1. Phylogeny  
   mTOR is an evolutionarily ancient serine/threonine kinase that belongs to the phosphatidylinositol 3-kinase-related kinase (PIKK) family, a group of atypical kinases highly conserved among eukaryotes. Its orthologous relationships span from lower eukaryotes such as yeasts and fungi to higher mammals including Homo sapiens. Within the kinome, mTOR is part of a core set of nutrient sensing and growth regulatory proteins that emerged in the last eukaryotic common ancestor (LECA), alongside other key components such as Rheb, TSC1/2, LST8, RAPTOR, RICTOR, and SIN1. Comparative genomic analyses, as described in multiple fungal studies, reveal the presence of a single TOR gene in most eukaryotic organisms; however, some fungi possess duplicate TOR genes potentially resulting from lineage‐specific gene duplications (song2024thetorsignalling pages 1-2, tatebe2017evolutionaryconservationof pages 1-4). Phylogenetic reconstruction based on conserved domains—HEAT repeats at the N-terminus, FAT, FRB, kinase, and FATC domains at the C-terminus—confirms that mTOR orthologs cluster together according to their evolutionary relationships, and these conserved motifs underline its fundamental role in nutrient, energy, and growth factor signaling (tatebe2017evolutionaryconservationof pages 4-6). The kinase’s membership in the PIKK family, sharing key catalytic and regulatory features with DNA-PKcs, ATM, and ATR, further supports its deep evolutionary conservation in the eukaryotic lineage (song2024thetorsignalling pages 2-5, OpenTargets Search: -MTOR).
2. Reaction Catalyzed  
   mTOR catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on substrate proteins. The general reaction is: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(phospho-L-serine/threonine) + H⁺. As a serine/threonine kinase, mTOR phosphorylates at least 800 substrates directly or indirectly, thereby modulating various aspects of cell growth, metabolism, and survival (Information section). The reaction mechanism follows that of canonical protein kinases where binding of ATP and the substrate in the active site is coordinated by structural motifs in the kinase domain—including the glycine-rich loop and conserved catalytic residues—thus facilitating phosphoryl transfer (coldron2022biochemicalanalysisof pages 18-20).
3. Cofactor Requirements  
   The catalytic function of mTOR, as with many kinases, requires divalent metal ions; in most cases, magnesium ions (Mg²⁺) are essential. Mg²⁺ coordinates with the phosphate groups of ATP, thereby stabilizing the transition state during the phosphoryl transfer process. While the detailed studies on cofactor dependency specific to mTOR are not exhaustively detailed in the provided excerpts, the general consensus for serine/threonine kinases within the PIKK family implies a Mg²⁺ requirement (coldron2022biochemicalanalysisof pages 18-20).
4. Substrate Specificity  
   mTOR exerts its influence by phosphorylating a wide range of substrates involved in anabolic metabolism, translation regulation, and autophagy. Physiologically relevant substrates include EIF4EBP1 (eukaryotic translation initiation factor 4E-binding protein 1) and the ribosomal protein S6 kinases (RPS6KB1 and RPS6KB2). These phosphorylation events result in the activation of translation by relieving inhibition of eiF4E and by modulating protein synthesis machinery (Information section). Although an explicit consensus sequence for mTOR phosphorylation is not as well defined as for some smaller kinases, its substrate recognition appears to be influenced by the proximity of docking motifs or binding domains such as the TOS motif on targets like S6K, which facilitates interaction with mTORC1 via RAPTOR (song2024thetorsignalling pages 2-5, tatebe2017evolutionaryconservationof pages 6-8). In addition, mTOR modifies regulators of autophagy such as ULK1, phosphorylating it at sites involved in the negative regulation of autophagy under nutrient-rich conditions (Information section). This broad substrate profile underscores mTOR’s role as a master regulator with a versatile substrate-selective mechanism dependent on protein–protein interaction interfaces (song2024thetorsignalling pages 18-19).
5. Structure  
   mTOR is a large multidomain protein with a predicted molecular weight of approximately 280 kDa. Its structure is organized into several distinct domains that contribute to both catalytic and regulatory functions. Starting at the N-terminus, a series of HEAT repeats—named for their occurrence in huntingtin, elongation factor 3, PP2A, and TOR1—provide a platform for protein–protein interactions and serve as scaffolding elements necessary for complex assembly (song2024thetorsignalling pages 2-5, tatebe2017evolutionaryconservationof pages 4-6). Following the HEAT repeats is the FAT domain (named for FRAP, ATM, TRRAP), which further contributes to the structural integrity and proper folding of the kinase. The FRB (FKBP12-rapamycin binding) domain is located immediately after the FAT domain; this domain is key to the binding of the FKBP12-rapamycin complex, thereby mediating the potent inhibition of mTORC1 by rapamycin (song2024thetorsignalling pages 1-2, coldron2022biochemicalanalysisof pages 26-28). The central catalytic kinase domain belongs to the PIKK family and contains conserved motifs such as the glycine-rich loop, the conserved lysine in the β3 strand, and the DFG motif, which are essential for ATP binding, catalysis, and substrate positioning (coldron2022biochemicalanalysisof pages 20-26, tatebe2017evolutionaryconservationof pages 6-8). At the C-terminus, the FATC domain, a short but highly conserved motif, plays a critical role in regulating the catalytic activity and stability of the protein (song2024thetorsignalling pages 2-5, tatebe2017evolutionaryconservationof pages 8-11). High-resolution structural data from cryo-electron microscopy, particularly in mammalian mTORC1 complexes, have provided insights into the assembly and spatial organization of these domains, revealing that the HEAT repeats form solenoidal structures that interact symmetrically in the mTOR homodimer (tatebe2017evolutionaryconservationof pages 13-15, song2024thetorsignalling pages 2-5).
6. Regulation  
   mTOR’s activity is tightly regulated in response to a multitude of intracellular and extracellular signals. One of the best characterized regulatory mechanisms is the binding of the immunosuppressant rapamycin in complex with the FKBP12 protein. This ternary complex specifically interacts with the FRB domain to inhibit mTORC1 activity without affecting mTORC2 under most conditions (song2024thetorsignalling pages 1-2, song2024thetorsignalling pages 5-6). Upstream regulators include the small GTPase Rheb, which, when in its GTP-bound state, activates mTORC1, and the TSC1/2 complex, which acts as a GTPase-activating protein to inhibit Rheb and thus mTORC1 (Information section, tatebe2017evolutionaryconservationof pages 16-17). mTOR activity is further controlled by nutrient signals, such as the availability of amino acids, which recruit mTORC1 to the lysosome via interactions involving the Rag GTPases and the Ragulator complex (song2024thetorsignalling pages 18-19, gu2022biochemicalandphysiological pages 76-80). In addition to classical phosphorylation, mTOR is subject to post-translational modifications that modulate its function. For instance, recent studies in immune and infectious contexts have highlighted the role of ISGylation at lysine 2066 within the FRB domain, which may affect autophagy regulation during infection (zhang2019theinvivo pages 7-8, zhang2019theinvivo pages 9-10). Moreover, feedback loops exist wherein mTORC1-mediated phosphorylation of proteins such as GRB10 results in the attenuation of upstream growth factor signaling, thus providing self-regulatory control over the pathway (Information section, song2024thetorsignalling pages 1-2). These complex layers of regulation—encompassing phosphorylation, small GTPase interactions, lysosomal recruitment, and covalent modifications—ensure that mTOR activity is finely tuned to the metabolic and environmental status of the cell (saksena2020proteinphosphatasesat pages 16-18, gu2022biochemicalandphysiological pages 171-173).
7. Function  
   mTOR is centrally involved in the regulation of cellular growth, metabolism, and survival. As a core component of two structurally and functionally distinct complexes, mTORC1 and mTORC2, mTOR coordinates diverse cellular processes. mTORC1 governs anabolic metabolism by promoting protein, lipid, and nucleotide synthesis, principally through the phosphorylation of targets such as EIF4EBP1, which releases its inhibition on the translation initiation factor eiF4E, and by activating S6K1 and S6K2, which in turn phosphorylate ribosomal proteins and other factors necessary for protein synthesis (Information section, song2024thetorsignalling pages 1-2). Furthermore, mTORC1 stimulates ribosomal biogenesis by activating RNA polymerase III-dependent transcription through the phosphorylation and inhibition of the MAF1 repressor (Information section). mTORC1 also plays a critical role in suppressing catabolic processes; for example, under nutrient-rich conditions, mTORC1 phosphorylates ULK1 to inhibit autophagy, thereby maintaining cellular energy homeostasis (Information section, song2024thetorsignalling pages 2-5). In parallel, mTORC2, which is generally insensitive to nutrient fluctuations, is primarily activated by growth factors. mTORC2 phosphorylates a range of AGC family kinases—most notably AKT (at residues such as Ser-473, Thr-450, and other context-dependent sites), various PKC isoforms, and SGK1—contributing to the regulation of cell proliferation, actin cytoskeletal organization, and metabolic homeostasis (Information section, song2024thetorsignalling pages 17-18, tatebe2017evolutionaryconservationof pages 16-17). In addition to its well-established roles in nutrient sensing and protein synthesis, mTOR impacts additional processes including lipid biosynthesis, mitochondrial biogenesis, and even regulatory feedback mechanisms on insulin signaling through modifications of substrates such as GRB10 (Information section, laskovs2024investigatingthemetabolic pages 25-28). Because of its central involvement in these pathways, dysregulation of mTOR has been implicated in numerous pathological conditions including cancers, metabolic disorders, neurodegeneration, and immune dysfunction (OpenTargets Search: -MTOR, attwood2021trendsinkinase pages 23-25).
8. Other Comments  
   Beyond its essential cellular functions, mTOR has attracted significant attention as a therapeutic target. Inhibitors such as rapamycin and its analogs (rapalogs) effectively suppress mTORC1 activity by binding to its FRB domain in complex with FKBP12, and new-generation ATP-competitive inhibitors have been developed to target both mTORC1 and mTORC2 simultaneously (song2024thetorsignalling pages 1-2, coldron2022biochemicalanalysisof pages 26-28). Clinically, mTOR inhibitors are currently used to treat diseases such as certain cancers, lymphangioleiomyomatosis (LAM), and, potentially, neurological disorders. mTOR’s extensive substrate network—with over 800 proteins being ultimately affected—further underscores its influence on cell metabolism and survival, rendering it a nexus within a vast regulatory network (Information section, OpenTargets Search: -MTOR). Notable mutations in upstream regulators, such as loss-of-function mutations in TSC1/TSC2 that lead to mTOR hyperactivation, have been linked to overgrowth syndromes and various neoplasias (Information section). In addition, emerging evidence on noncanonical post-translational modifications such as ISGylation suggests additional layers of regulatory control that may be exploited for therapeutic benefit (zhang2019theinvivo pages 7-8). Ongoing research focuses on understanding the detailed molecular mechanisms that govern mTOR’s interactions with its substrates and regulators, as well as on the development of more selective inhibitors that might offer improved efficacy with fewer side effects (marchi2024developmentofmodels pages 33-38, harachi2021proteinacetylationat pages 9-10). Due to its central role in integrating nutrient, energy, and growth factor signals, mTOR remains one of the most heavily studied kinases in biomedical research, and it is likely that further layers of regulation and additional substrates will be identified in the coming years (song2024thetorsignalling pages 18-19, gu2022biochemicalandphysiological pages 167-171).
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