1. Phylogeny  
Inositol polyphosphate multikinase (IPMK, gene: IPMK/IMPK; UniProt Q8NFU5) belongs to a conserved family of inositol phosphate kinases that emerged early in eukaryotic evolution and is ubiquitously present in organisms ranging from unicellular yeast to plants and mammals. Orthologs of IPMK have been identified in Saccharomyces cerevisiae (where it is referred to as Ipmk) as well as in Arabidopsis and animal systems, reflecting its critical role in inositol phosphate metabolism and cellular signaling. Comparative sequence analyses and phylogenetic studies using conserved catalytic domains and substrate‐binding motifs reveal that IPMK diverged from the ancestral inositol‐phosphate kinase family prior to the emergence of the metazoan lineage, thereby forming part of the evolutionary core set of kinases that regulate diverse signal transduction pathways in eukaryotes (malabanan2016inositolpolyphosphatemultikinase pages 1-3, shears2004howversatileare pages 1-2). Functional conservation among these orthologs is reinforced by the retention of key catalytic residues and structural domains across species, indicating that IPMK has maintained an essential function in the biosynthesis of higher inositol polyphosphates throughout evolution (rango2019inositolpolyphosphatemultikinase pages 6-8, cestari2016chemogeneticcharacterizationof pages 21-25).

2. Reaction Catalyzed  
IPMK catalyzes sequential phosphorylation reactions in the inositol phosphate metabolic pathway using ATP as a phosphate donor. In particular, the enzyme phosphorylates inositol 1,4,5-trisphosphate (Ins(1,4,5)P3) to produce inositol 1,3,4,5-tetrakisphosphate (Ins(1,3,4,5)P4) and then further phosphorylates it to generate inositol 1,3,4,5,6-pentakisphosphate (Ins(1,3,4,5,6)P5). Additionally, IPMK catalyzes the phosphorylation of inositol 1,3,4,6-tetrakisphosphate (Ins(1,3,4,6)P4), and it can convert glycero-3-phospho-1D-myo-inositol 4,5-bisphosphate to glycero-3-phospho-1D-myo-inositol 3,4,5-trisphosphate. The enzyme also exhibits phosphatidylinositol kinase activity by phosphorylating phosphatidylinositol 4,5-bisphosphate (PIP2) to generate phosphatidylinositol 3,4,5-trisphosphate (PIP3), thereby coupling soluble inositol phosphate metabolism with lipid signaling pathways. Overall, the catalytic reaction proceeds in a manner analogous to classical kinase reactions: ATP + [inositol phosphate substrate] → ADP + [phosphorylated inositol phosphate] + H⁺ (malabanan2016inositolpolyphosphatemultikinase pages 3-4, wang2017structuralfeaturesof pages 2-4, dailey2012inositolpolyphosphatemultikinase pages 1-2).

3. Cofactor Requirements  
IPMK’s catalytic activity requires ATP as the phosphate donor and depends critically on the presence of divalent metal ions—most notably Mg²⁺—which serve to stabilize negative charges during transition state formation in the phosphoryl transfer reaction. Structural characterization of human IPMK has revealed at least two magnesium ions coordinated in the nucleotide-binding pocket, a feature that is consistent with its function as a classical kinase and indispensable for its phosphotransferase activity (wang2017structuralfeaturesof pages 1-2, dailey2012inositolpolyphosphatemultikinase pages 1-2, wang2017structuralfeaturesof pages 5-6).

4. Substrate Specificity  
IPMK exhibits broad substrate specificity that is integral to its multifunctional role in the cell. It phosphorylates several inositol phosphate substrates including Ins(1,4,5)P3, converting it sequentially to Ins(1,3,4,5)P4 and then to Ins(1,3,4,5,6)P5, and it also acts on Ins(1,3,4,6)P4. In addition to its soluble inositol phosphate substrates, IPMK is capable of phosphorylating lipid-bound substrates; for example, it catalyzes the conversion of phosphatidylinositol 4,5-bisphosphate (PIP2) to phosphatidylinositol 3,4,5-trisphosphate (PIP3) in a phosphoinositide 3-kinase-like reaction. The enzyme’s active site is structured to accommodate these chemically diverse substrates, enabling it to perform dual functions in both inositol phosphate and phosphoinositide signaling pathways. This substrate versatility reflects the presence of a flexible inositol phosphate-binding loop and an overall positively charged catalytic pocket that selectively stabilizes a variety of phosphate group arrangements (malabanan2016inositolpolyphosphatemultikinase pages 3-4, wang2017structuralfeaturesof pages 8-9, dailey2012inositolpolyphosphatemultikinase pages 2-4).

5. Structure  
Human IPMK is organized around a central catalytic kinase domain that displays a classical bilobal architecture consisting of an N-terminal lobe (N-lobe) and a C-terminal lobe (C-lobe). The N-lobe is composed primarily of four antiparallel β-strands interspersed with several α-helices that form a well-defined nucleotide-binding region, while the C-lobe contains a mixed α/β structure responsible for substrate binding and catalysis. Key catalytic features include a conserved ATP-grasp fold and an inositol phosphate-binding loop (IP loop) that is critical for recognizing substrate molecules; the active site harbors residues such as Lys75, Gln163, Gln164, Lys167, Gln196, Asp385, and His388, which are essential for both ATP coordination and catalysis (wang2017structuralfeaturesof pages 2-4, wang2017structuralfeaturesof pages 5-6, shears2004howversatileare pages 10-11). Structural studies, including high-resolution crystallographic data, have further revealed a unique proline loop within the human enzyme that distinguishes its substrate interactions from those observed in yeast and plant orthologs. In addition, IPMK contains regions that mediate its subcellular localization, with nuclear localization signals and N-terminal extensions implicated in binding to regulatory complexes such as mTOR, thereby linking its enzymatic functions with broader signaling networks (wang2017structuralfeaturesof pages 8-9, malabanan2016inositolpolyphosphatemultikinase pages 8-9, xu2024acladeof pages 10-11).

6. Regulation  
Regulatory control of IPMK operates on several levels. Post-translational modifications, including specific phosphorylation events, modulate IPMK’s activity and subcellular localization. For instance, phosphorylation(s) mediated by kinases involved in nutrient signaling pathways have been shown to influence IPMK’s interaction with proteins such as mTOR and AMPK, thereby affecting downstream signal transduction (dailey2012inositolpolyphosphatemultikinase pages 1-2, dailey2012inositolpolyphosphatemultikinase pages 2-4). In certain pathological conditions, such as in models of Huntington’s disease, IPMK protein stability is compromised through enhanced lysosomal degradation which is triggered by interactions with mutant huntingtin protein; such degradation correlates with diminished Akt signaling and mitochondrial dysfunction (hong2024anoncatalyticrole pages 8-11, lee2023mirnainduceddownregulationof pages 10-11). In addition to these modifications, IPMK activity may be subject to substrate concentration-dependent feedback inhibition and allosteric changes induced by binding of its polyphosphate products, as revealed by kinetic analyses and structural studies (wang2017structuralfeaturesof pages 5-6, malabanan2016inositolpolyphosphatemultikinase pages 9-13).

7. Function  
IPMK plays a central role in the metabolism of inositol phosphates, catalyzing the conversion of Ins(1,4,5)P3 into higher-order inositol polyphosphates such as Ins(1,3,4,5)P4 and Ins(1,3,4,5,6)P5, molecules that are critical for numerous cellular processes. The production of InsP5 and subsequently inositol hexakisphosphate (InsP6) is essential for the execution of various signaling cascades that regulate cell growth, gene expression, and messenger RNA export from the nucleus. Through its dual kinase activity, IPMK also generates phosphatidylinositol 3,4,5-trisphosphate (PIP3), thereby linking soluble inositol phosphate metabolism with membrane-associated PI3K signaling pathways that control cell proliferation and survival. Furthermore, the highly phosphorylated inositol polyphosphates produced by IPMK play an important role in MLKL-mediated necroptosis; binding of these molecules to MLKL facilitates the release of an N-terminal auto-inhibitory domain that is required for necroptotic signaling (wang2017structuralfeaturesof pages 9-10, rango2019inositolpolyphosphatemultikinase pages 12-12). In addition to its catalytic functions, IPMK acts as a transcriptional coactivator by interacting with components of chromatin remodeling complexes and the mRNA export machinery, and is thus implicated in the regulation of gene expression. Expression studies indicate that IPMK is ubiquitously expressed with enrichment in tissues where rapid phosphoinositide turnover and inositol phosphate signaling are critical, such as the brain, liver, and immune cells (dailey2012inositolpolyphosphatemultikinase pages 1-2, rango2019inositolpolyphosphatemultikinase pages 8-10, cestari2016chemogeneticcharacterizationof pages 21-25).

8. Other Comments  
No selective small-molecule inhibitors of IPMK have been firmly established in the literature to date, although modulation of its kinase activity has been proposed as a potential strategy for therapeutic intervention in diseases characterized by aberrant inositol phosphate signaling. IPMK has been linked to a variety of pathological conditions; for example, reduced levels of IPMK have been observed in models of Huntington’s disease, where its destabilization contributes to insufficient Akt signaling and impaired mitochondrial function. Moreover, alterations in IPMK expression and activity have been associated with metabolic dysregulation and neurodegenerative disorders, and its regulatory role in both soluble inositol phosphate and phosphoinositide pathways makes it an attractive candidate for further investigation with respect to cancer and immune-related diseases. Recent studies have also suggested a potential connection between IPMK activity and human longevity, particularly in female populations, thereby highlighting its broader physiological importance (lee2023mirnainduceddownregulationof pages 10-11, rango2019inositolpolyphosphatemultikinase pages 8-10, maffucci2020signallingpropertiesof pages 7-9, mihiret2024proteinpyrophosphorylationby pages 12-13).

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