1. Phylogeny  
   Myotonin‐protein kinase (DMPK), also referred to as DM1 protein kinase, is a serine/threonine kinase that belongs to the AGC kinase group. Its closest phylogenetic relatives include the Rho‐associated kinases MRCK and ROCK, as well as citron kinase and other AGC members that regulate cytoskeletal dynamics. Orthologs of DMPK have been identified in mammals, and its domain architecture is evolutionarily conserved among vertebrates. The common structural organization—a leucine‐rich N‐terminal region, a catalytic kinase domain, a coiled‐coil segment, and alternatively spliced C‐terminal tails—indicates that DMPK emerged early in eukaryotic evolution and diversified to perform specialized functions in muscle, cardiac, and neural tissues (herpen2005divergentmitochondrialand pages 1-2, wansink2003alternativesplicingcontrols pages 1-2, arencibia2013agcproteinkinases pages 3-4).
2. Reaction Catalyzed  
   DMPK catalyzes the transfer of the γ‐phosphate group from ATP to specific serine or threonine hydroxyl groups on substrate proteins. The reaction can be represented as follows:  
     ATP + [protein]–(L‐serine or L‐threonine) → ADP + [protein]–(L‐serine/threonine)‐phosphate + H⁺  
   This phosphorylation event is essential for modulating the activity, localization, or interaction of the substrate proteins involved in muscle structure and ion homeostasis (magana2012myotonicdystrophyprotein pages 13-15, wansink2003alternativesplicingcontrols pages 11-12).
3. Cofactor Requirements  
   Consistent with the canonical mechanism of serine/threonine kinases, the catalytic activity of DMPK is Mg²⁺‐dependent. The binding of the Mg²⁺ ion facilitates the proper orientation of ATP within the active site and is essential for efficient phosphoryl transfer (wansink2003alternativesplicingcontrols pages 3-5).
4. Substrate Specificity  
   In vitro studies have demonstrated that DMPK exhibits a substrate specificity that is dependent on basic residues surrounding the phosphorylation site. The consensus motif recognized by DMPK can be represented as (R/K)XRRX(T/S)(L/V)X, where the presence of arginine or lysine residues N‐terminal to the target threonine or serine is critical for substrate recognition. This specificity aligns with its classification as a lysine/arginine‐directed kinase. Notably, substrates identified for DMPK include:  
     • The β‐subunit of the dihydropyridine receptor (DHPR), which plays a role in excitation–contraction coupling in skeletal muscle.  
     • Myosin phosphatase target subunit 1 (MYPT1), whose phosphorylation by DMPK contributes to the regulation of myosin light chain phosphatase activity and, consequently, muscle contractility.  
     • Phospholamban (PLN) and phospholemman (PLM), both of which are involved in the modulation of sarcoplasmic reticulum Ca²⁺ uptake and chloride currents in myocytes.  
   The precise amino acid preferences exemplified by the (R/K)XRRX(T/S)(L/V)X motif underscore DMPK’s role in signal transduction pathways critical for muscle function (pantic2012dmpkpreventsrosinduced pages 12-16, wansink2003alternativesplicingcontrols pages 11-12).
5. Structure  
   DMPK consists of a well‐defined multi‐domain structure that underpins its catalytic and regulatory functions. The structure can be divided into four principal regions:  
     • N‐terminal Leucine‐rich Domain: Present within the first ~70 amino acids, this domain is highly conserved among species and may be involved in protein–protein interactions that modulate localization or activity (herpen2005divergentmitochondrialand pages 1-2, magana2012myotonicdystrophyprotein pages 1-4).  
     • Catalytic Kinase Domain: Extending approximately from amino acid 71 to 339, this central domain embodies the canonical bilobal kinase structure. The N‐lobe features a β‐sheet scaffold while the C‐lobe is predominantly α‐helical; the two lobes create an ATP‐binding cleft. A critical lysine residue (commonly Lys100) is essential for ATP coordination, making this residue indispensable for catalytic activity (wansink2003alternativesplicingcontrols pages 3-5, sasagawa2003overexpressionofhuman pages 5-6).  
     • Coiled‐coil Region: Located C‐terminal to the catalytic domain, this α‐helical coiled‐coil segment promotes oligomerization and may facilitate substrate binding or interaction with regulatory proteins. The coiled‐coil region is implicated in the assembly of multiprotein complexes, particularly those associated with the mitochondrial outer membrane (wansink2003alternativesplicingcontrols pages 3-5, witherspoon2011insilicomining pages 19-27).  
     • Variable C‐terminal Tail: Due to alternative splicing, the C‐terminal tail exhibits considerable heterogeneity. Isoforms with longer hydrophobic tails tend to associate with cellular membranes such as the endoplasmic reticulum or mitochondrial outer membrane, whereas those with short tails remain cytosolic. A short five–amino acid motif (VSGGG) is present in some isoforms and modulates autophosphorylation efficiency, thereby influencing the overall conformation and activity of DMPK (wansink2003alternativesplicingcontrols pages 12-13, magana2012myotonicdystrophyprotein pages 27-29).  
   Recent structural predictions and crystallographic analyses support this modular organization and reveal that the flexible regions of DMPK, particularly within the activation loop and C‐terminal tail, play key roles in its allosteric regulation and substrate access (witherspoon2011insilicomining pages 19-27, arencibia2013agcproteinkinases pages 10-11).
6. Regulation  
   The activity of DMPK is finely tuned by several layers of regulation, including post‐translational modifications and alternative splicing. Major regulatory mechanisms include:

  • Autophosphorylation: DMPK can phosphorylate itself, and several autophosphorylation sites have been mapped, such as Ser234, Thr240, and Thr403. These phosphorylation events are critical for shifting DMPK into its catalytically active conformation and can influence its electrophoretic mobility (wansink2003alternativesplicingcontrols pages 8-9, magana2012myotonicdystrophyprotein pages 6-9).

  • Alternative Splicing: DMPK undergoes alternative splicing that yields several isoforms (A, B, C, D, E, F, and a minor G isoform). Variants differ mainly in their C‐terminal tails; these differences determine subcellular localization and modulate autophosphorylation rates. Isoforms with a hydrophobic tail are anchored to membranes such as the endoplasmic reticulum and mitochondria, whereas isoforms lacking these sequences are found primarily in the cytosol (wansink2003alternativesplicingcontrols pages 3-5, magana2012myotonicdystrophyprotein pages 25-27).

  • Protein–Protein Interactions: DMPK functions not only as a kinase but also as a scaffold protein. Notably, mitochondrial‐associated DMPK collaborates with hexokinase II (HK II) and Src tyrosine kinase on the outer mitochondrial membrane. Src‐mediated tyrosine phosphorylation of DMPK, along with reciprocal serine/threonine phosphorylation events, drives the assembly of this kinase complex, which is essential for the regulation of mitochondrial reactive oxygen species (ROS) and cell survival (pantic2012dmpkpreventsrosinduced pages 69-72, pantic2013myotonicdystrophyprotein pages 7-8).

  • Exogenous Modulators: Phosphatase inhibitors such as okadaic acid have been used to demonstrate that the phosphorylation status of DMPK is dynamically regulated. Treatment with okadaic acid induces an accumulation of the hyperphosphorylated form of DMPK, thereby altering its activity profile in vitro (wansink2003alternativesplicingcontrols pages 12-13, sasagawa2003overexpressionofhuman pages 5-6).

Collectively, these mechanisms ensure that DMPK activity is responsive to cellular conditions and that its signaling outputs are integrated within broader muscle and cardiac regulatory networks.

1. Function  
   DMPK is essential for the maintenance of muscle structure and function. Its biological roles include:

  • Skeletal Muscle Maintenance and Differentiation: DMPK is highly expressed in skeletal muscle, where it contributes to the stabilization of the cytoskeleton and the structural integrity of the nuclear envelope. Its phosphorylation activity on substrates such as MYPT1 influences myosin light chain phosphorylation and thereby regulates muscle contraction. During myogenesis, DMPK expression increases concomitant with myotube formation, highlighting its role in muscle cell differentiation (magana2012myotonicdystrophyprotein pages 11-13, wansink2003alternativesplicingcontrols pages 1-2).

  • Cardiac Function: In cardiac myocytes, DMPK phosphorylates key regulators such as phospholamban (PLN) and phospholemman (PLM). These phosphorylation events control sarcoplasmic reticulum Ca²⁺ uptake and modulate ion channel activity, which are critical for proper cardiac contractility and conduction. Deficiencies in DMPK function have been linked to conduction abnormalities and hypertrophic cardiomyopathy in animal models (magana2012myotonicdystrophyprotein pages 18-20, narang2000myotonicdystrophy(dm) pages 1-2).

  • Mitochondrial Integrity and ROS Regulation: Specific isoforms of DMPK localize to the outer mitochondrial membrane, where they participate in the assembly of a signaling complex comprising hexokinase II and Src kinase. This complex reduces mitochondrial superoxide levels and protects cells against ROS‐induced apoptosis, thereby contributing to cell survival under metabolic stress conditions (pantic2012dmpkpreventsrosinduced pages 12-16, pantic2012dmpkpreventsrosinduced pages 77-81, pantic2013myotonicdystrophyprotein pages 1-2).

  • Regulation of Gene Expression and Nuclear Envelope Integrity: DMPK has been implicated in controlling the expression of muscle-specific genes by maintaining the structural integrity of the nuclear envelope. This role is critical not only for myocyte differentiation but also for the long-term maintenance of muscle tissue architecture (magana2012myotonicdystrophyprotein pages 9-11).

By influencing diverse signaling pathways through phosphorylation events and scaffolding functions, DMPK plays a central role in coordinating the cellular processes essential for muscle contraction, structural integrity, and metabolic homeostasis.

1. Other Comments  
   DMPK is most notably implicated in myotonic dystrophy type 1 (DM1), a multisystemic disorder characterized by muscle wasting, myotonia, cardiac conduction defects, and metabolic disturbances. In DM1, pathogenic CTG repeat expansions in the 3′‐untranslated region of the DMPK gene lead to toxic RNA gain-of-function effects. The expanded mutant transcripts sequester RNA-binding proteins such as MBNL1, thereby disrupting normal alternative splicing patterns and contributing to disease pathogenesis (narang2000myotonicdystrophy(dm) pages 4-5, narang2000myotonicdystrophy(dm) pages 5-6). Although the CTG expansion primarily impacts RNA function rather than directly altering the protein sequence, altered expression levels, mislocalization, and dysregulation of DMPK kinase activity have been observed in affected tissues.

Currently, no clinical inhibitors selectively targeting DMPK have been established. However, its incorporation into multi-protein complexes—such as the HK II–Src complex on mitochondria—and its regulatory interactions make it an attractive candidate for future therapeutic modulation. Given its central role in muscle maintenance, cardiac contractility, and mitochondrial redox homeostasis, continued research into the regulatory mechanisms and potential inhibitors of DMPK may provide novel insights into therapeutic strategies for DM1 and related myopathies (arencibia2013agcproteinkinases pages 4-4, pantic2012dmpkpreventsrosinduced pages 69-72).

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