

Once blamed for aging, ROS molecules may actually extend life

June 8 2011



(Medical Xpress) -- In a new study, Yale University researchers have identified a pathway by which reactive oxygen species (ROS) molecules, which are usually implicated in the aging process due to their damage to DNA, can also act as cellular signaling molecules that extend lifespan. The study, which provides insights into the underlying mechanisms of the ROS signaling process, is published in the June issue of *Cell Metabolism*.

Increased ROS, and their effects at the <u>cellular level</u>, can lead to oxidative stress, which is involved in many diseases and aging. But ROS are also necessary for the proper functioning of the immune system and other biological functions. Using the <u>model organism</u> yeast, the Yale team set out to determine whether regulating ROS and their ability to act as signaling molecules could impact the <u>aging process</u>.



Inhibiting a signaling pathway called <u>Target</u> of Rapamycin (TOR), which is involved in sensing nutrients and cell growth, increases lifespan in yeast, as it does in mice. The Yale team found that a key way this occurs is by altering the function of cellular powerhouses called mitochondria so that they produce more signaling ROS.

"The concept that ROS are important cellular signaling molecules, and not just agents of damage and stress, has grown to be widely accepted," said lead author Gerald S. Shadel, Ph.D., professor of pathology and genetics at Yale School of Medicine. "Remarkably, in this study, we show that their purposeful production by mitochondria can even provide an adaptive signal that can delay aging."

Since the TOR pathway operates largely the same in yeast as it does in humans, the new connections to mitochondrial ROS signaling and aging in this study may be more widely applicable. Shadel said that new ways to intervene in age-related pathology may stem from these basic studies. "Trials targeting the TOR pathway as an anti-cancer strategy in humans are already underway. Our study suggests that carefully augmenting mitochondria and ROS production in humans may also be beneficial in combating aging and associated diseases."

Other authors are Yong Pan and Elizabeth A. Schroeder of Yale School of Medicine; Alejandro Ocampo of the Yale Department of Biochemistry and Molecular Biology; and Antonio Barrientos of Yale and the University of Miami Miller School of Medicine.

Provided by Yale University

Citation: Once blamed for aging, ROS molecules may actually extend life (2011, June 8) retrieved 19 April 2024 from

https://medicalxpress.com/news/2011-06-blamed-aging-ros-molecules-life.html



This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.