New cause discovered for arterial stiffness, a contributor to cardiovascular disease

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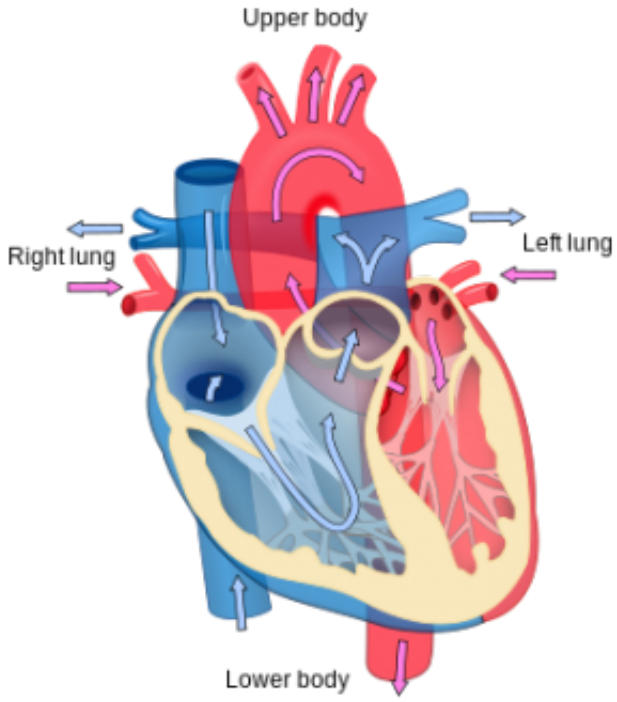

Heart diagram. Credit: Wikipedia

Increased vascular stiffness has been identified as an important part of hypertension in aging adults. Previous studies of aortic stiffness have focused on changes in structural proteins that alter the properties of vascular walls causing them to become rigid. Now, a research team led by scientists at the University of Missouri have determined that smooth muscle cells, which line the interior of vascular walls, are a major

contributing factor to vascular stiffness, one of the major causes of hypertension. Researchers believe that results from their study could help provide new possibilities for drug treatments for the disease in aging patients.

"Arterial and vascular stiffness occurs through the normal process of biological aging and is associated with an increased risk of heart attacks and strokes," said Gerald Meininger, director of the Dalton Cardiovascular Research Center and a professor of medical pharmacology and physiology in the School of Medicine at MU. "As we age, the aorta, which normally acts as a shock absorber dampening the pulse associated with each heartbeat, tightens and becomes rigid, causing a host of problems including high blood pressure, increased risk of adverse cardiovascular events and even death."

In the United States, the risk of developing hypertension due to aging is greater than 90 percent in both men and women. Recent studies have identified several mechanisms for arterial stiffness in humans. Research has focused on the structural matrix proteins, or non-living components that compose the outer walls of blood vessels, as well as endothelial cells which line the inner portion of the vascular walls. Meininger and his team focused on a new potential source—smooth muscle cells that are a major component of the "middle" of the blood vessel wall.

Teaming with researchers at Rutgers University and the New Jersey Institute for Technology, Meininger and his group isolated aortic cells from normal and hypertensive rat models in both young and aged animals. Then, using atomic force microscopy, an advanced microscope that incorporates a tiny probe that can interact with single cells and molecules, the team measured the compression force of the needle against the specimen and how the tip adhered to or "stuck" to smooth muscle cells.

"We found that hypertension increased both vascular smooth cell stiffness and adhesion or stickiness, and that these changes were augmented by aging," Meininger said. "Our results are adding to our understanding and taking studies in a different direction. Although all cells are contributing to arterial stiffness, it's important to identify the order in which they're adding to the problem. Identifying smooth muscle cells as a contributor can help identify possible preventatives and potential drugs to counteract and reverse the disease and keep vessels healthier as we age."

The early-stage results of this research are promising. If additional studies are successful within the next few years, MU officials will request authority from the federal government to begin human drug development (this is commonly referred to as the "investigative new drug" status). After this status has been granted, researchers may conduct human clinical trials with the hope of developing new treatments for arterial stiffness and resulting hypertension.

More information: "Increased Vascular Smooth Muscle Stiffness: A Novel Mechanism for Aortic Stiffness in Hypertension" *Hypertension*, 2015.

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