Extended Abstract Journal of Clinical Developmental Biology 2017
Vol.2 No.3

Metabolic Syndrome 2017: Developmental Programming of Obesity-related Hypertension.

Sarah L Henry[1], Benjamin Barzel[1], Ryan J Wood-Bradley[1], Sandra L Burke[1], Geoffrey A Head[1], James A Armitage[1]

[1]Department of Anatomy and Developmental Biology, Monash University, Clayton

The past 30 years has seen a secular trend of increased overweight and obese humans worldwide. In 2009, it was estimated that approximately 60% of Australian adults were obese or overweight. The prevalence of childhood obesity has also risen alarmingly; in Australia there was a 230% increase in obesity in young boys and a 170% increase in obesity in young girls between 1985 and 2004. These trends are not limited to Australia; The World Health Organization (WHO) predicts that, in the 21st century, the burden of obesity related disease will exceed that of infectious disease and malnutrition. Once considered to be exclusively within the purview of Western societies, the incidence and prevalence of obesity is rising sharply in developing nations. For example, a recent study of slum dwellers in New Delhi found obesity in approximately 10% of men and 40% of women. One of the major sequela of obesity is hypertension. Data from the Framingham Heart Study implicate obesity as a contributory factor in 60–70% of cases of essential hypertension. Although some early studies postulated that the hypertension associated with obesity was less of a cardiovascular risk, subsequent analyses of large cohorts indicated that obesity-related hypertension poses a significant risk to morbidity and mortality. In Australia, it is estimated that over 50% of the burden of diabetic and cardiovascular disease1 are obesity related and the financial cost of obesity in Australia is an estimated $3.5 billion per year. The steep increase in the prevalence of obesity worldwide is due to a range of factors; increased intake of total calories, fatty acids and simple sugars and a reduction in physical activity. Data from the UK suggest that the average person receives 36% of their daily total energy from all fats, with saturated fats a major component of this daily caloric intake. Individuals in many developed nations, including Australia, the US and Europe, derive more than 900 calories per day from fats, oil and sugar. In addition to the importance of the above risk factors, the aetiology of obesity is complex and additional factors must be contributing to overall prevalence rates.

In the past 30 years the prevalence of obesity and overweight have doubled. It is now estimated that globally over 500 million adults are obese and a further billion adults are overweight. Obesity is a cardiovascular risk factor and some studies suggest that up to 70% of cases of essential hypertension may be attributable, in part, to obesity. Increasingly, evidence supports a view that obesity-related hypertension may be driven by altered hypothalamic signalling, which results in inappropriately high appetite and sympathetic nerve activity to the kidney. In addition to the adult risk factors for obesity and hypertension, the environment encountered in early life may 'programme' the development of obesity, hypertension and cardiovascular disease. In particular, maternal obesity or high dietary fat intake in pregnancy may induce changes in fetal growth trajectories and predispose individuals to develop obesity and related sequelae. The mechanisms underlying the programming of obesityrelated hypertension are becoming better understood. However, several issues require clarification, particularly with regard to the role of the placenta in transferring fatty acid to the fetal compartment, the impact of placental inflammation and cytokine production in obesity. By understanding which factors are most associated with the development of obesity and hypertension in the offspring, we can focus therapeutic and behavioural interventions to most efficiently reduce the intergenerational propagation of the obesity cycle.

Hypertension in obesity is associated with elevated renal SNA and altered hypothalamic responses to insulin and leptin. Although clear adult risk factors exist, it is also likely that exposure to maternal obesity, overweight or a maternal diet rich in saturated fatty acids can programme changes in the hypothalamus that promotes the development of obesity-related hypertension in later life. The transfer of lipids across the placental barrier to the fetus may be one of the programming 'vectors'. Moreover, fetal exposure to cytokines of placental or fetal membrane origin may represent another programming vector. As we gradually identify the mechanisms that may drive the developmental programming of obesity and hypertension, we can then look to devise behavioral or therapeutic strategies to mitigate the development of a disease that affects billions of humans worldwide.

This work is partly presented at 2nd International Conference on Metabolic Syndrome, August 10-11, 2017, London, UK.