

## **TOR-MEDIATED NUTRIENT SIGNALING REGULATES AGING IN YEAST**

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Nutrient sensing and caloric intake regulate aging and longevity in nearly every organism studied. In yeast, two models of aging have been developed. Replicative life span is defined by the number of daughter cells produced prior to senescence, while chronological life span is defined by the length of time a cell can maintain viability in a non-dividing state. Both replicative and chronological aging are affected by nutrient levels. We report here the results of parallel genome-wide screens for single-gene deletions that increase either replicative or chronological life span. Among the genes identified, several are known components of the TOR signaling pathway, and deletion of Tor1 increases both replicative and chronological life span. Interestingly, the downstream effectors by which TOR regulates aging in dividing and non-dividing yeast cells appear to be distinct. We propose that the TOR pathway is a primary conduit through which excess nutrient availability promotes aging in eukaryotic cells.