on growth character and impact on the heart of the offspring subject is still subject to discussion. N-cadherin plays a critical role in myocardial development and function and is associated with the development of heart failure. Studies have shown a nicotine receptor mediated pathway was associated with the modulation of N-cadherin expression. In summary, we hypothesise that prenatal treatment of nicotine might alter myocardial N-cadherin expression which may predispose for impaired cardiac function in later life.

Methods Sprague–Dawley rats were infused with nicotine (12 mg/kg/day) or saline via subcutaneous osmotic minipumps throughout the gestation. We weighed brain, heart, lung, liver, and kidney of one randomly selected pup of each litter within 3–12 h after birth. The litter size was then reduced to eight pups to ensure equal nutrient access. Body weight was measured every 2 days after birth. Heart samples from the offspring at birth and 45 days of age were serially sliced for measuring N-cadherin by Immuno-histochemistry staining and analysed by western blot for N-cadherin expression.

Results The birth weights of prenatal nicotine exposure offspring were significantly reduced compared with control offspring (6.72 ± 0.38 g vs 7.40 ± 0.35 ; $p 0.05$). Brain weight-to-body weight was significantly higher, and the liver weight-to-body weight ratio was prominently lower in maternal hypoxia offspring compared with control, indicating a relative increased head circumference and decreased abdominal circumference at birth. But there were no obvious differences in mean weight ratios of heart, lung, and kidney. N-cadherin was expressed in both atrium and ventriculium, predominantly localised to adherent junctions in intercalated discs. There was a significant down-regulated protein expression of N-cadherin in prenatal nicotine exposure offspring when compare with control.

Conclusions Prenatal nicotine exposure resulted in intra-uterine growth restriction with disproportion in neonatal organ size as a sign of relative increased head circumference and decreased abdominal circumference at birth of offspring who exhibited a postnatal catch-up growth. These growth characters are associated with a higher risk of cardiovascular disease. Furthermore, we demonstrate a down-regulated protein expression of N-cadherin in the myocardium from prenatal nicotine exposure offspring, which might predispose for impaired cardiac function in later life.

[gw22-e0741]

PRENATAL NICOTINE EXPOSURE MODULATES FETAL GROWTH AND REDUCES MYOCARDIAL N-CADHERIN EXPRESSION

Wang Zhenhua, Huang Ziyang *Second Affiliated Hospital Of Fujian Medical University, Fujian, China*

10.1136/heartjnl-2011-300867.234

Objectives To investigate the impact of prenatal nicotine exposure on the birth weight, body shape at birth and postnatal growth of the offspring, and the expression of myocardial N-cadherin.

Background Epidemiological studies have demonstrated that low birth weight, relative increased head circumference, and decreased abdominal circumference at birth, and postnatal catch-up growth by the suboptimal intrauterine environment are associated with increased risk of death from cardiovascular disease. The influence of prenatal nicotine exposure