# Age-Associated Changes in the Vascular Renin-Angiotensin-System in Mice

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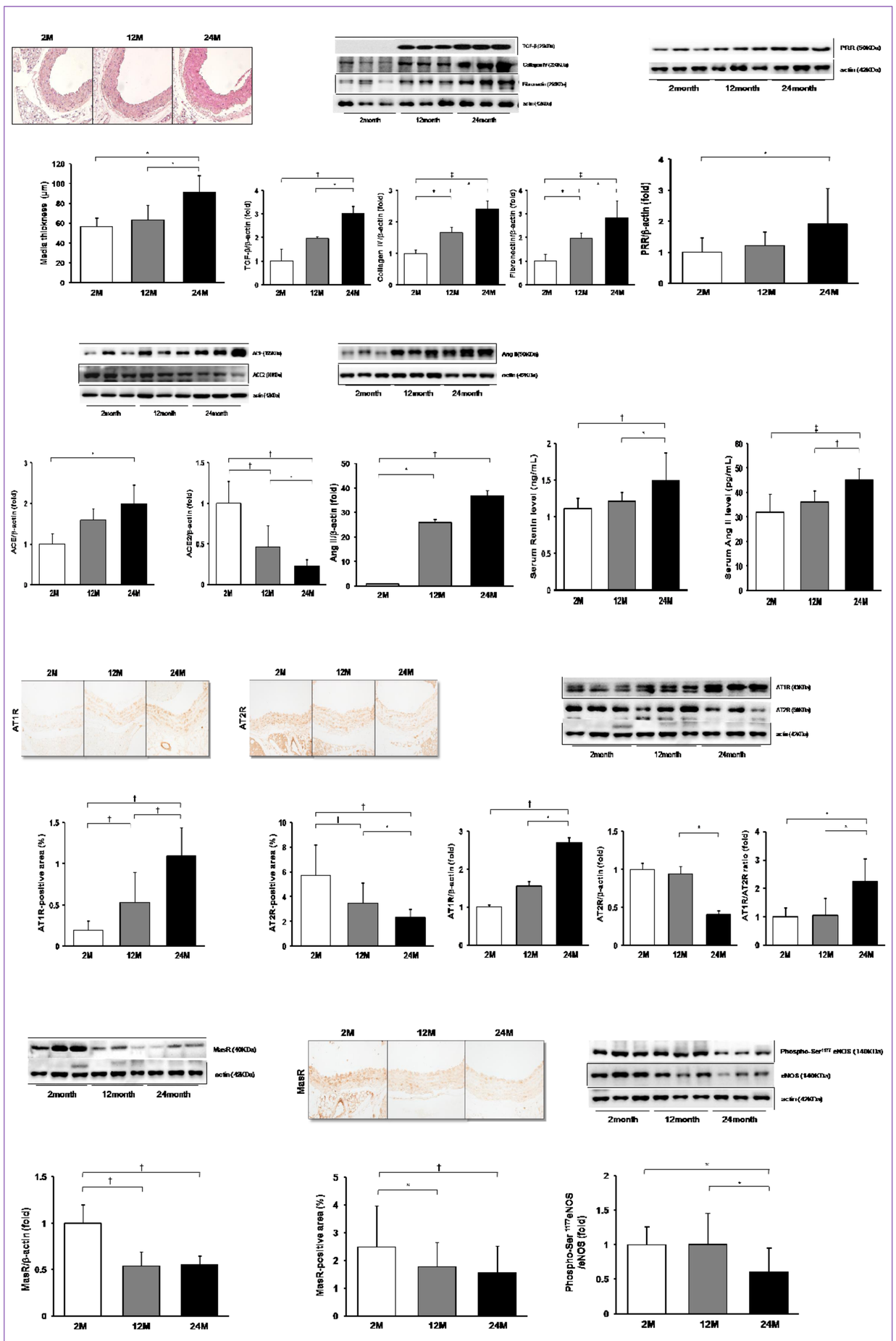
## OBJECTIVES

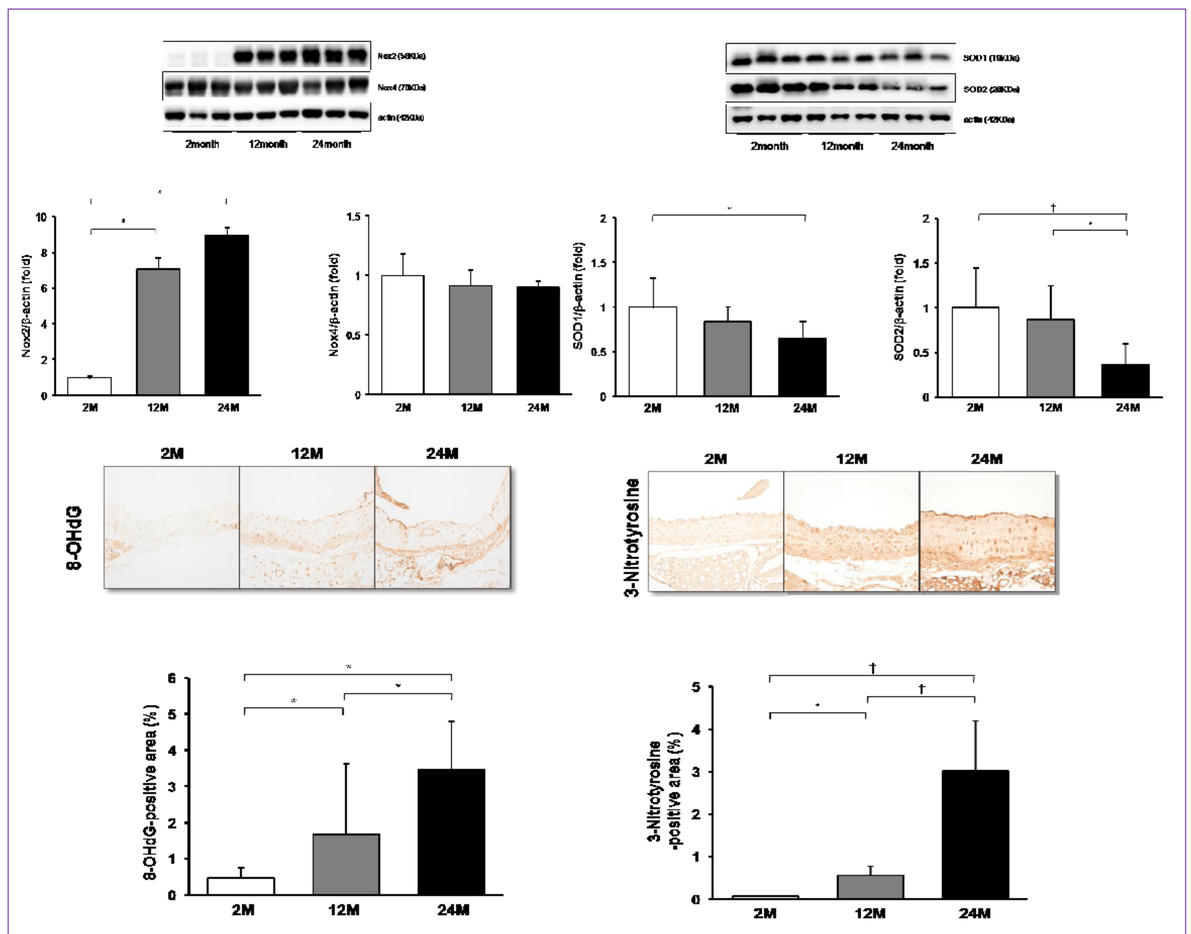
Aging is the major risk factor of cardiovascular disease and the renin-angiotensin system (RAS) is the key player in cardiovascular diseases. This study evaluated whether the change in the RAS is associated with arterial aging in mice.

## METHODS

Histologic changes and expressions of transforming growth factor-β (TGF-β), collagen IV, angiotensin II (Ang II), angiotensin converting enzyme (ACE), angiotensin converting enzyme 2 (ACE2), angiotensin II type 1 receptor (AT1R), angiotensin II type 2 receptor (AT2R), prorenin receptor (PRR), Mas receptor (MasR), endothelial nitric oxide synthase (eNOS), NADPH oxidase 2 and 4 (Nox2 and Nox4), 8-hydroxy-2′-deoxyguanosine (8-OHdG), and superoxide dismutase 1 and 2 (SOD1 and SOD2), were measured in the thoracic aortas from 2-month-old, 12-month-old, and 24-month-old C57/BL6 mice.




## RESULTS




Twenty-four-month-old mice showed significantly increased aortic media thickness and expressions of TGF-β, collagen IV, and fibronectin, compared to 2-month-old and 12-month-old mice. The expressions of PRR, ACE, and Ang II, and AT1R-positive area significantly increased, whereas expressions of ACE2 and MasR and AT2R-positive area decreased with age. The expressions of phosphorylated serine[1177]-eNOS, SOD1 and SOD2 decreased, and the 8-OHdG-positive area and the 3-nitrotyrosine-positive area increased with age. The expression of Nox2 significantly increased with age, but that of Nox4 did not change. ***Conclusions.*** The enhanced PRR-ACE-Ang II-AT1R axis and reduced ACE2-MasR axis were associated arterial aging in mice.

## CONCLUSIONS

The enhanced PRR-ACE-Ang II-AT1R axis and reduced ACE2-MasR axis were associated arterial aging in mice.

## REFERENCES:

•E. G. Lakatta and D. Levy, "Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease," Circulation, vol. 107, no. 1, pp. 139-146, 2003.

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