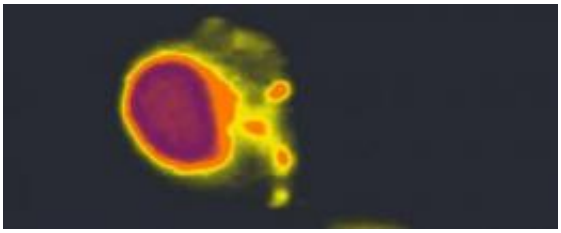
The body's power stations can affect aging

May 10 2011



This is a photo of the genetic material in the large cell nucleus and in the small mitochondria. Credit: University of Gothenburg

Mitochondria are the body's energy producers, the power stations inside our cells. Researchers at the University of Gothenburg, Sweden, have now identified a group of mitochondrial proteins, the absence of which allows other protein groups to stabilise the genome. This could delay the onset of age-related diseases and increase lifespan.

Some theories of human ageing suggest that the power generators of the cell, the mitochondria, play a part in the process. In addition to supplying us with energy in a usable form, mitochondria also produce harmful by-products – reactive oxyradicals that attack and damage various cell components. Eventually these injuries become too much for the cell to cope with, and it loses its capacity to maintain important functions, so the organism starts to age. That's the theory anyway. Oddly enough, several studies have shown that certain mitochondrial dysfunctions can actually delay ageing, at least in fungi, worms and flies. The underlying mechanisms have yet to be determined.

In a study from the Department of Cell and Molecular Biology at the University of Gothenburg, published in the journal *Molecular Cell*, a research team has now identified a group of mitochondrial proteins that are involved in this type of ageing regulation. The researchers found that a group of proteins called MTC proteins, which are normally needed for mitochondrial protein synthesis, also have other functions that influence genome stability and the cell's capacity to remove damaged and harmful proteins.

"When a certain MTC protein is lacking in the cell, e.g. because of a mutation in the corresponding gene, the other MTC proteins appear to adopt a new function. They then gain increased significance for the stabilisation of the genome and for combating protein damage, which leads to increased lifespan," says Thomas Nyström of the Department of Cell and Molecular Biology.

He adds, "These studies also show that this MTC-dependent regulation of the rate of ageing uses the same signalling pathways that are activated in calorie restriction – something that extends the lifespan of many different organisms, including yeasts, mice and primates. Some of the MTC proteins identified in this study can also be found in the human cell, raising the obvious question of whether they play a similar role in the regulation of our own ageing processes. It is possible that modulating the activity of the MTC proteins could enable us to improve the capacity of the cell to delay the onset of age-related diseases. These include diseases related to instability of the genome, such as cancer, as well as those related to harmful proteins, such as Alzheimer's disease and Parkinson's disease. At the moment this is only speculation, and the precise mechanism underlying the role of the MTC proteins in the ageing process is a fascinating question that remains to be answered."

Provided by University of Gothenburg