

# Number of genetic markers linked to lifespan triples

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A new large-scale international study expands the number of genetic markers now known to be associated with exceptional longevity.

Researchers at the University of Connecticut, University of Exeter, University of Wisconsin and University of Iowa undertook a genome-wide search for variants influencing how long participants' parents lived. Their findings indicated genes that could one day be targeted to help prolong human life.

The team studied 389,166 volunteers who took part in the U.K. Biobank, with confirmation in the U.S. Health and Retirement Study and the Wisconsin Longitudinal Study. DNA samples carry the genetics of biological parents, which provide a practical way of studying exceptionally long lifespans.

Eight genetic variants had already been linked to lifespan, mainly variants that are involved in heart disease and dementia. The latest study, published in the journal *Aging*, has expanded this to 25 genes in all, with some specific to mothers' or fathers' lifespan separately.

"We have identified new pathways that contribute

to survival, as well as confirming others," said study author Dr. Luke Pilling of the University of Exeter.

"These targets offer potentially modifiable targets to reduce risk of an earlier death and improve health."

Researchers found a genetic risk score combining the top 10 variants was statistically associated with parents being centenarians.

How long we live is determined by a range of factors including our lifestyle and how well we treat factors including blood pressure and cholesterol from midlife. However, genetics, and how long our parental relatives lived, also play a role.

Genes involved in senescence, the "frozen" state that cells enter into after being damaged, played an important role in longevity. Drugs targeting senescence have already been shown to extend life in laboratory animals.

Genes related to inflammation and auto-immunity related genes were also prominent, opening up the possibility that precision anti-inflammatory treatments may one day be helpful in extending life.

"These findings add to a growing body of knowledge highlighting specific targets and biological pathways useful for the development of interventions designed to help maintain health, function, and independence in later life," said Dr. George Kuchel, professor of geriatrics at the University of Connecticut School of Medicine and director of the UConn Center on Aging.

The results confirm that many genetic variants combine to influence human lifespan: no single gene [variant](#) has been found to be responsible.

Professor David Melzer, of the University of Exeter Medical School, who led the group said: "This study helps open the way to novel treatment, but the strong role for genes affecting [heart disease](#) risk again underlines the importance of controlling [blood](#)

[pressure](#) and cholesterol levels throughout the [lifespan](#). Of course, adopting healthy lifestyles is important, and can probably overcome the negative effects of most of the [genes](#) found so far."

**More information:** Pilling LC, Kuo C, Sicinski K, Tamosauskaite J, Kuchel GA, Harries LW, Herd P, Wallace R, Ferrucci L, Melzer D. Human longevity: 25 genetic loci associated in 389,166 UK biobank participants. *Aging* (Albany NY). 2017 Dec 6. [Epub ahead of print] [www.aging-us.com/article/101334/text](http://www.aging-us.com/article/101334/text)

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