

Conserved and tissue-specific genic and physiologic responses to caloric restriction and altered IGFI signaling in mitotic* and postmitotic tissues.**

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Caloric restriction (CR), the consumption of fewer calories without malnutrition, and reduced insulin and/or IGFI receptor signaling delay many age-related physiological changes and extend the lifespan of many model organisms. Here, we present and review microarray and biochemical studies indicating that the potent anticancer effects of CR and disrupted insulin/IGFI receptor signaling evolved as a by-product of the role of many mitotic tissues as reservoirs of metabolic energy. We argue that the longe-vity effects of CR are derived from repeated cycles of apoptosis and autophagic cell death in mitotically competent tissues and protein turnover and cellular repair in postmitotic tissues. We review studies showing that CR initiated late in life can rapidly induce many of the benefits of lifelong CR, including its anticancer effects. We also discuss evidence from liver and heart indicating that many benefits of lifelong CR are recapitulated in mitotic and postmitotic tissues when CR is initiated late in life.

*Descriptive of tissues whose cells multiply by mitosis – cell division that results in two cells, each with the full number of chromosomes and each just like the original cell

** Descriptive of tissues whose cells are mature and are no longer capable of undergoing mitosis

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