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Ageing Res Rev. 2010 Jul;9(3):324-53. doi: 10.1016/j.arr.2009.10.003. Epub 2009 Oct 21.

Caloric restriction: from soup to nuts.

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Author information

Abstract

Caloric restriction (CR), reduced protein, methionine, or tryptophan diets; and reduced insulin and/or IGFI intracellular signaling can extend mean and/or maximum lifespan and delay deleterious age-related physiological changes in animals. Mice and flies can shift readily between the control and CR physiological states, even at older ages. Many health benefits are induced by even brief periods of CR in flies, rodents, monkeys, and humans. In humans and nonhuman primates, CR produces most of the physiologic, hematologic, hormonal, and biochemical changes it produces in other animals. In primates, CR provides protection from type 2 diabetes, cardiovascular and cerebral vascular diseases, immunological decline, malignancy, hepatotoxicity, liver fibrosis and failure, sarcopenia, inflammation, and DNA damage. It also enhances muscle mitochondrial biogenesis, affords neuroprotection; and extends mean and maximum lifespan. CR rapidly induces antineoplastic effects in mice. Most claims of lifespan extension in rodents by drugs or nutrients are confounded by CR effects. Transcription factors and co-activators involved in the regulation of mitochondrial biogenesis and energy metabolism, including SirT1, PGC-1alpha, AMPK and TOR may be involved in the lifespan effects of CR. Paradoxically, low body weight in middle aged and elderly humans is associated with increased mortality. Thus, enhancement of human longevity may require pharmaceutical interventions.

PMID: 19853062 [PubMed - indexed for MEDLINE]

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