1. Phylogeny  
   PIKfyve (gene symbol PIKFYVE, also known as KIAA0981 or PIP5K3) is the mammalian ortholog of the yeast Fab1p lipid kinase and is evolutionarily conserved across eukaryotes. It belongs to the type III phosphoinositide kinase family and is a member of the fundamental kinase machinery that appeared early during evolution, being present from yeast to higher mammals. In many species, including mammals, PIKfyve shares significant sequence homology and domain organization with its yeast counterpart, indicating that its enzymatic and regulatory features have been maintained throughout evolution (dayam2017roleofpikfyve pages 124-129, shisheva2012pikfyveandits pages 1-4). Moreover, phylogenetic studies place PIKfyve within the core set of proteins that regulate phosphoinositide metabolism, which include other evolutionarily ancient components of lipid signaling pathways. Although PIKfyve is categorized separately from the serine/threonine protein kinases of the AGC family, its conservation alongside other lipid kinases such as PI3Ks underscores its pivotal role in intracellular membrane dynamics (rossignol2025phosphoinositidemetabolismbiochemistry pages 14-15, heck2007aconspicuousconnection pages 2-4).
2. Reaction Catalyzed  
   PIKfyve catalyzes the phosphorylation of phosphatidylinositol 3-phosphate (PI3P) by transferring a phosphate group from ATP to the 5-hydroxyl group of the myo-inositol ring, thereby producing phosphatidylinositol 3,5-bisphosphate [PI(3,5)P2] and ADP. In a concise chemical equation, the reaction can be represented as: ATP + PI3P → ADP + PI(3,5)P2. In addition, PIKfyve is also capable of catalyzing the phosphorylation of phosphatidylinositol (PI) at the similar 5-position to form phosphatidylinositol 5-phosphate (PI5P); this reaction may occur either directly or indirectly via the conversion of PI(3,5)P2 by phosphatases of the myotubularin family (dayam2017roleofpikfyve pages 124-129, poli2019phosphatidylinositol5phosphate pages 13-14). Thus, PIKfyve participates in the complex regulation of phosphoinositide pools that are critical for diverse cellular processes.
3. Cofactor Requirements  
   The catalytic activity of PIKfyve is dependent on the presence of divalent metal ions, with Mg²⁺ being required as an essential cofactor to facilitate ATP binding and the transfer of the phosphate group. Mg²⁺ stabilizes the negative charges on the phosphate groups of ATP and ensures proper coordination within the active site of the kinase (chen2022theroleof pages 3-4, heck2007aconspicuousconnection pages 2-4). Although the literature primarily highlights Mg²⁺ as the cofactor for lipid kinases such as PIKfyve, other divalent ions like Mn²⁺ may also support its activity under certain conditions; however, Mg²⁺ remains the predominant cofactor under physiological conditions (deliberty2024therapeutictargetingof pages 44-48, shisheva2012pikfyveandits pages 4-6).
4. Substrate Specificity  
   PIKfyve exhibits marked substrate specificity for phosphatidylinositol 3-phosphate (PI3P), which is recognized by the enzyme’s FYVE domain with high affinity. This preferential binding and subsequent phosphorylation of PI3P underpin the production of PI(3,5)P2, a key signaling lipid in membrane trafficking and endosomal homeostasis (liggins2016elucidatingtherole pages 48-52, shisheva2012pikfyveandits pages 6-9). In addition to PI3P, PIKfyve may phosphorylate phosphatidylinositol (PI) to yield PI5P, although this activity is generally considered secondary to its primary role in converting PI3P to PI(3,5)P2 (poli2019phosphatidylinositol5phosphate pages 13-14, shisheva2012pikfyveandits pages 1-4). The enzyme does not exhibit activity toward other phosphatidylinositols such as PI4P, and its substrate specificity is driven by structural motifs that facilitate the recognition and binding of the phosphoinositide headgroup rather than a peptide consensus motif seen in protein kinases (liggins2016elucidatingtherole pages 52-57, rodahl2004characterisationofthe pages 20-24).
5. Structure  
   PIKfyve is a large multifunctional protein that features a modular architecture comprised of several distinct structural domains, each contributing to its regulatory and catalytic functions. The N-terminal region harbors a FYVE finger domain, which is responsible for binding to PI3P and targeting the enzyme to endosomal membranes enriched in this phosphoinositide. This zinc finger motif confers high-affinity binding to PI3P through coordination of zinc ions and is critical for proper subcellular localization (shisheva2012pikfyveandits pages 1-4, chen2022theroleof pages 3-4). Following the FYVE domain, PIKfyve contains a DEP (Dishevelled, Egl-10, Pleckstrin) domain, a feature commonly found in proteins involved in signal transduction and membrane association. Although the precise role of the DEP domain in PIKfyve remains to be fully elucidated, it is thought to mediate specific protein–protein interactions and potentially contribute to the regulation of its catalytic activity (liggins2016elucidatingtherole pages 107-114, shisheva2012pikfyveandits pages 12-15). Centrally located within the protein is a chaperonin-like domain that may facilitate proper folding and stabilize interactions with other components of the phosphoinositide regulatory complexes, particularly VAC14 and FIG4. The C-terminal region encompasses the kinase domain, which adopts a bilobed architecture typical of protein kinases; it contains conserved catalytic motifs including an ATP-binding site with an essential lysine residue (for example, Lys1831) critical for phosphotransferase activity (shisheva2012pikfyveandits pages 4-6, shisheva2012pikfyveandits pages 6-9, currinn2016appcontrolsthe pages 15-16). Structural models suggest that the kinase domain is organized into an N-lobe and a C-lobe with a flexible activation loop that may undergo conformational changes upon substrate binding, thereby modulating catalytic efficiency. In addition, the overall three-dimensional organization of PIKfyve facilitates its integration into larger protein complexes, such as the PAS complex, which is essential for coordinating the synthesis and turnover of PI(3,5)P2 (dayam2017roleofpikfyve pages 124-129, shisheva2012pikfyveandits pages 32-34). The presence of multiple domains with distinct functions ensures that PIKfyve is capable of both lipid kinase and serine protein kinase activities, with its autophosphorylation and transphosphorylation activities being dependent on the integrity of the catalytic and regulatory domains (shisheva2012pikfyveandits pages 34-36, heck2007aconspicuousconnection pages 4-6).
6. Regulation  
   Regulatory mechanisms controlling PIKfyve activity are multifaceted and involve both post-translational modifications and dynamic protein–protein interactions. One major regulatory mechanism is autophosphorylation; PIKfyve can autophosphorylate several serine residues within its inter-domain regions, and this autophosphorylation event has been demonstrated to inhibit its lipid kinase activity while concomitantly stimulating the activity of the FIG4 lipid phosphatase. This regulatory feedback mechanism is crucial for maintaining proper levels of PI(3,5)P2 and ensuring the balance between lipid synthesis and turnover (shisheva2012pikfyveandits pages 34-36, shisheva2019severeconsequencesof pages 12-14). Furthermore, PIKfyve is regulated through its assembly into the PAS complex, which includes the scaffold protein VAC14 and the phosphatase FIG4; VAC14 facilitates binding of PIKfyve and FIG4, thereby stabilizing the complex and modulating enzymatic activities through regulated interactions (shisheva2012pikfyveandits pages 25-28, shisheva2012pikfyveandits pages 28-30). Upstream signals such as insulin and other growth factors positively influence PIKfyve activity, which in turn modulates endosomal trafficking and membrane recycling. Insulin signaling, for instance, has been shown to enhance PIKfyve activity, linking external signals to intracellular phosphoinositide dynamics (dayam2017roleofpikfyve pages 124-129, zhang2008therolesand pages 14-18). In addition, stress conditions such as hyperosmolarity and oxidative stress can also modulate PIKfyve function, thereby affecting the cellular response to environmental cues (zhang2008therolesand pages 14-18, foulger2014investigatingtheproduction pages 23-26). Spatial regulation is achieved by the FYVE domain, which ensures that PIKfyve is localized to PI3P-rich endosomal membranes where its substrates are concentrated, and interactions with small GTPases like Rab5 further refine this localization and activity (currinn2016appcontrolsthe pages 15-16, heck2007aconspicuousconnection pages 7-8). These combined regulatory mechanisms ensure that PIKfyve activity is tightly controlled to maintain endomembrane homeostasis and coordinate multiple downstream signaling pathways.
7. Function  
   PIKfyve is an essential regulator of intracellular membrane dynamics and phosphoinositide metabolism with a broad spectrum of cellular functions. Its primary role is the generation of PI(3,5)P2 from PI3P, an event that is critical for controlling endosomal and lysosomal membrane homeostasis. PI(3,5)P2 is involved in several key processes such as endocytic trafficking, vacuolar size control, and the maturation of lysosomes. In macrophages and neutrophils, for example, PIKfyve is crucial for proper lysosome morphology, phagosome maturation, and the activation of Rac GTPase, which plays a role in chemotaxis and reactive oxygen species production (dayam2017roleofpikfyve pages 180-187, shisheva2012pikfyveandits pages 1-4). In addition to its role in the endocytic-vacuolar pathway, PIKfyve influences nuclear transport and cell cycle progression, thereby integrating membrane dynamics with broader cellular signaling events (deliberty2024therapeutictargetingof pages 134-137, shisheva2012pikfyveandits pages 9-12). PIKfyve’s activity is also linked to autophagic processes; its inhibition leads to the accumulation of autophagic vacuoles due to impaired lysosomal function, which underscores its role in regulating autophagic flux (currinn2016appcontrolsthe pages 15-16, roy2023pip5k1cphosphoinositidekinase pages 13-15). Tissue expression studies reveal that PIKfyve is broadly distributed, with high expression observed in tissues that rely heavily on membrane trafficking and lysosomal degradation, such as the brain and immune system (poli2019phosphatidylinositol5phosphate pages 13-14, shisheva2012pikfyveandits pages 1-4). Moreover, PIKfyve is implicated in signal transduction pathways activated by hormones and stress, linking alterations in phosphoinositide signaling to changes in cell survival, migration, and metabolism (dayam2017roleofpikfyve pages 124-129, sharma2019afamilyof pages 23-24). The enzyme’s integration into protein complexes such as the PAS complex further emphasizes its role in coordinating the synthesis and turnover of PI(3,5)P2, which is critical for normal cellular physiology and for the response to oncogenic signals in cancer cells (roy2023pip5k1cphosphoinositidekinase pages 1-2, shisheva2012pikfyveandits pages 25-28). Taken together, the functional importance of PIKfyve is reflected in its requirement for embryonic development, its involvement in autophagy, and its ability to regulate vital cellular processes that include endosomal trafficking, stress response, and metabolic control.
8. Other Comments  
   Experimental inhibitors of PIKfyve, such as YM201636, apilimod, and WX8, have been employed to dissect its functions in various cellular contexts and to explore its therapeutic potential in diseases like multiple myeloma and other cancers. These inhibitors disrupt the production of PI(3,5)P2, leading to impaired lysosomal acidification, vacuolation, and the accumulation of autophagic vesicles (roy2023pip5k1cphosphoinositidekinase pages 1-2, sharma2019afamilyof pages 23-24, campos2019identificationofpikfyve pages 9-9). In addition, PIKfyve exhibits serine-protein kinase activity; it can autophosphorylate and transphosphorylate substrates, a feature that has been linked to the regulation of FIG4 lipid phosphatase activity within the phosphoinositide regulatory complex (shisheva2012pikfyveandits pages 6-9, shisheva2012pikfyveandits pages 34-36). Dysregulation of PIKfyve has been associated with several pathological conditions, including neurodegenerative disorders, due to its essential role in endosomal and lysosomal function, and in immune dysfunction, which may arise from defective phagosome maturation and receptor recycling (dayam2017roleofpikfyve pages 180-187, shisheva2019severeconsequencesof pages 12-14). Genetic studies have demonstrated that complete loss of PIKfyve function leads to embryonic lethality, underscoring its critical importance in development and cellular homeostasis (shisheva2019severeconsequencesof pages 12-14, dayam2017roleofpikfyve pages 124-129). Furthermore, alterations in PIKfyve activity are being explored as potential targets for cancer therapy, particularly in tumors that exhibit reliance on autophagy and lysosomal degradation pathways (roy2023pip5k1cphosphoinositidekinase pages 13-15, sharma2019afamilyof pages 23-24). The enzyme’s intricate regulation via autophosphorylation and its integration into multiprotein complexes provide multiple avenues for therapeutic intervention, and its inhibitors have entered early-phase clinical evaluation in certain cancers, highlighting its translational relevance (currinn2016appcontrolsthe pages 15-16, campos2019identificationofpikfyve pages 9-9). Collectively, these insights emphasize the multifaceted role of PIKfyve as a central node in phosphoinositide metabolism and cellular signaling.
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