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Letter to Editor **Open Access**

The Functional Role Of B-Type Natriuretic Peptide in Preeclampsia

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Preeclampsia, complicating 2–8% of pregnancies, is a leading cause of maternal and perinatal mortality and morbidity. This pregnancy-specific disease characterized by development of concurrent hypertension and proteinuria, sometimes progressing into a multiorgan cluster of varying clinical features. Pre-eclampsia is characterized physiologically by plasma volume contraction and intense vasoconstriction. It was originally thought that the renin-angiotensin-aldosterone (RAA) system, -what plays an important role in regulating blood pressure and electrolyte balance- would be overactive but studies have shown a more complex picture. Several study found higher concentration of BNP in preeclamptic patients compared to healthy controls. BNP increase glomerular infiltration and natriuresis, supressing sodium reabsorption and relax the vessel's smooth musculature causing a decrease in the cardiac preload and afterload and a decrease in blood arterial pressure. BNP also reduce renin-angiotensinaldosterone activities and inhibit endothelin-1 production. In our measurement BNP showed significantly higher levels in preeclamptic patients.

The (TTTC) polymorphism in the 5'-flanking region of the NPPB gene repeats showed association with the BNP concentrations. BNP levels were higher in early-onset than in late-onset preeclamptic patients. The distinction criterion for early versus late onset was set as week 34 of gestation. The amount of proteinuria and total protein levels correlate with the elevation of the BNP levels. In early-onset preeclampsia the extent of proteinuria is higher than in the late-onset preeclampsia and a significant positive correlation was observed between plasma levels of BNP and hematocrit. A BNP cut-off <24.5 pg/ml had a negative predictive value of 85.1% in excluding preeclampsia.

In pregnancy, uterine spiral artery remodeling is an adaptive morphological change at the maternal and fetal interface, which is critical for dilating the artery and promoting blood flow to the fetus. Incompletely remodeled spiral arteries have been recognized as a common pathological feature in preeclamptic patients. To date, the molecular mechanism that controls spiral artery remodeling is not well defined. Corin is a transmembrane serine protease discovered in the heart, where it converts pro-atrial natriuretic peptide (pro-ANP) to active ANP, a cardiac hormone that regulates salt-water balance and blood pressure. Recent studies show that corin is up-regulated in the decidua of the pregnant uterus, suggesting a potential role of corin in pregnancy. In mice lacking corin or ANP, high blood pressure and proteinuria were found at late gestational stages. Histological analysis indicated delayed trophoblast invasion and impaired spiral artery remodeling in the uterus. In humans, *CORIN* gene mutations were identified in patients with preeclampsia. In this review, we discuss the function of corin and ANP in regulating blood pressure and their potential role in preeclampsia.

Preeclampsia is a serious disease, causing premature births and contributing to maternal and neonatal mortality. To date, the underlying disease mechanism remains poorly understood. Spiral artery remodeling is a major adaptive change in the pregnant uterus, which is critical for decreasing maternal blood flow resistance and increasing unteroplacental perfusion. Failures in remodeling uterine spiral arteries have long been recognized as an important factor in the pathogenesis of preeclampsia.

In pregnancy, many molecules such as hormones, growth factors, vasodilators, adhesion proteins and proteases act collectively to regulate spiral artery remodeling. Defects in signaling pathways mediated by these proteins are likely to prevent a healthy uteroplacental interface, thereby contributing to preeclampsia that is known for its multifactorial nature. Indeed, vascular endothelial growth factor, placenta growth factor, soluble fms-like tyrosine kinase-1 (sFlt1), angiotensin, estradiol, and endoglin have been found to play a role in the disease.

Corin is a protease discovered in the heart. Its biological function is to activate atrial natriuretic peptide (ANP), a cardiac hormone that regulates blood pressure and salt-water balance. Interestingly, corin expression was detected in the decidua of the pregnant uterus, suggesting a potential role of corin in pregnancy. Most recently, corin has been shown to have a local function in the uterus to promote trophoblast invasion and spiral artery remodeling. In this review, we briefly describe the biology of corin and discuss a possible role of corin and ANP in preeclampsia.