

Hypertension is a multi-factorial disorder thought to result from both genetic and environmental factors. However, numerous epidemiological studies report an inverse relationship between birth weight and blood pressure suggesting that an increased risk of cardiovascular disease and hypertension may also be consequence of poor fetal growth and low birth weight.

Within the United States, a higher percentage of low birth weight babies are born relative to other Western countries with the highest rates of low birth weight localized in the Southern states; higher rates of hypertension and cardiovascular disease are also concentrated in the South.

Our laboratory uses a unique model of placental insufficiency to investigate the mechanisms linking low birth weight and hypertension. A reduction in nephron number, hyperfiltration and increased susceptibility to renal injury, activation of the sympathetic and renin angiotensin systems are potential mediators of post-natal hypertension programmed in response to developmental insult. However, the quantitative importance and integration of these mechanistic pathways in the developmental programming of hypertension has not been clearly elucidated.

Moreover, sex differences are observed in our model of programmed hypertension, and recent studies from our laboratory suggest that their effects may not be direct, but may influence blood pressure via modulation of regulatory systems critical to the long-term control of blood pressure. Understanding the complexity of the developmental programming of adult disease may lead to preventive measures and early detection of cardiovascular risk and help define the role of sex hormones in mediating sexual dimorphism in response to a suboptimal fetal environment.