Aging may trigger adaptive response to offset effects of oxidative stress on blood vessels

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Although the causes of many age-related diseases remain unknown, oxidative stress is thought to be the main culprit. Oxidative stress has been linked to cardiovascular and neurodegenerative diseases including diabetes, hypertension and age-related cancers. However, researchers at the University of Missouri recently found that aging actually offered significant protection against oxidative stress. These findings suggest that aging may trigger an adaptive response to counteract the effects of oxidative stress on blood vessels.

"Molecules known as reactive oxygen species, or ROS, play an important role in regulating cellular function," said Steven Segal, a professor of medical pharmacology and physiology at the MU School of Medicine and senior author of the study. "However, the overproduction of ROS can help create a condition referred to as oxidative stress, which can alter the function of cells and interfere with their growth and reproduction."

To understand the effects of aging on the function of blood vessels when they are exposed to oxidative stress, Segal's team studied the inner lining, or endothelium, of small resistance arteries. Resistance arteries are important to cardiovascular function because they regulate both the amount of blood flow into tissues and systemic blood pressure.

"We studied the endothelium from resistance arteries of male mice at 4 months and 24 months of age, which correspond to humans in their early 20s and mid-60s," Segal said. "We first studied the endothelium under resting conditions and in the absence of oxidative stress. We then simulated oxidative stress by adding hydrogen peroxide. When oxidative stress was induced for 20 minutes, the endothelial cells of the younger mice had abnormal increases in calcium when compared to the endothelial cells of the older mice. This finding is important because when calcium gets too high, cells can be severely damaged."

When oxidative stress was extended to 60 minutes, Segal's team found that the death of endothelial cells in the younger mice was seven times greater than those from the older mice. These findings indicated that with advancing age, the endothelium had adapted to preserve cellular integrity when confronted with oxidative stress.

"The most surprising thing we found is that the endothelium was much less perturbed by oxidative stress during advanced age when compared to younger age," Segal said. "This finding contrasts with the generally held belief that the functional integrity of the endothelium is compromised as we age. Our study suggests that blood vessels adapt during the aging process to regulate ROS and minimize cell death when subjected to an abrupt increase in oxidative stress. This adaptation helps to ensure that the arteries of older individuals can still do their jobs."

"Although more studies are needed to identify the mechanism by which the endothelium adapts to advanced age, our study provides evidence that the natural tendency of the body is to adapt to oxidative stress during healthy aging," Segal said.

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University of Missouri-Columbia
