TOR1 and TOR2 encode two closely related factors that regulate cell growth in response to nutrient availability and cellular stresses. TOR1 and TOR2 are involved in the regulation of many cellular processes including: protein synthesis, ribosome biogenesis, autophagy, transcriptional activation, meiosis, cell cycling, nutrient permease sorting and turnover, and actin organization. These processes are carried out by two functionally distinct TOR complexes. The TOR complex 1is responsible for most of the aforementioned processes and modulates translation initiation, inhibits protein turnover, contributes to tetrad formation, and represses the transcription of specific genes that are induced by nutrient starvation. TORC1 consists of either Tor1p or Tor2p, together with Kog1p, Lst8p, and Tco89p. TORC1 is sensitive to the drug rapamycin, which forms a complex with Fpr1p that binds to the Tor protein and inhibits complex activity. TOR complex 2is involved in regulating actin cytoskeleton polarization during cell cycle progression, cell wall integrity, and receptor endocytosis. TORC2 does not include Tor1p and contains only Tor2p along with Avo1p, Avo2p, Tsc11p, Lst8p, Bit61p, Slm1p, and Slm2p. TORC2 is rapamycin insensitive because the rapamycin-Fpr1p complex does not bind to Tor2p when it is present in TORC2.Tor1p and Tor2p are peripheral membrane proteinscomprised of 2470and 2474amino acid residues, respectively, and the two proteins have 80% overall amino acid similarity. The Tor proteins consist of several functional domains. The amino-terminal 1200 residues consist of stretches of HEATrepeats, which typically mediate protein-protein interactions. Following the HEAT repeats is a 550-amino acid-long FATdomain that has also been suggested to facilitate protein binding. The FAT domain is adjacent to the amino side of the FKBP12-rapamycin binding site that is flanked on its C-terminal side by the catalytic serine/threonine kinase domain. This kinase domain contains a conserved lipid kinase motif, making Tor1p and Tor2p members of the phosphatidylinositol-kinase-related kinase family. Finally, the carboxyl-terminal 33 residues of Tor1/2p comprise a FATCdomain that is postulated to contribute to redox-dependent Tor protein degradation.Under nutrient-rich conditions, TORC1 inhibits the function of transcriptional activators that are involved in nitrogen catabolite-repression, retrograde response, and stress-response, while activating those involved in ribosome biosynthesisusually by affecting the cellular translocation of these transcription factors. One common mechanism by which this occurs is through a TORC1-influenced change to the phosphorylation state of these factors leading them to bind a cytoplasmic anchor protein, thus preventing nuclear localization. These phosphorylation/dephosphorylation events are not directly mediated by the TORC1 complex but instead are carried out by an upstream regulator on which TORC1 acts, such as the phosphatase Sit4p or the RAS/cAMP signaling-related kinase Yak1p. Loss of Tor protein also results in a rapid and strong inhibition of translation initiation. Biochemical analysis indicates that TORC1 is involved in translation by stabilizing eIF4G, encoded by TIF4631 and TIF4632, an essential initiation factor required for mRNA translation via the 5' cap structure. TORC2-mediated regulation of signal transduction cascades required for actin organization, cell integrity, and endocytosis involves direct phosphorylation of the effector protein Ypk2p.Although S. cerevisiae has two TOR genes, all other eukaryotes appear to have only one. However, higher eukaryotes do have both TORC1 and TORC2 complexes and studies demonstrate that the complexes are both structurally and functionally conserved. In higher eukaryotes, TOR activity has also been shown to participate in apoptosis, hypoxia, and aging. The upstream regulators of TOR have been more extensively studied in Drosophila and mammalian systems and appear to be more differentially regulated than yeast Tor1/2p as they involve factors and TOR domains not conserved in S. cerevisiae. Because both the upstream and downstream signaling pathways of mammalian TOR are deregulated in tuberous sclerosis complex, Peutz-Jeghers syndromeand many malignant human cancers, TOR-targeting drugs are being clinically developed as anti-tumor therapies.