

The effects of chronic moderate prenatal ethanol exposure on cardiovascular and renal artery function in adult rats

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Maternal alcohol consumption during pregnancy remains common in society today. Prenatal exposure to high levels of alcohol can cause developmental abnormalities. The effect of more moderate alcohol exposure on the offspring remains unclear. An adverse environment in early life can increase the risk of cardiovascular disease in adulthood. The aim of this study was to examine the effects of chronic moderate fetal alcohol exposure on arterial pressure and vascular function in adult rat offspring. Female rats were given a complete liquid diet containing either ethanol (6% v/v equating to 15% of total calories and a peak blood alcohol content of 0.03 – 0.05%) or an isocaloric equivalent during pregnancy. Male ($n = 6-9$) and female ($n = 7-8$) offspring were studied at 1 year of age. Mean arterial pressure (MAP) was recorded under basal conditions and during restraint stress *via* radiotelemetry. At *post mortem*, the kidneys were removed and the renal interlobar arteries were isolated. Segments of renal interlobar artery were mounted onto a wire myograph for testing of smooth muscle and endothelial function. Arteries were bathed in warmed, oxygenated physiological saline (PSS). Other segments of artery were mounted onto a pressure myograph and bathed in 0mM Ca^{2+} PSS containing 1mM EGTA, for the assessment of passive wall stiffness. Basal MAP and heart rate (HR) were not different between vehicle and alcohol-treated groups. MAP and HR increased significantly in response to restraint stress, however, the increase in MAP was lower in alcohol-treated groups of both sexes ($p = 0.001$). Constriction of the renal interlobar artery evoked by single pulses of perivascular nerve stimulation was smaller in alcohol-treated females ($p = 0.012$), but not in the males. Vasoconstriction evoked by angiotensin II and phenylephrine, and vasodilation evoked by the nitric oxide donor sodium nitroprusside, were not altered with alcohol treatment. Total endothelium-dependent relaxation and the relaxation due to endothelium-derived hyperpolarizing factor were not different between treatment groups. Interlobar arteries from alcohol-treated females were modestly more compliant ($p = 0.02$), but arterial stiffness was not different between treatment groups for the males. In conclusion, this study demonstrates that even moderate maternal alcohol consumption, equivalent to 2 standard drinks per day, during pregnancy does have lasting effects on the cardiovascular system of the offspring.