Quality Control Assessment of Vascular Reactivity in Aorta and Uterine Arteries Using Wire Myography

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During pregnancy, vascular adaptations are important to meet the oxygen and nutrient demands of a growing fetus. Maternal blood flow and cardiac output increase while systemic vascular resistance decreases. Nitric oxide (NO), an endothelium-derived vasodilator, promotes smooth muscle relaxation, reducing vascular resistance. However, recent studies suggest that particle exposure impairs NO signaling and disrupts vascular relaxation during pregnancy. The purpose of this study was to establish quality control methodology by ensuring vascular responses from 2026 Sprague-Dawley rats are consistent with established 2023 laboratory data. Uterine arteries and aortas were collected from virgin female Sprague-Dawley rats and placed in chilled physiological salt solution (PSS). Vessel segments (2 mm) were mounted on a wire myograph (Danish Myo Technology, Inc) and placed in warmed PSS to equilibrate for 20 minutes. Chemical agonists, methacholine (MCh), an endothelium-dependent agonist, and sodium nitroprusside (SNP), an endothelium-independent agonist, were added to the vessel bath in increased concentrations (10^{-9}–10^{-4} M) following high-potassium physiological salt solution (KPSS)-induced contraction. Force measurements (mN; LabChart) were recorded and compared to historical data. Aortic MCh responses demonstrated similar maximal responses (99.69% vs. 95.72%) and slightly greater relaxation at the highest concentration (18.19% vs. 21.12%). In contrast, uterine artery MCh responses showed similar peaks (96.43% vs. 98.16%) but did not replicate the relaxation-curve slope or reach equivalent relaxation (60.04% vs. 22.54%). SNP responses were attenuated in both, with lower peak responses. This indicates that greater consistency with 2023 data is needed before evaluating particle exposure on vascular function and therapeutic interventions. Supported by: NIH R01-ES031285, R25-ES020721, T32-ES007148, and P30-ES005022