Dietary restriction (DR) is a conserved metabolic intervention that extends the lifespan of multiple species, including yeast, flies, nematodes, rodents and, arguably, rhesus monkeys and humans. Hallmarks of lifelong DR are reduced body size, fecundity, fat accumulation and slower development. We have identified \textit{lmn-1}, the \textit{Caenorhabditis elegans} lamin gene, and \textit{atx-2}, the \textit{Caenorhabditis elegans} homolog of the human ATAXIN-2-like and ATAXIN-2 genes, as regulators of these multiple DR phenotypes. Downregulation of either lamin or atx-2 increases the body, cell size and fat content of DR animals and speeds up animal development, while overexpression of atx-2 is sufficient to reduce the body and brood size of wild-type animals. We genetically mapped \textit{atx-2} and \textit{lmn-1} to the mTOR pathway, downstream of AMPK and upstream of S6K and TORC1. It regulates TORC1 via its direct association with Rab GDP dissociation inhibitor beta (GDI-1), which probably regulates RHEB shuttling between GDP-bound and GTP-bound forms. Taken together, this work identifies a novel mechanism regulating multiple aspects of dietary restriction, as well as new mTOR pathway components. It also extends our understanding of diet-dependent growth retardation, and offers a potential mechanism to treat obesity.