1. Phylogeny  
   ATM (Ataxia Telangiectasia Mutated) is a large serine/threonine kinase that is a member of the phosphatidylinositol 3‐kinase‐related kinase (PIKK) family. This evolutionary conserved family also comprises ATR, DNA-PKcs, mTOR, SMG1 and TRRAP, and its origin can be traced back to early eukaryotes, reflecting an ancient origin found in organisms ranging from yeast to mammals (bhatti2011atmproteinkinase pages 1-3, shiloh2013theatmprotein pages 2-3). Orthologs of ATM have been identified in all metazoans with conservation of key domains such as the FAT, kinase and FATC domains, which are the characteristic hallmarks of the PIKK family (bhatti2011atmproteinkinase pages 1-3, ueno2022atmfunctionsof pages 1-2). Evolutionarily, ATM shares structural and mechanistic similarities with yeast Tel1, and the divergence within the PIKK family is thought to have occurred prior to the evolution of higher eukaryotes, situating ATM within a core group of kinases essential for maintaining genomic integrity (lee2021cellularfunctionsof pages 2-3, pavletich2022structureofthe pages 5-8).
2. Reaction Catalyzed  
   ATM catalyzes the transfer of a phosphate group from ATP to serine or threonine residues in its target proteins. The reaction can be summarized as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺. This phosphorylation is central to the activation of numerous substrates involved in the DNA damage response (shiloh2013theatmprotein pages 1-2, putti2021atmkinasedead pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of ATM is dependent on the presence of magnesium ions (Mg²⁺). Mg²⁺ ions are required for proper ATP binding and kinase activity, functioning as essential cofactors to facilitate the phosphoryl transfer reaction (bhatti2011atmproteinkinase pages 1-3, pavletich2022structureofthe pages 1-5).
4. Substrate Specificity  
   ATM recognizes and phosphorylates substrates primarily on serine/threonine residues that are immediately followed by a glutamine residue, i.e. the SQ or TQ motifs. This consensus motif is critical for substrate recognition and ensures that ATM selectively targets proteins involved in chromatin remodeling, DNA repair, and cell cycle checkpoint control. Notable substrates include the histone variant H2AX (phosphorylated on serine-139 to form γH2AX), p53, Chk2, and several components of the MRN complex (shiloh2013theatmprotein pages 1-2, putti2021atmkinasedead pages 1-2, bhatti2011atmproteinkinase pages 20-22).
5. Structure  
   ATM is organized into several distinct domains that collectively mediate its catalytic and regulatory functions. The N-terminal region is composed largely of HEAT repeat motifs that form a flexible solenoid and provide a scaffold for protein-protein interactions and binding of partner proteins (pavletich2022structureofthe pages 5-8, bhatti2011atmproteinkinase pages 3-4). Downstream of the HEAT repeats, the C-terminal region of ATM contains the highly conserved FAT domain, the central kinase domain and the short FATC domain. The FAT domain plays a dual role in structural stability and autoinhibitory regulation, as it forms extensive contacts that help maintain ATM in an inactive dimeric state prior to activation (pavletich2022structureofthe pages 51-55, bhatti2011atmproteinkinase pages 7-9). The kinase domain is structurally similar to phosphatidylinositol 3-kinases, possessing an N-lobe and a C-lobe that form the catalytic cleft where ATP binds; critical lysine residues within the N lobe align ATP’s phosphate groups for phosphotransfer (lau2016structureofthe pages 8-8, pavletich2022structureofthe pages 21-25). The FATC domain at the extreme C-terminus is essential for full catalytic activity and proper folding, and is known to undergo post-translational modifications such as acetylation (bhatti2011atmproteinkinase pages 3-4, putti2021atmkinasedead pages 11-13). Moreover, ATM exists in an inactive homodimeric form in resting cells; activation by DNA double-strand breaks leads to autophosphorylation (notably at Ser1981) and dissociation into active monomers (shiloh2013theatmprotein pages 1-2, lau2016structureofthe pages 1-2).
6. Regulation  
   ATM regulation is multifaceted and involves several layers of control. In response to DNA double-strand breaks, ATM is recruited to the sites of damage by the MRN complex (MRE11-RAD50-NBS1) and is activated by autophosphorylation events following conformational changes in its dimeric structure (shiloh2013theatmprotein pages 7-8, bhatti2011atmproteinkinase pages 9-10). Post-translational modifications are critical for the fine-tuning of ATM activity. Key modifications include autophosphorylation at Ser1981, which is required for dissociation of the inactive dimer to form the active monomer (shiloh2013theatmprotein pages 1-2, putti2021atmkinasedead pages 1-2). In addition, the acetylation of ATM by the Tip60 histone acetyltransferase, particularly at lysine residues such as K3016, is necessary for full activation (bhatti2011atmproteinkinase pages 16-18, shiloh2013theatmprotein pages 14-14). Other regulatory mechanisms involve phosphorylation-dependent interactions with downstream effectors (e.g., phosphorylation of p53, CHK2, and BRCA1) and the action of phosphatases such as PP2A and PP5, which serve to dephosphorylate and thereby inactivate ATM once the DNA damage response is resolved (oberle2010regulationofthe pages 3-4, shiloh2013theatmprotein pages 5-6). Furthermore, oxidative stress can independently activate ATM through disulfide bond formation involving specific cysteine residues, thereby linking ATM’s activity to the cellular redox state (bhatti2011atmproteinkinase pages 20-22, shiloh2013theatmprotein pages 6-7).
7. Function  
   The biological role of ATM centers on its function as a master regulator of the cellular response to DNA damage and genotoxic stress. Upon sensing DNA double-strand breaks, ATM phosphorylates a wide array of substrates that coordinate cell cycle checkpoints, DNA repair processes (including homologous recombination and non-homologous end joining), and apoptosis. For example, phosphorylation of histone H2AX at Ser-139 facilitates the recruitment of DNA repair complexes, while modification of p53 leads to cell cycle arrest or programmed cell death as required (shiloh2013theatmprotein pages 1-2, putti2021atmkinasedead pages 1-2). Beyond nuclear DNA repair, ATM activity is implicated in other cellular pathways; it is involved in the regulation of the immune response through control of pre-B cell allelic exclusion, in vesicle and protein transport, and in signal transduction processes that affect cell cycle progression (bhatti2011atmproteinkinase pages 20-22, putti2021atmkinasedead pages 2-3). Additionally, ATM plays roles in metabolism and oxidative stress response by modulating pathways that influence mitochondrial function and pexophagy, as evidenced by its phosphorylation of proteins like PEX5 in response to reactive oxygen species (ueno2022atmfunctionsof pages 13-14, shiloh2013theatmprotein pages 14-14). Expression of ATM is ubiquitous, with particularly high expression in tissues where genomic maintenance is critical, such as the brain, immune organs, and dividing somatic cells (amirifar2019ataxia‐telangiectasiaareview pages 1-4, shiloh2013theatmprotein pages 11-12).
8. Other Comments  
   ATM is a key tumor suppressor and its dysfunction is associated with a number of clinical conditions. Germline mutations in the ATM gene cause Ataxia-Telangiectasia (A-T), a recessive disorder characterized by neurodegeneration, immunodeficiency, predisposition to cancer (especially lymphoid malignancies), and radiosensitivity (bhatti2011atmproteinkinase pages 20-22, amirifar2019ataxia‐telangiectasiaareview pages 16-19). In cancer, heterozygous mutations as well as somatic mutations in ATM can contribute to genomic instability and influence treatment response; in particular, “kinase-dead” mutations where ATM protein is expressed but lacks catalytic activity have been shown to enhance oncogenic phenotypes and modulate sensitivity to specific chemotherapy agents such as PARP inhibitors and topoisomerase inhibitors (putti2021atmkinasedead pages 7-8, putti2021atmkinasedead pages 8-10). Several experimental inhibitors targeting ATM kinase activity are under investigation in preclinical studies and early clinical trials, with the goal of sensitizing tumor cells to DNA-damaging agents and radiotherapy (williams2020structuresandregulations pages 8-8, shiloh2013theatmprotein pages 6-7). These inhibitors, while demonstrating potential therapeutic benefits, require further development to achieve specificity given ATM’s broad role in genome maintenance (putti2021atmkinasedead pages 7-8, shiloh2013theatmprotein pages 14-14).
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