



[How a Low-Carbohydrate Diet Slows Aging](#)

Low-carbohydrate, high-fat diets (LCHF) have many health benefits. [They can cause weight loss in overweight people without hunger, and improve insulin resistance and dramatically improve diabetes.](#) Even more, [LCHF diets greatly improve lipid markers of cardiovascular disease risk.](#) Besides the beneficial effect on cardiovascular markers and weight loss, I'll show here how a low-carbohydrate diet slows aging.

Glucose shortens lifespan in *C. elegans*

Carbohydrates are long chains of sugar molecules; in the case of common foods like wheat, rice, and potatoes, the carbohydrates are long chains of glucose, the same type of sugar as in the blood. The influence of glucose as a food source has been studied in aging research.

In the worm *C. elegans*, [dietary glucose shortens lifespan.](#) One of the most important ways it seems to do this is through the production of advanced glycation end products, or AGEs. These molecules result from [the attachment of glucose to proteins](#) (hence glycation), and they are implicated in diabetic complications. AGE's may also be important in [the buildup of irremovable cellular junk \(lipofuscin\), resulting in the garbage crisis of aging.](#)

On the other hand, [glucose restriction increases lifespan in *C. elegans*.](#) Restricting glucose activates the equivalent of [AMPK](#), the cellular energy sensor, which in turn inhibits mTOR and increases stress defense mechanisms, notably Nrf2. Essentially, it acts as a form of [hormesis](#).

Important to note that the biochemical pathways involved in *C. elegans* lifespan extension are evolutionarily conserved mechanisms, so these results are of relevance to humans, though how much is a different question.

Glucosamine extends lifespan by reducing glucose metabolism

[Glucosamine](#) is an [over-the-counter supplement](#), and [it extends lifespan not only in *C. elegans*, but in mice too](#). (Important because that gets us closer to human physiology.) Mice who got glucosamine

show an induction of mitochondrial biogenesis, **lowered blood glucose levels**, enhanced expression of several murine amino-acid transporters, as well as increased amino-acid catabolism. Taken together, we provide evidence that GlcN [glucosamine] **extends life span in evolutionary distinct species by mimicking a low-carbohydrate diet**.

So, this is further evidence: reducing glucose metabolism increases lifespan. You could take glucosamine, or you could just cut out the middleman and reduce your carbohydrate consumption.

Calorie restriction extends lifespan and reduces glucose metabolism

[Calorie restriction](#) (CR), that is, the reduction in food given to lab animals or humans, is the most reliable and robust life-extension intervention there is, extending lifespan in rodents as much as 50%. The greater the restriction, the longer the life extension.

As one might expect, massive amounts of research has been done on CR attempting to pin down the means by which it counteracts aging. CR effects many biochemical/physiological changes, and some or all of these may be important to its benefits. One thing CR does is to decrease the metabolism of glucose, and to increase fat burning.

A key metabolic change during CR is a shift from carbohydrate metabolism to fat metabolism.

Once again, AMPK is involved, which coordinates a series of biochemical effects, including the shift to fat metabolism.

Carbohydrate restriction lowers insulin and IGF-1

CR lowers levels of both insulin and IGF-1 (insulin-like growth factor), and this is thought to play a large role in lifespan extension. Animals that have modified insulin signaling live longer, and IGF-1 is important in the

development of cancer.

Humans that eat a carbohydrate-restricted diet [see a large drop \(50%\) in plasma insulin, and about a 30% decrease in plasma IGF-1](#). This happened on a diet that contained 5% carbohydrate, as opposed to 60% before. Of interest, protein is thought to be important to IGF-1 levels, and this diet *increased* protein, to 35%, and IGF-1 still dropped, although muscle IGF-1 increased.

To what extent does carbohydrate restriction mimic calorie restriction? Probably a fair amount: [restricting carbohydrate alone is responsible for about 70% of the benefits of intermittent fasting](#).

Conclusion: Burning fat instead of glucose increases lifespan

The evidence above suggests that less metabolism of glucose and more of fat increases lifespan.

If you want to implement a low-carbohydrate, high fat diet, here's what you can eat ([source](#)):

'Green list': recommended foods on a Banting (low-carbohydrate high-fat) diet

Animal protein	Dairy	Fats	Nuts and seeds	Vegetables
Eggs Meats Poultry Game Seafood	Cottage cheese Cream Full-cream Greek Yogurt Cheeses	Olive oil Avocados Coconut oil Macadamia nut oil	Almonds Flaxseeds Macadamia nuts Pecans Pine nuts	All green leafy vegetables, cruciferous vegetables or above ground vegetables

Adapted, with permission, from Noakes *et al.* 19 Fruits are also recommended, but in controlled quantities based on carbohydrate content and the patient's level of IR.

PS: For more on fighting aging, read my book, [Stop the Clock](#).

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