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Protective effects of dietary restriction and glycolytic inhibition depend on PPAR

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We previously reported evidence that some protective effects of dietary restriction are dependent on the transcriptional co-activator CREB-binding protein (CBP) entailing a metabolic switch away from glucose and toward lipid metabolism. Furthermore, at least some forms of neurodegeneration, such as Huntington's disease, entail inhibition of CBP. Peroxisome proliferating factor receptor (PPAR) is a transcription factor that recruits CBP for transcription and functions to switch from glucose metabolism and toward lipid metabolism during nutritional deprivation. Here we report that protective effects of dietary restriction and glycolytic inhibition on lifespan and in a model of polyQ proteotoxicity in C. elegans are similarly dependent on PPAR. Conversely, as with lifespan, elevated glucose or CBP inhibition potentiates polyQ proteotoxicity, associated with increased production of NADH and, apparently, increased utilization of ETC complex I but decreased utilization of complex II. Finally, we report that the PPAR agonist, pioglitazone protects against polyQ-induced proteotoxicity and increases lifespan in C. elegans. These results establish a putative mechanism for the protective effects of dietary restriction and glycolytic inhibition.

Biography

Charles Mobbs completed his Ph.D. from University of Southern California and carried out post-doctoral studies at Rockefeller University, where he went on to join the faculty. He is now a professor of Neuroscience, Endocrinology, and Geriatrics at Mount Sinai School of Medicine. He has published more than 150 papers and edited 4 books on aging. Bridget Marcellino completed her Ph.D. in 2012 and is now in medical school at , as is Nydia Ekasumara Mount Sinai School of Medicine.