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Unaltered Long-Term Maternal Endothelin System in a Gestational Hypertensive Rat Model
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Abstract
INTRODUCTION: Cardiovascular diseases (CVDs) are two to four times more likely to affect women with a history of hypertensive disorders of pregnancy (HDPs). One of the etiologies of CVDs is endothelial dysfunction, which results from an imbalance in the production of endothelin-1 (ET-1) and nitric oxide (NO). Although blood pressure (BP) is normalized postpartum, we hypothesize that a transient increase in BP during HDPs may cause ongoing endothelial dysfunction. We aimed to discover the impact of HDPs in the development of cardiovascular diseases after long-term postpartum changes in endothelin-A receptor (ETAR) and endothelin-B receptor (ETBR) expression and the concentration of ET-1 and NO. MATERIALS AND METHOD: Twenty-four female Sprague-Dawley (SD) rats were assigned into four groups (n=6), including two treatment and two control groups. All rats were sacrificed on Day 30 postpartum. The NO and ET-1 concentrations were determined by ELISA and the ETAR and ETBR expression of the mesenteric arteries were measured by immunohistochemistry studies. RESULTS: The mean concentrations of ET-1 and NO were not significantly different in all groups. There was no significant difference in the mean immunoreactivity of ETAR and ETBR percentages area in the tunica intima and media in all groups. CONCLUSION: No evidence demonstrates significant changes in the endothelin system of the resistance arteries at the proteomic level in the long-term duration following HDP. Further investigation of its potential chronic effect warrants a deeper analysis at the molecular and ultrastructural levels.

Author Keywords
endothelial dysfunction; endothelin; Hypertension; postpartum; pregnant

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