1. Phylogeny  
   The ataxia telangiectasia mutated (ATM) protein is an evolutionarily conserved serine/threonine kinase that belongs to the phosphatidylinositol 3-kinase–related kinase (PIKK) family, a large family of atypical protein kinases that also includes ATR, DNA‐PKcs, mTOR, and hSMG1 (barila2013molecularbasesof pages 1-3, derheimer2010multiplerolesof pages 1-2). Phylogenetic studies indicate that the PIKK family is ancient and its members can be traced back to early eukaryotic evolution, with ATM present in all higher eukaryotes; orthologs exist in diverse metazoans and mammals, ensuring the fidelity of the DNA damage response across species (barila2013molecularbasesof pages 25-28, mckinnon2012atmandthe pages 1-2). Within the human kinome, ATM is grouped in a distinct branch of the PIKK family that serves as a central regulator of genome stability, and its evolutionary relationship with ATR and DNA‐PKcs is supported by their shared domain architecture and substrate preferences (barila2013molecularbasesof pages 3-6, ditch2012theatmprotein pages 1-2).
2. Reaction Catalyzed  
   ATM catalyzes the phosphorylation of substrate proteins by transferring the γ‐phosphate from ATP to serine or threonine residues in target proteins. The general reaction can be formulated as follows:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (barila2013molecularbasesof pages 1-3, lavin2007atmactivationand pages 1-2). This reaction is critical for propagating DNA damage signals to downstream effectors and enacting cell cycle checkpoints.
3. Cofactor Requirements  
   The catalytic activity of ATM requires the presence of divalent cations, with Mg²⁺ being the essential cofactor needed to coordinate ATP and facilitate efficient phosphoryl transfer. Although specific experimental details on additional cofactors are not always provided in every study, the requirement for Mg²⁺ is consistent with the biochemical properties of serine/threonine kinases of the PIKK family (barila2013molecularbasesof pages 1-3, mand2015mechanismsandconsequences pages 13-18).
4. Substrate Specificity  
   ATM displays a preference for phosphorylation sites that conform to a consensus sequence consisting of a serine or threonine residue followed by a glutamine residue (the [ST]-Q motif). This substrate specificity has been demonstrated by its action on key targets such as the histone variant H2AX (phosphorylation at Ser139), p53, checkpoint kinase 2 (CHEK2), and numerous other proteins involved in the DNA damage response (barila2013molecularbasesof pages 3-6, xu2014theversatilefunctions pages 1-2). The intrinsic substrate specificity of ATM is well documented in recent atlas studies of the human serine/threonine kinome, which highlight a consistent preference for S/T-Q motifs (barila2013molecularbasesof pages 3-6, xu2014theversatilefunctions pages 2-4).
5. Structure  
   ATM is a large protein kinase with a molecular mass of approximately 350–370 kDa and is composed of multiple distinct domains that are essential for its function. The N-terminal region of ATM consists of numerous HEAT repeats – α-helical motifs that form an extended solenoid structure and are thought to serve as scaffolds for protein–protein interactions and flexible docking of regulatory factors (cremona2014atmsignallingand pages 1-2, lavin2008ataxiatelangiectasiafroma pages 1-2). Following the HEAT repeats, ATM contains a conserved FAT (FRAP–ATM–TRRAP) domain that is important for maintaining the structural integrity of the kinase. This domain is intimately associated with the central catalytic kinase domain, which shares a structural homology with phosphoinositide 3-kinases; the kinase domain is responsible for the actual phosphotransfer reaction (cremona2014atmsignallingand pages 1-2, mand2015mechanismsandconsequences pages 13-18). Adjacent to the kinase domain, ATM harbors the PIKK regulatory domain (PRD) and a FAT-C terminal (FATC) domain that plays a critical role in the proper folding and stability of the active enzyme as well as in its regulation by post-translational modifications (lau2016structureofthe pages 8-8, mand2015mechanismsandconsequences pages 29-34). Moreover, structural studies using electron microscopy have demonstrated that ATM exists as an inactive dimer in the absence of DNA damage, with the kinase active sites occluded; activation involves autophosphorylation and dimer dissociation, producing active monomeric forms capable of engaging substrates (lavin2007atmactivationand pages 1-2, shiloh2013theatmprotein pages 5-6).
6. Regulation  
   ATM is regulated at multiple levels through post-translational modifications and dynamic protein–protein interactions. A key regulatory event in the ATM activation cycle is autophosphorylation at serine 1981, which is widely used as a marker for ATM activation following the presence of double-strand breaks (barila2013molecularbasesof pages 1-3, lavin2007atmactivationand pages 1-2). In addition to autophosphorylation, ATM is further regulated by phosphorylation at several other sites, including serines 367, 1893, and 2996, that modulate its kinase activity and influence downstream signaling (barila2013molecularbasesof pages 13-15, mand2015mechanismsandconsequences pages 24-29). Acetylation also plays a significant role in ATM regulation; for example, acetylation by the histone acetyltransferase Tip60 at specific lysine residues within the FATC domain (notably Lys3016 in some reports) is essential for full kinase activation (barila2013molecularbasesof pages 1-3, cremona2014atmsignallingand pages 1-2). Furthermore, ATM can be activated in a DNA damage–independent manner under conditions of oxidative stress through the formation of intermolecular disulfide bonds, with critical cysteine residues (such as Cys2991) contributing to the redox-dependent activation process (barila2013molecularbasesof pages 8-10, mand2015mechanismsandconsequences pages 18-24). Negative regulation is mediated by dephosphorylating enzymes including protein phosphatase 2A (PP2A), PP5, and the phosphatase WIP1, which work to reset ATM activity after repair is complete (lavin2007atmactivationand pages 6-7, mand2015mechanismsandconsequences pages 24-29). The interaction of ATM with the Mre11-Rad50-Nbs1 (MRN) complex is also essential; the MRN complex acts as a sensor of double-strand breaks and recruits ATM to sites of damage, enhancing its activation and substrate targeting (barila2013molecularbasesof pages 3-6, mckinnon2012atmandthe pages 3-5).
7. Function  
   ATM functions as a master regulator of the cellular response to DNA double-strand breaks, orchestrating a network of signaling pathways that culminate in cell cycle arrest, DNA repair, apoptosis, or senescence when genomic integrity is compromised. Upon activation, ATM phosphorylates hundreds of substrates, including the histone variant H2AX at serine 139 (forming γ-H2AX), which serves to recruit additional DNA repair factors and facilitate chromatin remodeling (barila2013molecularbasesof pages 3-6, lavin2007atmactivationand pages 2-3). Key substrates of ATM include p53, checkpoint kinase 2 (CHEK2), BRCA1, and NBS1; through these phosphorylation events, ATM enforces cell cycle checkpoints at the G1/S, intra-S, and G2/M transitions to allow time for repair or to trigger programmed cell death if damage is irreparable (barila2013molecularbasesof pages 17-19, lee2021cellularfunctionsof pages 1-2). In the context of the immune system, ATM is also involved in pre-B cell allelic exclusion by mediating the repositioning of the unrearranged immunoglobulin heavy chain allele to pericentromeric heterochromatin, thereby ensuring monospecificity of B-cell antigen receptors (information section). Additionally, ATM has been implicated in processes such as replication-dependent histone mRNA degradation and vesicle or protein transport, and it plays roles in signaling pathways that affect insulin signaling, pexophagy, and the regulation of receptor tyrosine kinase pathways such as MET (information section, barila2013molecularbasesof pages 17-19, xu2014theversatilefunctions pages 2-4). ATM’s functions extend to both the nuclear compartment, where it coordinates DNA repair and checkpoint responses, and to the cytoplasm, where it modulates responses to oxidative stress and mitochondrial homeostasis (barila2013molecularbasesof pages 22-25, lee2021cellularfunctionsof pages 15-15).
8. Other Comments  
   ATM is a clinically significant protein with numerous inhibitors developed for experimental and therapeutic purposes. Specific ATP-competitive inhibitors, such as KU-55933 with an IC₅₀ of approximately 13 nM and its more potent analogue KU-60019, have been used to study ATM function and sensitization of tumors to DNA-damaging therapies; another agent, CP466722, is noted for its rapid and reversible inhibition of ATM without affecting ATR (barila2013molecularbasesof pages 17-19). In addition to its role in the DNA damage response, ATM’s involvement in pathways such as redox signaling, pexophagy mediated by phosphorylation of the peroxisomal receptor PEX5, and the regulation of metabolic processes places it at the crossroads of several critical cellular functions (information section, barila2013molecularbasesof pages 8-10). Mutations in the ATM gene are causative for the autosomal recessive disorder ataxia telangiectasia (A-T), which is characterized by neurodegeneration, immunodeficiency, radiosensitivity, cancer predisposition (notably lymphoid malignancies, breast cancer, and other tumors), and metabolic abnormalities including insulin resistance (information section, lavin2008ataxiatelangiectasiafroma pages 10-10, stankovic2014theroleof pages 5-7). Recent studies have also highlighted ATM’s role in regulating vesicle and protein transport and in modulating the stability of key proteins such as DYRK2 through phosphorylation events that prevent proteasomal degradation (information section). Known disease-associated mutations often lead to truncated or unstable ATM proteins, thereby compromising the kinase’s ability to initiate appropriate DNA damage responses and maintain genomic integrity (barila2013molecularbasesof pages 25-28, mand2015mechanismsandconsequences pages 29-34). These multifaceted roles have prompted ongoing research into targeted inhibitors and imaging agents for ATM, and an increasing interest in exploiting ATM dysfunction in cancer therapy (huang2021sensorsandinhibitors pages 1-2, berger2017atmdependentpathwaysof pages 11-11).
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