1. Phylogeny – The 5′-AMP-activated protein kinase catalytic subunit alpha-2, encoded by PRKAA2 (also known as AMPKα2 or AMPK2), is a member of the AMP-activated protein kinase (AMPK) family and is evolutionarily conserved across eukaryotes, being present in species ranging from yeast to mammals (horikoshi2006apolymorphismin pages 1-2). It belongs to the serine/threonine protein kinase group and, along with the alternate isoform encoded by PRKAA1, forms part of the ancient AMPK catalytic core that arose through gene duplication events early during eukaryotic evolution, as described in comparative kinome analyses (ross2016amp‐activatedproteinkinase pages 1-2, mccallum2015prkaa1andprkaa2 pages 7-12). Phylogenetic studies indicate that AMPKα2 clusters with other energy sensor kinases and can be traced back to the ancestral kinases present in the Last Eukaryotic Common Ancestor (LECA), sharing key structural and functional features with other kinases involved in cellular energy regulation such as those seen in the AGC and CAMK families (ross2016amp‐activatedproteinkinase pages 2-4, horikoshi2006apolymorphismin pages 1-2). Moreover, in the broader context of the human kinome, AMPKα2 is a 2R-ohnologue that emerged from the two rounds of whole-genome duplication that significantly expanded the protein kinase repertoire in vertebrates (ross2016amp‐activatedproteinkinase pages 1-2, randrianarisoa2020ampksubunitsharbor pages 4-5).
2. Reaction Catalyzed – The catalytic reaction mediated by PRKAA2 involves the transfer of a phosphate group from adenosine triphosphate (ATP) to specific serine/threonine residues on substrate proteins. In biochemical terms, the reaction can be represented as follows: ATP + [protein]–(L-serine or L-threonine) → ADP + [protein]–(L-serine/threonine)-phosphate + H⁺ (ross2016amp‐activatedproteinkinase pages 1-2).
3. Cofactor Requirements – The kinase activity of the PRKAA2 protein is dependent on the presence of Mg²⁺ as a cofactor, which is required to coordinate the binding of ATP within the active site and facilitate the phosphoryl transfer reaction (ross2016amp‐activatedproteinkinase pages 2-4).
4. Substrate Specificity – As a serine/threonine kinase, AMPKα2 phosphorylates substrates that often present consensus motifs characterized by the presence of basic residues in positions proximal to the target serine or threonine. Recent systematic studies on the substrate specificity of human serine/threonine kinases have demonstrated that many such kinases, including AMPK isoforms, exhibit a preferential sequence context, though for AMPK specifically the substrate recognition appears to be less strictly defined than in some other kinases (Johnson2023AnAtlas pages 759-766). In general, substrates of AMPKα2 include metabolic enzymes such as acetyl-CoA carboxylases (ACACA and ACACB), hydroxymethylglutaryl-CoA reductase (HMGCR), and other regulatory molecules that control lipid metabolism, glucose uptake, and protein synthesis (apoorv2018ampactivatedproteinkinase pages 5-8, mei2022effectofampk pages 6-7). The kinase may recognize target motifs that are enriched for hydrophobic and basic residues surrounding the phosphoacceptor site, thereby linking its substrate specificity to its central role in energy homeostasis (Johnson2023AnAtlas pages 759-766).
5. Structure – PRKAA2 consists of a central catalytic kinase domain that is highly conserved among serine/threonine kinases, featuring the typical bilobal structure with an N-terminal lobe harboring β-sheets and a C-terminal lobe predominated by α-helices (ross2016amp‐activatedproteinkinase pages 2-4). The kinase domain includes critical structural features such as an activation loop, wherein phosphorylation of threonine 172 (Thr172) is essential for full kinase activation, a C-helix that participates in the formation of a hydrophobic spine, and an ATP-binding pocket that accommodates Mg²⁺-coordinated ATP (lee2024catalyticisoformsof pages 1-2, ross2016amp‐activatedproteinkinase pages 4-6). Besides the kinase domain, additional regions may contribute to regulatory functions; an autoinhibitory domain adjacent to the catalytic core modulates basal activity and kinetics in response to conformational changes triggered by upstream signals (lee2024catalyticisoformsof pages 5-7, mccallum2015prkaa1andprkaa2 pages 30-34). Computational models and recent experimentally derived crystal structures or AlphaFold predictions reveal that AMPKα2’s C-terminal domain facilitates interactions with regulatory β and γ subunits, thereby integrating signals from cellular energy levels through binding of AMP, ADP, and ATP to the γ subunit (ross2016amp‐activatedproteinkinase pages 2-4, lee2024ampkisa pages 38-42).
6. Regulation – PRKAA2 activity is tightly regulated both allosterically and through post-translational modifications. A key regulatory event is the phosphorylation of Thr172 in the activation loop, primarily mediated by upstream kinases such as LKB1 and, under certain conditions, CaMKK, which dramatically increases the catalytic activity of the enzyme (ross2016amp‐activatedproteinkinase pages 1-2, mei2022effectofampk pages 6-7). AMPKα2 also harbors a serine residue (Ser491), which in contrast to the equivalent residue in AMPKα1, is a poor substrate for inhibitory phosphorylation by Akt and tends to be regulated by autophosphorylation events; such modifications modulate the enzyme’s sensitivity and sustain its activation under energy-depleted conditions (lee2024ampkisa pages 25-29, ross2016amp‐activatedproteinkinase pages 7-9). The binding of AMP (and ADP to a lesser extent) to the regulatory γ subunit not only promotes Thr172 phosphorylation but also protects it from dephosphorylation by protein phosphatases, thereby ensuring that PRKAA2 remains in an active conformation when cellular ATP levels are low (ross2016amp‐activatedproteinkinase pages 2-4, liu2024znfx1promotesampkmediated pages 10-13). In addition, changes in subunit composition and interactions with other proteins, such as those involved in metabolic signaling and autophagy (for example, interactions with ULK1 or IMPDH in specialized tissues) further modulate the activity of AMPKα2 (lee2024ampkisa pages 29-34, matthey reading from mccallum2015prkaa1andprkaa2 pages 7-12).
7. Function – PRKAA2 is a key regulator of cellular energy homeostasis. Upon sensing an increase in the AMP/ATP ratio—an indicator of energy stress—AMPKα2 becomes activated and phosphorylates a broad array of targets, resulting in a dual response: activation of catabolic pathways that generate ATP and repression of anabolic pathways that consume ATP (apoorv2018ampactivatedproteinkinase pages 5-8, mei2022effectofampk pages 6-7). Its substrates include enzymes involved in fatty acid and cholesterol biosynthesis, such as acetyl-CoA carboxylase (ACACA and ACACB) and hydroxymethylglutaryl-CoA reductase (HMGCR), thereby decreasing lipid and cholesterol synthesis (apoorv2018ampactivatedproteinkinase pages 5-8, monteverde2015evidenceofcancer‐promoting pages 4-6). Furthermore, AMPKα2 participates in the regulation of glucose homeostasis in liver and muscle by phosphorylating glycogen synthase and modulators of insulin signaling (ross2016amp‐activatedproteinkinase pages 7-9, matheny2017skeletalmusclepi3k pages 1-2). In the neurosensory retina, tissue-specific studies have shown that PRKAA2 plays distinct, non-redundant roles in rod photoreceptors; loss of PRKAA2 disrupts metabolic homeostasis, leading to structural and functional defects mediated at least in part through dysregulation of IMPDH activity and alterations in purine nucleotide levels (lee2024ampkisa pages 25-29, lee2024ampkisa pages 29-34). In addition to its metabolic roles, AMPKα2 is involved in broader cellular processes such as autophagy regulation through phosphorylation of ULK1, modulation of transcription via phosphorylation of key transcription regulators (e.g., FOXO3, ChREBP, CRTC2), and maintaining cell growth inhibition by down-regulating mTORC1 activity (ross2016amp‐activatedproteinkinase pages 2-4, mei2022effectofampk pages 6-7). Its expression is generally widespread, with significant levels in tissues such as skeletal muscle, liver, brain, and retina, where its activity is essential for adapting to energetic stress (apoorv2018ampactivatedproteinkinase pages 5-8, randrianarisoa2020ampksubunitsharbor pages 21-22).
8. Other Comments – PRKAA2 has garnered interest as a potential therapeutic target because its modulation affects metabolic balance and energy homeostasis in various diseases including type 2 diabetes mellitus, insulin resistance, cardiovascular disorders, and certain cancers; genetic polymorphisms in PRKAA2 have been associated with altered serum lipid profiles and susceptibility to metabolic diseases (randrianarisoa2020ampksubunitsharbor pages 20-21, horikoshi2006apolymorphismin pages 1-2). Additionally, in the context of cancer biology, while alterations in the expression or mutation status of AMPKα1 are more commonly linked to oncogenic processes, emerging evidence suggests that PRKAA2 may function as a tumor suppressor, as decreased expression or activity of AMPKα2 has been linked to enhanced tumor growth and metabolic reprogramming (ross2016amp‐activatedproteinkinase pages 7-9, monteverde2015evidenceofcancer‐promoting pages 4-6). Several small-molecule modulators and inhibitors have been developed to target various AMPK complexes, and while these agents can affect both AMPKα1 and AMPKα2 isoforms, ongoing research continues to delineate the isoform-specific effects that could lead to selective therapeutic strategies (su2022atrialampactivatedprotein pages 12-13, randrianarisoa2020ampksubunitsharbor pages 26-27).
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