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Slowing Down the Clock: Cellular Senescence and Aging

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Title: Slowing Down the Clock: Cellular Senescence and Aging

Author: [Saad Bhatti](#)

Recently, anti-aging fads have been designed to slow down the aging process and in some cases, reverse it. Yet, these so-called “breakthroughs” have little to no evidence supporting their effectiveness. Researchers have turned towards an alternative way to combat the aging process by targeting molecular pathways. Morrigan McKernan, a Sophomore Biology major in the lab of Dr. Alaattin Kaya, is examining these pathways.

Dr. Kaya and McKernan seek to understand the effect certain genes in eukaryotic yeast have on its lifespan, both in terms of the number of replication events and length of the lifespan. Using polymerase chain reaction to knock down particular genes and determine their effect, McKernan and their team discovered that deleting certain enzymes in the electron transport chain, including adenosine triphosphate (ATP) synthase and complexes III and IV, decreased the chronological lifespan of the yeast. The deletion of ATP synthase and certain mitochondrial ribosomal proteins decreased the replicative lifespan of yeast. Such findings could be applied in clinical settings by upregulating these proteins in patients who show decreased levels, or mutations, in these proteins.

The larger implication of McKernan’s project is that the process of aging ultimately lies at the cellular level. Recently, researchers have looked towards cellular senescence, a period in which cells cease to divide but are still metabolically active, as a potential target for delaying aging. Cellular senescence has previously been a target to prevent tumor initiation, and so its role in aging has just been elucidated.

For example, researchers discovered senescence can hasten the aging process, as well as exacerbate age-related diseases such as Alzheimer’s disease and osteoarthritis. Senescent cells accumulate in tissues as one becomes older and may release proteases leading to tissue dysfunction and inflammation. By clearing tissues of senescent cells, one may be able to slow down the aging process and mitigate age-related diseases.

Aging is an inevitable, universal process. For those who are stricken with an age-related disease, prolonging age is a possible treatment. As new knowledge is discovered about the molecular pathways involving drugs, new treatment options are being developed with the intent to prolong life. The goal is to make anti-aging therapies not commercial gimmicks, but treatments backed up by scientific evidence.

To learn more about Morrigan McKernan’s research in Dr. Alaattin Kaya’s lab, email mckernanme@vcu.edu.

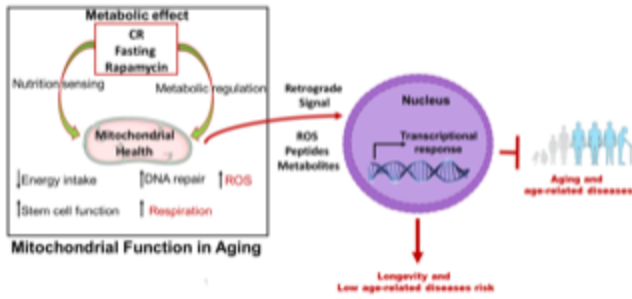


Figure: All these alterations in mitochondrial physiology have been associated with many hallmarks of aging and age-related diseases. Improving mitochondrial function with pharmaceutical drugs or genetic manipulations have been shown to increase lifespan in many organisms.