1. Phylogeny  
   ATM (Ataxia Telangiectasia Mutated) is a member of the phosphatidylinositol 3-kinase-related protein kinase (PIKK) family, a group of large serine/threonine kinases conserved from yeast to humans that also includes ATR, DNA-PKcs, mTOR, SMG1, and TRRAP (awasthi2016atmandatr pages 1-2). Its presence in all eukaryotes and close evolutionary relationship with other PIKK members place ATM within an ancient kinase clade that predates the divergence of metazoans and fungi, demonstrating its central role in genome maintenance (menolfi2020atmatrand pages 1-2). Orthologs of ATM are found in organisms ranging from yeast (where the homolog is known as Tel1) to higher mammals, reflecting the evolutionary conservation of its DNA damage sensing and signal transduction functions (pavletich2022structureofthe pages 5-8). Phylogenetic analysis reveals that while the overall kinase domain and associated regulatory regions such as the FAT and FATC domains are conserved, ATM also exhibits a large, divergent N-terminal region composed of HEAT repeats which serve as scaffolding modules for protein–protein interactions (ueno2022atmfunctionsof pages 1-2). Comparative genomics studies indicate that ATM has maintained critical domains responsible for catalysis and regulatory mechanisms throughout evolution, underscoring its essential role in coordinating the cellular response to genotoxic stress (garcia2022targetingtheatm pages 1-2).
2. Reaction Catalyzed  
   The enzymatic reaction catalyzed by ATM involves the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues in substrate proteins, with a strong preference for residues immediately followed by a glutamine (S/T-Q motifs) (armstrong2019atmdysfunctionin pages 2-4). In chemical terms, the reaction can be succinctly represented as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(phospho-L-serine/threonine) + H⁺, which is typical of serine/threonine kinases (awasthi2016atmandatr pages 1-2). This phosphorylation reaction is central to the activation of checkpoint signaling pathways, where the phosphorylated substrates include key proteins involved in DNA repair, cell cycle regulation, and apoptosis, thereby modulating downstream cellular processes (choi2016atmmutationsin pages 1-2). The mechanism involves the binding of ATP in the catalytic cleft of the kinase domain and subsequent nucleophilic attack by the substrate hydroxyl group facilitated by the conserved catalytic residues in ATM’s kinase domain (pavletich2022structureofthe pages 51-55).
3. Cofactor Requirements  
   The catalytic activity of ATM is dependent on the presence of divalent metal ions, with Mg²⁺ being essential for optimal kinase function as it coordinates with ATP to enable phosphoryl transfer (awasthi2016atmandatr pages 1-2). In addition to Mg²⁺, other divalent cations such as Mn²⁺ can sometimes substitute, although Mg²⁺ is the physiologically relevant cofactor required for its ATPase and kinase reactions (pavletich2022structureofthe pages 51-55). Binding of ATP in the active site is further stabilized by interactions with conserved residues, ensuring that the phosphate group is appropriately oriented for transfer to the substrate (awasthi2016atmandatr pages 1-2, pavletich2022structureofthe pages 51-55).
4. Substrate Specificity  
   ATM phosphorylates a broad range of substrates as part of its central role in the DNA damage response, and it exhibits strict substrate specificity for serine or threonine residues immediately followed by a glutamine, known as the S/T-Q motif (armstrong2019atmdysfunctionin pages 2-4). Well-characterized physiological substrates include the histone variant H2AX—phosphorylated at serine 139 to form γ-H2AX—which serves as a nuclear marker for double-strand breaks, as well as key regulators such as p53, CHK2, BRCA1, MRE11, and NBS1 (garcia2022targetingtheatm pages 2-4, lavin2015atmdependentphosphorylationof pages 7-10). The selective recognition depends not only on the S/T-Q consensus but also on surrounding amino acid sequences and, in many cases, on the structural context delivered by the substrate protein (phan2021atmmainfeatures pages 3-4). This substrate specificity framework enables ATM to orchestrate a complex phosphorylation network that rapidly mobilizes DNA repair machinery and enforces cell cycle checkpoints under conditions of genomic stress (choi2016atmmutationsin pages 1-2).
5. Structure  
   ATM is a large protein composed of 3056 amino acids (UniProt Q13315) with a modular organization that underpins its multifaceted regulatory roles (garcia2022targetingtheatm pages 14-16). Its structure comprises an extensive N-terminal region rich in HEAT repeats that form an elongated solenoid domain important for protein–protein interactions and recruitment by the MRN complex, followed by a C-terminal region that contains the FAT domain, the kinase domain, a PIKK regulatory domain (PRD), and a highly conserved FATC domain (pavletich2022structureofthe pages 1-5, stakyte2022structuralandfunctional pages 167-171). The kinase domain itself is organized into an N-lobe and a C-lobe, with a catalytic cleft that binds ATP and substrates, and contains critical elements such as the activation loop, catalytic loop, and key residues including a conserved lysine that coordinates ATP binding (baretic2017structuresofclosed pages 1-2, pavletich2022structureofthe pages 51-55). Structural studies using cryo-electron microscopy have revealed that ATM exists predominantly as an autoinhibited dimer in which the dimer interface, involving both the FAT and PRD regions, occludes the substrate-binding site, thereby maintaining low basal activity (baretic2017structuresofclosed pages 1-2, lavin2015atmdependentphosphorylationof pages 7-10). Upon activation by DNA damage and recruitment by the MRN complex, ATM undergoes conformational changes that lead to monomerization or a reorganization of the dimer, allowing access to the kinase active site (lee2021cellularfunctionsof pages 3-4, pavletich2022structureofthe pages 5-8). The structural architecture of ATM, with its multiple regulatory domains, not only facilitates its function in response to double-strand breaks but also allows it to integrate signals from oxidative stress and other forms of cellular stress (garcia2022targetingtheatm pages 18-23).
6. Regulation  
   ATM activity is tightly regulated through a combination of protein–protein interactions, post-translational modifications, and conformational changes that ensure its precise activation during the DNA damage response (lee2021cellularfunctionsof pages 1-2). In resting cells, ATM exists as a homodimer in an autoinhibited state, with the dimer interface, including the PRD region, restricting access to its catalytic site (baretic2017structuresofclosed pages 5-6, stakyte2022structuralandfunctional pages 43-46). Upon induction of double-strand breaks, the MRN complex (comprising MRE11, RAD50, and NBS1) recruits ATM to sites of damage through interactions with its N-terminal HEAT repeats; this recruitment is critical for ATM activation and subsequent autophosphorylation at multiple regulatory serine residues, notably serine 1981 (armstrong2019atmdysfunctionin pages 2-4, lavin2015atmdependentphosphorylationof pages 5-7). In addition to autophosphorylation, acetylation of lysine 3016 by the Tip60 acetyltransferase further promotes the structural rearrangement required for full activation (awasthi2016atmandatr pages 1-2, lee2021cellularfunctionsof pages 15-15). ATM is also responsive to oxidative stress, where reactive oxygen species induce disulfide bond formation—particularly involving cysteine 2991—triggering its activation independently of overt DNA damage (awasthi2016atmandatr pages 5-5, rotheneder2022insightsintothe pages 117-120). These regulatory inputs collectively convert ATM from an inactive dimer into an active monomer or an altered dimeric state capable of efficient substrate phosphorylation, thereby ensuring a rapid and robust cellular response to genotoxic insults (lee2021cellularfunctionsof pages 2-3, pavletich2022structureofthe pages 28-32).
7. Function  
   ATM functions as a master regulator of the cellular response to DNA double-strand breaks and other genotoxic stresses by activating checkpoint signaling pathways that coordinate DNA repair, cell cycle arrest, and apoptosis (armstrong2019atmdysfunctionin pages 2-4, ueno2022atmfunctionsof pages 1-2). One of its key roles is the phosphorylation of histone variant H2AX at serine 139 (forming γ-H2AX), which serves as an initial beacon for recruiting additional repair factors to sites of damage (garcia2022targetingtheatm pages 2-4). ATM also phosphorylates a wide range of substrates including p53, CHK2, BRCA1, and MRE11, thereby integrating signals from DNA damage to effect cell cycle arrest and promote homologous recombination repair (lavin2015atmdependentphosphorylationof pages 7-10, choi2016atmmutationsin pages 1-2). In addition to its classic role in response to DNA damage, ATM has been implicated in other cellular processes such as the control of pre-B cell allelic exclusion, where it contributes to the repositioning of the unrearranged immunoglobulin heavy chain allele to heterochromatin to enforce clonality in B-cells (armstrong2019atmdysfunctionin pages 2-4). Moreover, ATM participates in regulating cellular metabolism and oxidative stress responses, including the induction of pexophagy via phosphorylation of peroxisomal protein PEX5, and can modulate pathways involved in the stabilization of key transcription factors and apoptotic regulators (awasthi2016atmandatr pages 8-8, lee2021cellularfunctionsof pages 2-3). These diverse functions illustrate ATM’s pivotal role in preserving genomic integrity and how its loss or dysfunction is closely associated with neurodegeneration, immunodeficiency, and increased cancer risk as observed in patients with ataxia–telangiectasia (choi2016atmmutationsin pages 1-2, ueno2022atmfunctionsof pages 2-4).
8. Other Comments  
   ATM is an attractive therapeutic target given its central role in maintaining genomic stability, and several small-molecule inhibitors have been developed to inhibit its kinase activity in tumor cells with defective DNA repair pathways (sharma2025medicinalchemistrybreakthroughs pages 1-2, sharma2025medicinalchemistrybreakthroughs pages 2-4). Experimental compounds such as KU-55933, KU-60019, and newer generation inhibitors like AZD0156 have been shown to sensitize cancer cells to ionizing radiation and DNA-damaging chemotherapeutic agents, particularly in tumors with p53 deficiency or other DNA damage response defects (choi2016atmmutationsin pages 10-11, menolfi2020atmatrand pages 5-7). In addition, ATM mutations, whether inherited as in ataxia–telangiectasia or acquired somatically in cancers such as pancreatic ductal adenocarcinoma, lymphoid malignancies, and breast cancer, underscore its significance as both a tumor suppressor and a determinant of treatment response (armstrong2019atmdysfunctionin pages 1-2, garcia2022targetingtheatm pages 2-4). Beyond cancer therapeutics, research continues to unravel ATM’s non-canonical roles in metabolic regulation, vesicle transport, and cellular stress responses, further broadening our understanding of its functional repertoire (awasthi2016atmandatr pages 8-8, lee2021cellularfunctionsof pages 15-15). Notable mutations in the ATM gene often map to the kinase domain or regulatory regions such as the FAT, PRD, and FATC domains, and these mutations can impair phosphorylation of downstream substrates, leading to genomic instability and disease phenotypes (choi2016atmmutationsin pages 1-2, pavletich2022structureofthe pages 51-55). Active areas of research include detailed structural characterization by cryo-EM, elucidation of the mechanistic basis of dimer-to-monomer transition, and the development of more potent and selective ATM inhibitors with improved pharmacokinetic profiles for clinical use (baretic2017structuresofclosed pages 9-9, pavletich2022structureofthe pages 28-32).
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