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Extending Life-Span in C. elegans

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The life-span of the nematode *Caenorhabditis elegans* can be extended by at least six different mechanisms, including calorie restriction, reduced Ins/IGF-1 signaling, germline ablation, food sensing amphid ablation, mitochondrial deficiency, and decreased temperature. Reduced Ins/IGF-1 signaling and calorie restriction can also increase the life-span of flies and mice. The Brevia "Healthy animals with extreme longevity" by N. Arantes-Oliveira *et al.* (24 Oct. 2003, p. <u>611</u>) showed that *daf-2* RNAi treatment and gonad ablation of worms carrying the *daf-2(e1368)* hypomorphic mutation in the gene encoding the *C. elegans* Ins/IGF-1 receptor increases their life-span 6.0-fold. We have found that the average life-span of *daf-2(e1370)* mutants grown in axenic medium [a sterile liquid medium based on yeast extract, soy peptone, and hemoglobin; see (<u>1</u>)] was 90.9 days, representing a 6.3-fold life extension and a 7.5-fold adult life-span extension relative to wild-type controls grown on plate cultures seeded with live *E. coli* cells (<u>1</u>).

Arantes-Oliveira *et al.* also note the health of their long-lived worms. We observed that worms grown in axenic medium appear more vigorous than their monoxenically grown counterparts and that these worms exhibit an increase in metabolic rate (2), counter to the idea that a reduction of the metabolic rate is associated with a longer life-span. Moreover, both caloric restriction and reduced Ins/IGF-1 signaling increase the resistance to heat and oxidative stressors (1), and calorically restricted mice are less prone to age-related diseases. Thus, the life of worms can be extended without diminishing health. These results might be important for human aging as well, because both caloric restriction and cell signaling have been shown to regulate the aging rate in organisms ranging from yeast to mammals.

References and Notes

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- 2. K. Houthoofd et al., Exp. Gerontol. 37, 1371 (2002).
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