Slowing the ageing process—it's in your genes

September 5 2013

Dr Austen Ganley, senior lecturer in genetics and evolution in the Institute of Natural and Mathematical Sciences.

Imagine being able to take a drug that can reduce the rate at which you age. Research by Massey University senior lecturer in genetics Dr Austen Ganley is making this dream one step closer to reality.

A new study, published in the prestigious scientific journal *Current*

Biology, provides evidence that aging works through a special set of genes that everyone has - the rDNA genes.

Dr Ganley was part of an international team, led by Dr Takehiko Kobayashi from the National Institute of Genetics in Mishima, Japan. The team found that by improving the stability of the rDNA genes, which are usually quite unstable, they could extend the lifespan of baker's yeast, a model system for studying cell aging.

"This work is exciting because it shows that rDNA instability is a new factor in aging," says Dr Ganley.

The researchers set out to understand how the Sir2 gene reduces aging in yeast. Sir2 genes shot to prominence as potential human anti-aging genes with the finding that resveratrol, a component of red wine, activates them. However, subsequent research has found that resveratrol doesn't extend lifespan in mammals.

The yeast Sir2 gene controls rDNA stability, but also has many other targets in the cell. The breakthrough came when the scientists found a way to separate Sir2's effect on the rDNA from its other effects. This allowed them to show that Sir2's anti-aging effect comes exclusively through stabilisation of the rDNA genes.

"This is significant," says Dr Ganley, "because in humans there are seven Sirtuins (the equivalent of the Sir2 gene), and they all behave very differently to the yeast Sir2 gene. In contrast, the rDNA genes are very similar between yeast and humans, therefore rDNA gene instability may be the common factor in aging across life."

Professor Kobayashi originally proposed a role for rDNA instability in aging five years ago, but unequivocal support for this theory has been lacking until now. These new results suggest that finding a way to

artificially improve rDNA gene stability may delay the aging process in humans too.

However, Dr Ganley cautions that the role of the rDNA genes in human aging still needs to be clarified.

"Although we know human rDNA genes are unstable, we don't know if this instability affects lifespan. Finding this out is the next critical step, and the challenge lies in doing these experiments with human cells, which are more difficult to work with than yeast."

Dr Ganley and Professor Kobayashi will publish an update of this rDNA aging theory in a special "Cell aging and death" issue of the scientific journal *FEMS Yeast Research* early next year.

This study is a part of Dr Ganley's research program being carried out at the Albany campus into the effects of the rDNA genes, and is supported by the Marsden Fund. He is also interested in the involvement of the rDNA genes in other biological processes, such as cancer and chromosome inheritance.

More information: www.sciencedirect.com/science/ … ii/S0960982213008713

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