1. Phylogeny  
   AMP‐activated protein kinase catalytic subunit alpha‑1 (PRKAA1, also known as AMPK1) belongs to the highly conserved family of AMP‐activated protein kinases that are present in all eukaryotes. This protein is evolutionarily related to yeast SNF1 and plant SnRK1, indicating that the kinase originated prior to the divergence of yeast, plants, and animals (sanz2008ampactivatedproteinkinase pages 1-5). In mammals, two distinct catalytic isoforms—alpha‑1 and alpha‑2—are expressed, with the alpha‑1 isoform being ubiquitously expressed and maintaining a central role in cellular energy homeostasis (hardie2012ampactivatedproteinkinase pages 1-2). The conservation of domain architecture across species, as well as the presence of orthologous regulatory subunits, places AMPK within an evolutionarily ancient core of energy‐sensing kinases that are traceable back to the Last Eukaryotic Common Ancestor (arad2007ampactivatedproteinkinase pages 1-3, sanz2008ampactivatedproteinkinase pages 14-17). In the wider kinome, AMPK alpha‑1 shares structural and mechanistic features with other serine/threonine kinases and is grouped with kinases that are tightly regulated by adenine nucleotide binding and phosphorylation events (kurumbail2016structureandregulation pages 1-4).
2. Reaction Catalyzed  
   AMPK alpha‑1 catalyzes the phosphorylation of serine or threonine residues on target proteins using ATP as a phosphate donor. The general reaction can be written as:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (sanz2008ampactivatedproteinkinase pages 5-8, arad2007ampactivatedproteinkinase pages 1-3).
3. Cofactor Requirements  
   Catalytic activity of AMPK alpha‑1 requires divalent metal ions for its kinase function. Specifically, Mg²⁺ is essential for ATP binding and proper enzymatic activity, serving as a cofactor in the phosphoryl transfer reaction (hardie2012ampactivatedproteinkinase pages 1-2, sanz2008ampactivatedproteinkinase pages 5-8).
4. Substrate Specificity  
   AMPK alpha‑1 phosphorylates a broad range of substrates that are central to cellular metabolic control. Its direct substrates include key metabolic enzymes such as acetyl‐CoA carboxylase (ACACA and ACACB), hormone‑sensitive lipase (LIPE), and regulatory proteins involved in insulin signalling and autophagy (arad2007ampactivatedproteinkinase pages 1-3, sanz2008ampactivatedproteinkinase pages 14-17). Although a precise consensus sequence for phosphorylation is not detailed in the provided context, the enzyme typically targets serine/threonine residues in regulatory regions of metabolic proteins, thereby modulating their activity in response to cellular energy status (li2019ampkandautophagy pages 97-100).
5. Structure  
   The catalytic subunit alpha‑1 of AMPK is approximately 63 kDa and is composed of multiple distinct regions that together form the structural basis for its kinase activity and regulation. The N‑terminal region contains a classical serine/threonine kinase domain that is organized into a small N‑lobe and a larger C‑lobe, a configuration typical of protein kinases (sanz2008ampactivatedproteinkinase pages 1-5). Within this domain lies the activation loop, which includes a critical threonine residue (Thr172 in AMPK‑α2; Thr174 in AMPK‑α1) whose phosphorylation increases kinase activity by more than 100-fold (hardie2011ampactivatedproteinkinase pages 2-3, saiу2010structuralandfunctional pages 11-16). Directly following the kinase domain is an autoinhibitory domain (AID) that serves to restrain enzymatic activity in the absence of allosteric activators (kurumbail2016structureandregulation pages 4-6). Adjacent to the AID, a flexible linker region containing regulatory interacting motifs (α-RIM1 and α-RIM2) connects the catalytic core to the C‑terminal region, which participates in interactions with the regulatory beta and gamma subunits (kurumbail2016structureandregulation pages 15-17, kurumbail2016structureandregulation pages 17-19). This C‑terminal domain also contains nuclear export sequences and a serine/threonine-rich loop, which is subject to additional post‑translational modifications. Structural studies have revealed that the kinase domain possesses key catalytic features such as a well‑defined ATP binding cleft, a correctly oriented C-helix, and a hydrophobic spine that are essential for substrate phosphorylation (saiу2010structuralandfunctional pages 16-21, sanz2008ampactivatedproteinkinase pages 40-46). The overall three-dimensional structure, as elucidated by crystallographic studies and supported by AlphaFold predictions, shows an elongated arrangement that allows dynamic interactions between the subunits of the AMPK heterotrimer (kurumbail2016structureandregulation pages 12-15).
6. Regulation  
   Regulation of AMPK alpha‑1 is achieved through multiple layers of control including allosteric activation, phosphorylation, and post‑translational modifications. A key regulatory mechanism is the phosphorylation of the activation loop threonine (Thr174 in AMPK‑α1), which is performed by upstream kinases such as LKB1, Ca²⁺/calmodulin-dependent protein kinase kinase‑β (CaMKKβ), and under certain conditions by TAK1 (arad2007ampactivatedproteinkinase pages 3-4, li2019ampkandautophagy pages 97-100, salt2017ampactivatedproteinkinase pages 1-2). Binding of AMP (and to a lesser extent ADP) to the gamma subunit allosterically promotes this phosphorylation and simultaneously protects the phosphorylated threonine from dephosphorylation by phosphatases, including PP2A and PP2C (hardie2011ampactivatedproteinkinase pages 2-3, russell2020ampactivatedproteinkinase pages 23-24). In addition to phosphorylation, AMPK alpha‑1 is subject to various post‑translational modifications that modulate its activity. These include ubiquitination events that can mark the protein for proteasomal degradation, as well as additional phosphorylation events within the serine/threonine‑rich (ST) loop that can negatively regulate activation by interfering with LKB1-mediated phosphorylation (ovens2021posttranslationalmodificationsof pages 5-6, ovens2021posttranslationalmodificationsof pages 12-14). The autoinhibitory domain (AID) of AMPK alpha‑1 also plays a significant role in maintaining the enzyme in an inactive conformation until appropriate allosteric signals relieve the inhibition (kurumbail2016structureandregulation pages 4-6, salt2017ampactivatedproteinkinase pages 2-3). This multi‑layered regulatory network ensures that AMPK is activated only under conditions of energetic stress, thereby coordinating downstream metabolic responses (tarasiuk2022ampkanddiseases pages 1-3, hardie2011ampactivatedproteinkinase pages 3-4).
7. Function  
   AMPK alpha‑1 functions as the catalytic core of the heterotrimeric AMPK complex and is a central regulator of cellular energy homeostasis. In response to a reduction in intracellular ATP levels and a concomitant increase in AMP or ADP, AMPK alpha‑1 is activated via phosphorylation and allosteric mechanisms, leading to the stimulation of catabolic pathways that generate ATP while inhibiting anabolic pathways that consume ATP (arad2007ampactivatedproteinkinase pages 1-3, wang2012ampactivatedproteinkinase pages 1-2). Its substrates include metabolic enzymes such as acetyl‑CoA carboxylase (regulating fatty acid synthesis and oxidation), hormone‑sensitive lipase (involved in lipid mobilization), and various proteins that modulate glucose uptake via the translocation of GLUT4 in muscle tissues (sanz2008ampactivatedproteinkinase pages 14-17, tarasiuk2022ampkanddiseases pages 5-6). AMPK alpha‑1 also phosphorylates transcriptional regulators—including FOXO3, p53, and TORC2—to affect gene expression programs related to mitochondrial biogenesis, autophagy, and cell cycle control (sanz2008ampactivatedproteinkinase pages 5-8, li2019ampkandautophagy pages 97-100). In the liver, it plays a critical role in regulating glucose homeostasis by controlling pathways that balance gluconeogenesis and glycolysis. Moreover, AMPK alpha‑1 is implicated in the regulation of cell growth and proliferation through its inhibitory effects on the mTORC1 complex via phosphorylation of RPTOR and activation of TSC2 (arad2007ampactivatedproteinkinase pages 4-5, tarasiuk2022ampkanddiseases pages 3-5). The enzyme is expressed widely across tissues, including in cardiac, skeletal muscle, liver, brain, and pancreatic cells, and contributes to adaptive responses during stress, such as ischemia, exercise, and nutrient deprivation (steinberg2009ampkinhealth pages 1-2, russell2020ampactivatedproteinkinase pages 23-24).
8. Other Comments  
   Several small molecules and pharmacological compounds have been developed to modulate AMPK activity, with some designed to specifically activate AMPK via the allosteric drug and metabolite (ADaM) site located at the interface between the alpha and beta subunits (kurumbail2016structureandregulation pages 6-9, smiles2024themetabolicsensor pages 2-3). Agents such as metformin and salicylate are known to indirectly activate AMPK and are under investigation for their potential in treating metabolic diseases including type 2 diabetes and non‑alcoholic fatty liver disease (wang2012ampactivatedproteinkinase pages 1-2, tarasiuk2022ampkanddiseases pages 5-6). Disease associations for AMPK alpha‑1 include its involvement in cancer, where alterations in upstream kinases like LKB1 and downstream effects on mTOR signaling have been noted, as well as in cardiac hypertrophy and neurodegenerative conditions where energy imbalance is a key factor (steinberg2009ampkinhealth pages 7-8, russell2020ampactivatedproteinkinase pages 4-6). Furthermore, aberrant post‑translational modifications such as increased ubiquitination or mis‑regulation of inhibitory phosphorylations have been linked to metabolic syndrome and insulin resistance (ovens2021posttranslationalmodificationsof pages 7-9, ovens2021posttranslationalmodificationsof pages 9-10). The kinase also exhibits tau‑protein kinase activity, with potential implications in tau pathology although the in vivo relevance of this activity remains to be fully established (arad2007ampactivatedproteinkinase pages 4-5). These additional layers of regulation and their disease associations underscore the clinical significance of AMPK alpha‑1 as a therapeutic target (smiles2024themetabolicsensor pages 26-27).
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