circuits (forse2015distinctivestructureof pages 1-2, arora2023ephreceptorsin pages 19-20).

In addition to its developmental roles, EPHA3 is expressed in various adult tissues at low levels and has been found to be aberrantly expressed in several human tumors. In cancer, EPHA3 exhibits context-dependent behavior, functioning either as a promoter or suppressor of tumorigenesis. In certain malignancies, upregulation of EPHA3 contributes to enhanced tumor cell migration, invasiveness, and interactions with the tumor microenvironment. Conversely, when expressed in specific contexts such as colorectal cancer, its lower expression has been associated with tumor‐suppressive functions, potentially through the maintenance of adequate cell–cell adhesion and the inhibition of uncontrolled migration (arora2023ephreceptorsin pages 18-19, lisabeth2012cancersomaticmutations pages 7-8).

EPHA3 also interfaces with multiple downstream signaling pathways. Upon activation, the formation of phosphorylated docking sites enables the recruitment of adaptor proteins and enzymes that guide changes in the actin cytoskeleton – a process fundamental to both developmental cell positioning and pathological processes such as metastasis. Through these interactions, EPHA3 impacts integrin-mediated adhesion and modulates small GTPase activity, such as that of Rho, Rac, and Cdc42, which oversee focal adhesion dynamics and actin filament assembly. This coordination of cytoskeletal reorganization is central to the receptor’s role in both physiological developmental events and pathological states like cancer progression (clifford2008theepha3receptor pages 10-10, keane2012epha3asa pages 13-14).

1. Other Comments  
   EPHA3 has attracted significant interest as a potential therapeutic target, particularly in the oncology field. Experimental inhibitory agents, including monoclonal antibodies such as IIIA4 and ifabotuzumab (KB004), have been developed to bind the extracellular domain of EPHA3, thereby blocking ligand binding and perturbing receptor clustering. Such agents have been employed in preclinical studies and early-phase clinical trials to assess their efficacy in disrupting EPHA3-mediated signaling in tumors (arora2023ephreceptorsin pages 23-24, keane2012epha3asa pages 12-13).

Furthermore, somatic mutations in EPHA3 have been identified in a range of cancers including lung adenocarcinoma, colorectal cancer, and glioblastoma. These mutations, which affect critical domains such as the ligand-binding and kinase domains, can disrupt normal receptor function by altering phosphorylation events or receptor surface localization. The dual role of EPHA3 in cancer – acting either as a tumor suppressor or as a promoter of tumorigenicity depending on cellular context – has spurred ongoing research into its function as both a biomarker and a therapeutic target (lisabeth2012cancersomaticmutations pages 10-11, keane2012epha3asa pages 1-2).

In addition to its implications in cancer, EPHA3 participates in normal developmental processes including cardiac morphogenesis and axon guidance, underscoring its broader biological significance. The receptor’s ability to engage in bidirectional signaling provides an intricate mechanism for coordinating cellular responses between adjacent cells, influencing not only the behavior of the receptor-expressing cell but also that of the neighboring ephrin-bearing cell (arora2023ephreceptorsin pages 22-23, gu2001theepha8receptor pages 19-19).

Recent structural studies have further emphasized that unique structural features, such as the extensive and specific interaction interface between EPHA3 and ephrin-A5, may be exploited for the design of selective inhibitors. These structural insights, combined with detailed analyses of downstream signaling cascades, continue to expand the potential for targeted therapeutic interventions in diseases where EPHA3 signaling is dysregulated (forse2015distinctivestructureof pages 9-12, keane2012epha3asa pages 3-4).

1. References  
   Forse, G.J., Uson, M., Nasertorabi, F., Kolatkar, A., Lamberto, I., Pasquale, E., & Kuhn, P. (2015). Distinctive structure of the epha3/ephrin-a5 complex reveals a dual mode of eph receptor interaction for ephrin-a5. PLoS ONE, May 2015. (forse2015distinctivestructureof pages 1-2; forse2015distinctivestructureof pages 2-4; forse2015distinctivestructureof pages 9-12; forse2015distinctivestructureof pages 15-16).  
   Keane, N., Freeman, C., Swords, R., & Giles, F.J. (2012). Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. (keane2012epha3asa pages 2-3; keane2012epha3asa pages 3-4; keane2012epha3asa pages 12-13; keane2012epha3asa pages 13-14; keane2012epha3asa pages 1-2).  
   Lisabeth, E.M., Fernandez, C., & Pasquale, E.B. (2012). Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51(7):1464-1475, Feb 2012. (lisabeth2012cancersomaticmutations pages 1-2; lisabeth2012cancersomaticmutations pages 4-6; lisabeth2012cancersomaticmutations pages 7-8; lisabeth2012cancersomaticmutations pages 10-11; lisabeth2012cancersomaticmutations pages 13-14).  
   Arora, S., Scott, A.M., & Janes, P.W. (2023). Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. (arora2023ephreceptorsin pages 3-4; arora2023ephreceptorsin pages 4-5; arora2023ephreceptorsin pages 18-19; arora2023ephreceptorsin pages 19-20; arora2023ephreceptorsin pages 20-22; arora2023ephreceptorsin pages 22-23; arora2023ephreceptorsin pages 23-24).  
   Brantley-Sieders, D.M., Schmidt, S., Parker, M., & Chen, J. (2004). Eph receptor tyrosine kinases in tumor and tumor microenvironment. Current Pharmaceutical Design, 10(27):3431-3442, Sep 2004. (brantleysieders2004ephreceptortyrosine pages 1-2; brantleysieders2004ephreceptortyrosine pages 2-3; brantleysieders2004ephreceptortyrosine pages 3-4; brantleysieders2004ephreceptortyrosine pages 8-9).  
   Chen, X., Lu, B., Ma, Q., Ji, C.D., & Li, J.Z. (2019). Epha3 inhibits migration and invasion of esophageal cancer cells by activating the mesenchymal‑epithelial transition process. International Journal of Oncology, Nov 2019. (chen2019epha3inhibitsmigration pages 11-11).  
   Clifford, N., Smith, L.M., Powell, J., Gattenlöhner, S., Marx, A., & O’Connor, R. (2008). The epha3 receptor is expressed in a subset of rhabdomyosarcoma cell lines and suppresses cell adhesion and migration. Journal of Cellular Biochemistry, Dec 2008. (clifford2008theepha3receptor pages 10-10).  
   Gu, C., & Park, S. (2001). The epha8 receptor regulates integrin activity through p110γ phosphatidylinositol-3 kinase in a tyrosine kinase activity-independent manner. Molecular and Cellular Biology, 21:4579-4597, Jul 2001. (gu2001theepha8receptor pages 19-19).  
   Anderton, M., van der Meulen, E., Blumenthal, M.J., & Schäfer, G. (2021). The role of the eph receptor family in tumorigenesis. Cancers, 13:206, Jan 2021. (anderton2021theroleof pages 1-2; anderton2021theroleof pages 2-4).

References

1. (arora2023ephreceptorsin pages 20-22): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
2. (forse2015distinctivestructureof pages 1-2): G. J. Forse, M. Uson, F. Nasertorabi, A. Kolatkar, Ilaria Lamberto, E. Pasquale, and P. Kuhn. Distinctive structure of the epha3/ephrin-a5 complex reveals a dual mode of eph receptor interaction for ephrin-a5. PLoS ONE, May 2015. URL: https://doi.org/10.1371/journal.pone.0127081, doi:10.1371/journal.pone.0127081. This article has 13 citations and is from a peer-reviewed journal.
3. (forse2015distinctivestructureof pages 15-16): G. J. Forse, M. Uson, F. Nasertorabi, A. Kolatkar, Ilaria Lamberto, E. Pasquale, and P. Kuhn. Distinctive structure of the epha3/ephrin-a5 complex reveals a dual mode of eph receptor interaction for ephrin-a5. PLoS ONE, May 2015. URL: https://doi.org/10.1371/journal.pone.0127081, doi:10.1371/journal.pone.0127081. This article has 13 citations and is from a peer-reviewed journal.
4. (keane2012epha3asa pages 3-4): Niamh Keane, Ciara Freeman, Ronan Swords, and Francis J Giles. Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. URL: https://doi.org/10.1586/ehm.12.19, doi:10.1586/ehm.12.19. This article has 36 citations and is from a peer-reviewed journal.
5. (lisabeth2012cancersomaticmutations pages 1-2): Erika M. Lisabeth, Carlos Fernandez, and Elena B. Pasquale. Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51 7:1464-75, Feb 2012. URL: https://doi.org/10.1021/bi2014079, doi:10.1021/bi2014079. This article has 78 citations and is from a peer-reviewed journal.
6. (arora2023ephreceptorsin pages 19-20): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
7. (arora2023ephreceptorsin pages 22-23): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
8. (arora2023ephreceptorsin pages 23-24): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
9. (arora2023ephreceptorsin pages 4-5): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
10. (brantleysieders2004ephreceptortyrosine pages 2-3): Dana M. Brantley-Sieders, Sonja Schmidt, M. Parker, and Junya Chen. Eph receptor tyrosine kinases in tumor and tumor microenvironment. Current pharmaceutical design, 10 27:3431-42, Sep 2004. URL: https://doi.org/10.2174/1381612043383160, doi:10.2174/1381612043383160. This article has 144 citations and is from a peer-reviewed journal.
11. (brantleysieders2004ephreceptortyrosine pages 3-4): Dana M. Brantley-Sieders, Sonja Schmidt, M. Parker, and Junya Chen. Eph receptor tyrosine kinases in tumor and tumor microenvironment. Current pharmaceutical design, 10 27:3431-42, Sep 2004. URL: https://doi.org/10.2174/1381612043383160, doi:10.2174/1381612043383160. This article has 144 citations and is from a peer-reviewed journal.
12. (brantleysieders2004ephreceptortyrosine pages 8-9): Dana M. Brantley-Sieders, Sonja Schmidt, M. Parker, and Junya Chen. Eph receptor tyrosine kinases in tumor and tumor microenvironment. Current pharmaceutical design, 10 27:3431-42, Sep 2004. URL: https://doi.org/10.2174/1381612043383160, doi:10.2174/1381612043383160. This article has 144 citations and is from a peer-reviewed journal.
13. (chen2019epha3inhibitsmigration pages 11-11): Xia Chen, Bin Lu, Qian Ma, Cheng‑Dong Ji, and Jian‑Zhong Li. Epha3 inhibits migration and invasion of esophageal cancer cells by activating the mesenchymal‑epithelial transition process. International Journal of Oncology, Nov 2019. URL: https://doi.org/10.3892/ijo.2018.4639, doi:10.3892/ijo.2018.4639. This article has 20 citations and is from a peer-reviewed journal.
14. (clifford2008theepha3receptor pages 10-10): Noretta Clifford, Loraine M. Smith, James Powell, Stefan Gattenlöhner, Alexander Marx, and Rosemary O’Connor. The epha3 receptor is expressed in a subset of rhabdomyosarcoma cell lines and suppresses cell adhesion and migration. Journal of Cellular Biochemistry, Dec 2008. URL: https://doi.org/10.1002/jcb.21926, doi:10.1002/jcb.21926. This article has 48 citations and is from a peer-reviewed journal.
15. (forse2015distinctivestructureof pages 2-4): G. J. Forse, M. Uson, F. Nasertorabi, A. Kolatkar, Ilaria Lamberto, E. Pasquale, and P. Kuhn. Distinctive structure of the epha3/ephrin-a5 complex reveals a dual mode of eph receptor interaction for ephrin-a5. PLoS ONE, May 2015. URL: https://doi.org/10.1371/journal.pone.0127081, doi:10.1371/journal.pone.0127081. This article has 13 citations and is from a peer-reviewed journal.
16. (forse2015distinctivestructureof pages 9-12): G. J. Forse, M. Uson, F. Nasertorabi, A. Kolatkar, Ilaria Lamberto, E. Pasquale, and P. Kuhn. Distinctive structure of the epha3/ephrin-a5 complex reveals a dual mode of eph receptor interaction for ephrin-a5. PLoS ONE, May 2015. URL: https://doi.org/10.1371/journal.pone.0127081, doi:10.1371/journal.pone.0127081. This article has 13 citations and is from a peer-reviewed journal.
17. (keane2012epha3asa pages 1-2): Niamh Keane, Ciara Freeman, Ronan Swords, and Francis J Giles. Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. URL: https://doi.org/10.1586/ehm.12.19, doi:10.1586/ehm.12.19. This article has 36 citations and is from a peer-reviewed journal.
18. (keane2012epha3asa pages 12-13): Niamh Keane, Ciara Freeman, Ronan Swords, and Francis J Giles. Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. URL: https://doi.org/10.1586/ehm.12.19, doi:10.1586/ehm.12.19. This article has 36 citations and is from a peer-reviewed journal.
19. (keane2012epha3asa pages 13-14): Niamh Keane, Ciara Freeman, Ronan Swords, and Francis J Giles. Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. URL: https://doi.org/10.1586/ehm.12.19, doi:10.1586/ehm.12.19. This article has 36 citations and is from a peer-reviewed journal.
20. (keane2012epha3asa pages 2-3): Niamh Keane, Ciara Freeman, Ronan Swords, and Francis J Giles. Epha3 as a novel therapeutic target in the hematological malignancies. Expert Review of Hematology, 5:325-340, Jun 2012. URL: https://doi.org/10.1586/ehm.12.19, doi:10.1586/ehm.12.19. This article has 36 citations and is from a peer-reviewed journal.
21. (lisabeth2012cancersomaticmutations pages 10-11): Erika M. Lisabeth, Carlos Fernandez, and Elena B. Pasquale. Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51 7:1464-75, Feb 2012. URL: https://doi.org/10.1021/bi2014079, doi:10.1021/bi2014079. This article has 78 citations and is from a peer-reviewed journal.
22. (lisabeth2012cancersomaticmutations pages 13-14): Erika M. Lisabeth, Carlos Fernandez, and Elena B. Pasquale. Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51 7:1464-75, Feb 2012. URL: https://doi.org/10.1021/bi2014079, doi:10.1021/bi2014079. This article has 78 citations and is from a peer-reviewed journal.
23. (lisabeth2012cancersomaticmutations pages 4-6): Erika M. Lisabeth, Carlos Fernandez, and Elena B. Pasquale. Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51 7:1464-75, Feb 2012. URL: https://doi.org/10.1021/bi2014079, doi:10.1021/bi2014079. This article has 78 citations and is from a peer-reviewed journal.
24. (lisabeth2012cancersomaticmutations pages 7-8): Erika M. Lisabeth, Carlos Fernandez, and Elena B. Pasquale. Cancer somatic mutations disrupt functions of the epha3 receptor tyrosine kinase through multiple mechanisms. Biochemistry, 51 7:1464-75, Feb 2012. URL: https://doi.org/10.1021/bi2014079, doi:10.1021/bi2014079. This article has 78 citations and is from a peer-reviewed journal.
25. (anderton2021theroleof pages 1-2): Meg Anderton, Emma van der Meulen, Melissa J. Blumenthal, and Georgia Schäfer. The role of the eph receptor family in tumorigenesis. Cancers, 13:206, Jan 2021. URL: https://doi.org/10.3390/cancers13020206, doi:10.3390/cancers13020206. This article has 67 citations and is from a peer-reviewed journal.
26. (anderton2021theroleof pages 2-4): Meg Anderton, Emma van der Meulen, Melissa J. Blumenthal, and Georgia Schäfer. The role of the eph receptor family in tumorigenesis. Cancers, 13:206, Jan 2021. URL: https://doi.org/10.3390/cancers13020206, doi:10.3390/cancers13020206. This article has 67 citations and is from a peer-reviewed journal.
27. (arora2023ephreceptorsin pages 18-19): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
28. (arora2023ephreceptorsin pages 3-4): Sakshi Arora, Andrew M. Scott, and Peter W. Janes. Eph receptors in cancer. Biomedicines, 11:315, Jan 2023. URL: https://doi.org/10.3390/biomedicines11020315, doi:10.3390/biomedicines11020315. This article has 36 citations and is from a peer-reviewed journal.
29. (brantleysieders2004ephreceptortyrosine pages 1-2): Dana M. Brantley-Sieders, Sonja Schmidt, M. Parker, and Junya Chen. Eph receptor tyrosine kinases in tumor and tumor microenvironment. Current pharmaceutical design, 10 27:3431-42, Sep 2004. URL: https://doi.org/10.2174/1381612043383160, doi:10.2174/1381612043383160. This article has 144 citations and is from a peer-reviewed journal.
30. (gu2001theepha8receptor pages 19-19): Changkyu Gu and Soochul Park. The epha8 receptor regulates integrin activity through p110γ phosphatidylinositol-3 kinase in a tyrosine kinase activity-independent manner. Molecular and Cellular Biology, 21:4579-4597, Jul 2001. URL: https://doi.org/10.1128/mcb.21.14.4579-4597.2001, doi:10.1128/mcb.21.14.4579-4597.2001. This article has 131 citations and is from a domain leading peer-reviewed journal.