TORC1 Controls Degradation of Transcription Factor Stp1, a Key Effector of the SPS Amino Acid-Sensing Pathway in *Saccharomyces cerevisiae*

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The target of rapamycin (TOR) signaling pathway plays crucial roles in the regulation of eukaryotic cell growth. In *Saccharomyces cerevisiae*, nitrogen sources in the extracellular environment activate the TOR signaling pathway. However, the precise mechanisms underlying the regulation of TOR activity in response to extracellular nitrogen sources are poorly understood. Here, we report that degradation of Stp1, a transcription factor for amino acid uptake and a key effector of the SPS amino acid-sensing pathway, is controlled by TOR activity in *S. cerevisiae*. Using a genome-wide protein localization study, we found that Stp1 disappeared from the nucleus upon inactivation of TOR complex 1 (TORC1) by rapamycin, suggesting the involvement of Stp1 in the TOR signaling pathway. Supporting this notion, knockout mutant for the *STP1* gene was found to be hypersensitive to rapamycin, and overexpression of *STP1* conferred resistance to rapamycin. Interestingly, we found that the rapamycin-induced disappearance of Stp1 from the nucleus resulted from Stp1 degradation, which was dependent on the activity of a protein phosphatase 2A (PP2A)-like phosphatase Sit4, a well-known downstream effector of TORC1. Taken together, our findings highlight an intimate connection between the amino acid-sensing pathway and the rapamycin-sensitive TOR signaling pathway.