Changes in the biomechanics and stiffness in resistance arteries

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Introduction: Arterial stiffness and alteration in vascular biomechanics play pivotal roles in circulatory dysfunction, leading to myriad of circulatory disorders. Our objectives were to compare structural and functional alterations related to vascular stiffness in small arteries.

Methods: Hemodynamic assessments were made in Dahl salt-sensitive rats (male (M) versus female (F)) (n=5-8/group, mean ± s.e.m), and third-order mesenteric arteries were isolated for functional, biochemical and ultrastructure evaluations.

Results: We found that rats on a high salt (H) compared to regular (R) diets developed hypertension with elevated pulse wave velocity. Morphometric assessment of the vessel wall indicated an increase in collagen (H: 12.2 ± 1.5 μm^2; R: 6.8 ± 1.3 μm^2) and smooth muscle cells (H: 36.8 ± 7.5 μm^2; R: 19.8 ± 1.5 μm^2) areas of the ultrastructure of the hypertensive males compared to females and the normotensive group. Vasoconstriction resulted in significantly higher Composite Young Modulus (CYM) in H males (8.6 ± 1 KPa) than R (4.5 ± 0.8 KPa), and the corresponding females (H: 5.6 ± 0.6 KPa and R: 5 ± 0.9 KPa). In contrast, vasodilation significantly reduced CYM in the male groups (H: 2.5 ± 0.4 KPa and R: 2.7 ± 0.5 KPa) compared to the corresponding values in females (H: 4.2 ± 0.6 KPa and R: 5 ± 0.5 KPa). Inhibition of endothelial cell (EC) function significantly increased CYM in the normotensive (M: 7.4 ± 0.7 KPa, F: 8.6 ± 1 KPa) and female hypertensive (F: 7.1 ± 0.3 KPa) groups but not in the male hypertensive group (M: 4.7 ± 1 KPa).

Conclusion: Our findings support a link between high salt intake, elevated blood pressure, and changes in the ultrastructure of the vessels to be sex-specific. This may be important in disturbing normal wave reflection and crosstalk between micro- and macro-circulations.