Why we outlive our ape ancestors

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In spite of their genetic similarity to humans, chimpanzees and great apes have maximum lifespans that rarely exceed 50 years. The difference, explains USC Davis School of Gerontology Professor Caleb Finch, is that as humans evolved genes that enabled them to better adjust to levels of infection and inflammation and to the high cholesterol levels of their meat rich diets.

In the December issue of *PNAS Early Edition*, Finch reveals that these evolutionary genetic advantages, caused by slight differences in DNA sequencing and improvements in diet, make humans uniquely susceptible to diseases of aging such as cancer, heart disease and dementia when compared to other primates.

Finch, the ARCO & William F. Kieschnick Professor in the Neurobiology of Aging and a distinguished University Professor, argues that a major contributor to longevity for humans is the genes that adapt to higher exposure to inflammation.

"Over time, ingestion of red meat, particularly raw meat infected with parasites in the era before cooking, stimulates chronic inflammation that leads to some of the common diseases of aging," Finch said.

In addition to differences in diets between species of primates, humans evolved unique variants in a cholesterol transporting gene, apolipoprotein E, which also regulates inflammation and many aspects of aging in the brain and arteries.

ApoE3 is unique to humans and may be what Finch calls "a meat-adaptive gene" that has increased the human lifespan.

However, the minor allele, apoE4, when expressed in humans, can impair neuronal development, as well as shorten human lifespan by about four years and increase the risk of heart disease and Alzheimer disease by several-fold. ApoE4 carriers have higher totals of blood cholesterol, more oxidized blood lipids and early onset of coronary heart disease and Alzheimer's disease.

"The chimpanzee apoE functions more like the "good" apoE3, which contributes to low levels of heart disease and Alzheimer's," Finch said. Correspondingly, chimpanzees in captivity have unusually low levels of heart disease and Alzheimer-like changes during aging.

Finch hypothesizes that the expression of ApoE4 could be the result of the antagonistic pleiotropy theory of aging, in which genes selected to fight diseases in early life have adverse affects in later life.

"ApoeE may be a prototype for other genes that enabled the huge changes in human lifespan, as well as brain size, despite our very unape-like meat-rich diets," Finch said. "Drugs being developed to alter activities of apoE4 may also enhance lifespan of apoE4 carriers."

Source: University of Southern California (news : web)