

Longevity, cancer and diet connected: New research in worms could apply to humans

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Researchers have discovered a connection between genes that could hold the key to a longer, healthier life.

Using worms that share similar genetics to humans, scientists from Huntsman Cancer Institute (HCI) identified a previously unknown link between two genes- one associated with aging, the other with certain types of cancer. The research also indicates calorie intake can affect how these genes operate, possibly increasing lifespan in animals, an effect which has been previously observed but is not yet fully explained. The paper appears today in the journal *Current Biology*.

Scientists studied a gene called TOR, which regulates cell growth and plays a role in the development of cancer. "In *C. elegans*, the tiny roundworm that our lab studies, as well as some other animals, a loss of TOR has been shown to slow aging. Our work with *C. elegans* reveals that TOR depends on a second gene called *pha4/FoxA* to control the aging process," says study co-author Susan Mango, PhD, HCI investigator and professor in the University of Utah Department of Oncological Sciences.

The study also reveals calorie restriction plays a role in how these genes work. "When there's lots of food, TOR gets active, which decreases the action of *pha4/FoxA* down the line, and that in turn shortens the lifespan of *C. elegans*," says Mango. "When there's little food, there's little TOR and more *pha4/FoxA*, and that results in a longer lifespan." In short, a low calorie diet can affect the TOR and *pha4/FoxA* genes in worms,

slowing the progression of aging.

Many organisms have a TOR gene and a gene similar to pha4/FoxA, such as single-cell yeasts, roundworms, and mammals including humans. In mammals, FoxA controls cell metabolism and there is a lot of it in breast and prostate cancers. The findings of this research establish that animals use both genes to sense the amount of food that is available and control the length of lifespan.

Further research will be required to establish whether a similar relationship between these factors can control metabolism, longevity or disease in humans.

Source: University of Utah

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