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Preventing cardiovascular disease in old aortas

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Cardiovascular disease (CVD) is the No. 1 killer of people in the U.S. An early sign of CVD is increased aortic stiffness, a change that becomes more prevalent as we age. Now, researchers at Sargent College of Boston University have made some unexpected discoveries into the causes of and anatomical structures involved in arterial stiffening. Their findings could contribute to the development of medical therapies aimed at reducing or preventing this CVD risk factor.

The aorta is the main artery of the body. It is connected to the heart and carries oxygen-rich blood pumped from the left ventricle to the rest of the circulatory system. Pumping blood from the heart causes pulsing waves that reverberate into the aorta. As it branches off into smaller blood vessels, the aorta acts as a shock absorber, blunting the impact of these waves. But with age, changes in the blood vessel wall can cause the aorta to lose some of its flexibility and its ability to buffer high-pressure waves as they travel to the smaller vessels. The reduced shock-absorbing capacity can lead to changes in microcirculations and negative effects on organ function.

The underlying cause of aortic stiffening is unclear. While much of the previous research pointed to the extracellular matrix (ECM)—a group of molecules secreted by the cells that support cell attachment and communication—as the culprit, a few studies suggest that vascular smooth muscle may play a role.

In this study, Yuan Z. Gao et al. directly measured the mechanical

properties of the aortas of young and old mice to observe how smooth muscle cells factor into aortic wall stiffness. They also observed how focal adhesion signaling—which helps promote arterial flexibility in young mice—is impaired with aging.

They used a novel biomechanical method to distend the aorta, mimicking circumferential strain, to measure how the smooth muscle affected arterial stiffness. "A major finding of the present study is that the smooth muscle cell is a major source and regulator of vascular stiffness, in contrast with the often-assumed dominance of ECM in effecting changes in wall stiffness with aging," the researchers wrote. They also found that the decrease in the focal adhesion signaling mechanism led to higher stiffness in old vessels.

"We conclude from our results that the smooth muscle focal adhesions represent a potential therapeutic target in the context of preventing or reversing increases in aortic stiffness," they wrote. "An understanding of this mechanism may lead to an approach to reverse this aging-induced deficiency."

The article "Aging impairs smooth muscle-mediated regulation of aortic stiffness: a defect in shock absorption function?" is published in the *American Journal of Physiology* – Heart and Circulatory Physiology. It is highlighted as one of this month's "best of the best" as part of the American Physiological Society's APSselect program.

More information: Aging impairs smooth muscle-mediated regulation of aortic stiffness: a defect in shock absorption function? *American Journal of Physiology* Published 15 October 2014Vol. 307no. 8, H1252-H1261DOI: 10.1152/ajpheart.00392.2014