1. Phylogeny  
The cAMP‐dependent protein kinase catalytic subunit alpha (PKAα), encoded by the PRKACA gene, is a prototypical member of the AGC kinase family and is highly conserved across eukaryotes. Orthologs of PRKACA have been identified in mammals, birds, fish, and many invertebrate species, reflecting an ancient origin in the common ancestor of eukaryotes. Gene duplication events early in vertebrate evolution led to the emergence of two closely related catalytic isoforms, PRKACA and PRKACB, which share approximately 93% sequence identity and preserve the essential catalytic motifs necessary for kinase activity (søberg2013evolutionarypathsof pages 1-2, søberg2013evolutionarypathsof pages 2-4). In addition, a transcribed retroposon, PRKACG, and kinases encoded on the sex chromosomes (PRKX and PRKY) further underscore the evolutionary branching within this kinase family. The high degree of conservation in the catalytic core, including the ATP-binding pocket, activation loop, and regulatory interfaces, highlights the fundamental role of PKAα in cAMP-mediated signaling throughout evolution (søberg2017evolutionofthe pages 1-2).

2. Reaction Catalyzed  
PKAα catalyzes a phosphorylation reaction in which the γ-phosphate from ATP is transferred to the hydroxyl group of serine or threonine residues on target proteins. The chemical reaction can be summarized as:  
  ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
This reaction is central to modulating the function of numerous substrates involved in metabolism, gene transcription, cell cycle regulation, and other critical cellular processes (turnham2016proteinkinasea pages 1-3, shabb2001physiologicalsubstratesof pages 1-5).

3. Cofactor Requirements  
The catalytic activity of PKAα is dependent on the presence of divalent metal ions. In particular, Mg²⁺ is required as an essential cofactor to coordinate ATP binding within the active site and to facilitate the transfer of the phosphate group to the substrate (taylor2005dynamicsofsignaling pages 1-2, turnham2016proteinkinasea pages 1-3).

4. Substrate Specificity  
PKAα exhibits a strong preference for phosphorylating serine/threonine residues that are embedded within a consensus sequence characterized by basic residues. Typically, its optimal recognition motif is defined as RRXS/T, where arginine residues at the –3 and –2 positions are critical for substrate interaction and alignment in the active site. Phosphoproteomic analyses have confirmed that the catalytic subunit preferentially targets substrates possessing these positively charged residues upstream of the phosphorylation site, thus ensuring high catalytic efficiency and fidelity. Notably, alterations in the substrate specificity have been observed in mutant forms of PRKACA, which modify the amino acid preferences surrounding the phosphoacceptor site (bathon2019alterationsinprotein pages 1-2, søberg2018themolecularbasis pages 8-9, taylor2022thetailsof pages 13-14).

5. Structure  
The three-dimensional structure of PKAα features a highly conserved catalytic core that adopts a bilobal architecture. The smaller N-terminal lobe is predominantly composed of β-sheets and a single conserved α-helix, whereas the larger C-terminal lobe is mainly α-helical and houses the catalytic machinery responsible for substrate binding and phosphoryl transfer. Within the catalytic domain, key structural elements include:  
 • The glycine-rich loop (P-loop), which is involved in anchoring and positioning ATP within the active site.  
 • The catalytic loop that contains a conserved lysine residue essential for orienting ATP for phosphotransfer, and the activation loop which, upon phosphorylation (notably at Thr197), stabilizes the active conformation of the kinase.  
 • The C-helix, whose proper positioning is critical for coordinating interactions between the ATP molecule and the substrate peptide.

Flanking the catalytic domain are variable N- and C-terminal tails that contribute to additional regulatory functions. The N-terminal tail, encoded by exon 1, comprises approximately 14 amino acids and contains a conserved myristoylation site at Gly1. This modification plays a crucial role in subcellular localization and might facilitate interactions with regulatory subunits and anchoring proteins. Other post-translational modifications, such as the deamidation of Asn2 and phosphorylation of Ser10 within the N-terminal region, further modulate PKAα conformation and stability. High-resolution crystal structures, complemented by predictive models such as those from AlphaFold, have delineated the active site architecture, the hydrophobic pockets that may accommodate the myristoyl moiety, and the interaction interfaces for inhibitor peptides like PKI (turnham2016proteinkinasea pages 3-4, taylor2022thetailsof pages 17-19).

6. Regulation  
The regulation of PKAα is achieved primarily through its association with regulatory subunits and by an array of post‐translational modifications. Under basal conditions, PKAα forms a holoenzyme complex with two regulatory subunits (either RI or RII isoforms), which inhibit its activity by occupying the catalytic cleft in a pseudosubstrate conformation. When intracellular cAMP levels rise, cAMP binds to specific domains within the regulatory subunits, inducing conformational changes that release the catalytic subunits and permit their full enzymatic activity (bathon2019alterationsinprotein pages 1-2, turnham2016proteinkinasea pages 1-3).

Additional regulation is mediated by post-translational modifications. The N-terminal myristoylation of PKAα, an irreversible modification at Gly1, is critical for targeting the kinase to particular cellular membranes and for interacting with A-kinase anchoring proteins (AKAPs). Moreover, phosphorylation events—most notably at Thr197 within the activation loop—are required for full activation and structural stabilization of the kinase. Other modifications, such as deamidation of Asn2 and phosphorylation at Ser10 in the N-terminal tail, can influence both the subcellular distribution and dynamic interactions of the enzyme with diverse protein partners (taylor2022thetailsof pages 17-19, søberg2018themolecularbasis pages 8-9).

Regulatory control is also exerted by downstream effectors. For instance, PKAα phosphorylates PJA2, a protein that binds to the regulatory subunits to promote their ubiquitination and proteasomal degradation. This feedback mechanism helps modulate the local abundance of regulatory subunits, thereby indirectly influencing the activity of the catalytic subunit (bathon2019alterationsinprotein pages 1-2, turnham2016proteinkinasea pages 6-8).

7. Function  
PKAα is a central mediator of cAMP‐dependent signaling cascades and is ubiquitously expressed in a wide range of tissues. Upon activation and release from the holoenzyme complex, the catalytic subunit translocates to both cytoplasmic and nuclear compartments, where it phosphorylates a diverse set of substrates. These substrates include key regulators of cell cycle progression, transcription factors, metabolic enzymes, and cytoskeletal proteins. Notable targets of PKAα include CDC25B, ABL1, NFKB1, CLDN3, and components of the proteasome complex (PSMC5/RPT6), as well as PJA2, RYR2, RORA, SOX9, and VASP (bathon2019alterationsinprotein pages 1-2, berthon2015prkacathecatalytic pages 1-2). Phosphorylation of histone H1 variants, for example, plays a role in chromatin condensation and mitotic progression, highlighting the kinase’s involvement in regulating gene expression and cell division (bathon2019alterationsinprotein pages 6-7).

Beyond its well-established role in modulating transcription factors such as CREB and RORA, PKAα is essential in mediating endocrine responses. It is required for processes such as glucose-mediated adipogenic differentiation and the inhibition of osteogenic differentiation in osteoblasts. The spatial restriction of its activity, achieved through interactions with AKAPs, ensures that phosphorylation events occur in specific subcellular domains, thereby fine-tuning cellular responses to hormonal signals (caretta2011proteinkinasea pages 1-3, turnham2016proteinkinasea pages 8-9).

Moreover, PKAα participates in feedback regulatory loops that control the abundance and compartmentalization of its regulatory subunits. By phosphorylating PJA2, it indirectly triggers the ubiquitination and subsequent degradation of these inhibitory subunits, a process that is crucial for resetting the cAMP signaling axis (bathon2019alterationsinprotein pages 1-2). Collectively, these functions position PKAα as a pivotal regulator of cell growth, differentiation, metabolism, and stress responses (caretta2011proteinkinasea pages 6-8, berthon2015prkacathecatalytic pages 1-2).

8. Other Comments  
A number of inhibitors of PKAα have been characterized in experimental systems. Endogenous inhibitors, such as the PKA inhibitor protein (PKI), act as potent pseudosubstrate inhibitors by binding directly to the catalytic cleft and thus preventing substrate phosphorylation. In addition, small-molecule inhibitors targeting the ATP-binding site of the kinase have been developed, although their specificity remains a subject of ongoing research (turnham2016proteinkinasea pages 1-3, shabb2001physiologicalsubstratesof pages 1-5).  
Mutations in the PRKACA gene, including the well-characterized L206R substitution, have been implicated in endocrine pathologies such as cortisol-producing adrenocortical adenomas, leading to Cushing syndrome. Such mutations compromise the normal interaction between the catalytic and regulatory subunits, resulting in constitutive kinase activation and aberrant phosphorylation of critical substrates (bathon2019alterationsinprotein pages 1-2, bathon2019alterationsinprotein pages 4-5). In addition, oncogenic fusion proteins involving PRKACA, for example the DNAJB1-PRKACA fusion identified in fibrolamellar hepatocellular carcinoma, further emphasize the pathological importance of dysregulated PKAα activity (turnham2016proteinkinasea pages 16-19). These disease associations highlight the clinical relevance of targeting PKAα in therapeutic strategies aimed at treating endocrine disorders and certain cancers (caretta2011proteinkinasea pages 1-3, stratakis2019calledanduncalled pages 1-1).

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