Plenary 1: Nutrition in-utero

Beyond birthweight: findings from the Avon Longitudinal Study of Parents and Children

AR Ness

Department of Social Medicine, University of Bristol, UK

Background – Observational studies have reported associations between size at birth and both higher levels of cardiovascular disease risk factors and increased risk of cardiovascular disease in adult life. It has been suggested that these associations provide evidence of programming in humans. While programming is well recognised in animals the existence and importance of programming in humans is disputed. In this paper I will summarise analyses from the Avon Longitudinal Study of Parents and Children (ALSPAC) that have attempted to clarify the meaning of these associations with size at birth and to explore associations between early life exposures and offspring blood pressure.

Methods – ALSPAC is a prospective study investigating the health and development of children. 14,541 pregnant women living in one of three Bristol-based health districts in the former County of Avon with an expected delivery date between April 1991 and December 1992 were recruited. Detailed information has been collected from pregnancy onwards using questionnaires, data extraction from medical notes, linkage to routine information and at clinics that the children have been invited to attend from the age of seven years.

Results – Birthweight was associated with blood pressure at age 7 - in unadjusted models a 1kg higher birthweight was associated with lower systolic blood pressure 0.8mmHg (95% CI -1.4, -0.3) in boys but not in girls. (1) We have showed that birthweight was positively associated with measures of lean mass (320g per SD increase in birthweight, p=0.001) and fat mass (2.5% per SD increase in birthweight, p =0.001) at age 9 and that ponderal index at birth was more strongly associated with fat mass than birthweight. (2) Using results from several studies including ALSPAC a common variant in the glucokinase gene - the A allele at GCK(-30) - was shown to be associated with a 0.075 mmol/L (p=0.003) increase in fasting plasma glucose in pregnancy. (3) Maternal genotype was associated with a higher offspring birthweight of 64g (95% CI 25-102) but child genotype was not associated with birthweight. (3) We showed that birthweight was associated with fat intake at 43 months but not at age 7. (4) We found no evidence that maternal age (5), maternal diet (6) or maternal smoking were associated with blood pressure. (7) Conclusions – We have found some evidence to support the suggestion that shared genetics may in part explain birthweight-cardiovascular disease associations. We have found little evidence that early life exposures are associated with offspring blood pressure in this well-nourished contemporary population.

References