Concurrent Session 1: Nutrition for Childhood and Adolescence

Birth size, postnatal growth and obesity

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Obesity is a serious public health problem in Australia. The most recently available national representative data from the 1990s suggest that approximately 19-23% of Australian children and adolescents are overweight or obese and there is evidence to suggest that the prevalence is increasing.¹ ² Data from five recent surveys over the periods 1969 to 1997 show that the prevalence of overweight increased by 60-70% and the prevalence of obesity trebled. The changes in prevalence between 1969 and 1985 were smaller than the subsequent 12 years indicating that the rates of obesity may also be accelerating.¹

Treatment of established obesity is challenging and a focus on primary prevention is essential. Currently, there is considerable interest in the role of birth size, a proxy for prenatal growth, growth in infancy and childhood, early puberty and the risk of obesity. Several studies, including data from a prospective study in western Sydney, the Nepean Study, have shown that lower birth size is associated with early pubertal development and increased risk of adult disease including cardiovascular disease and type 2 diabetes.³ ⁴

Most studies report a direct association between birth weight and attained body mass index (BMI): high birth weight is associated with a high BMI. The positive association appears to contradict evidence that low birth weight programs an increase risk of cardiovascular disease and type 2 diabetes. However, BMI is a measure of relative weight and does not distinguish between lean and fat mass. There is limited evidence, including results from the Nepean Study, that lower birth weight is associated with a subsequent greater abdominal or central fat mass and a higher ratio of fat mass to lean mass.³ ⁶ In contrast, high birth weight is associated with a relatively greater proportion of lean mass.

Both obesity and birth size have also been associated with earlier pubertal development. It is not clear if increased adiposity in childhood causes earlier sexual maturation, if earlier sexual maturation induces an increase in adiposity, or whether both these phenomena co-occur. Results from the Nepean Study indicate that after adjusting for BMI at 8 years of age girls who were long and light at birth attained menarche 1 year earlier (mean age of menarche ± SEM: 12.0 ± 0.3 yr) than girls who were short and heavy (13.0 ± 0.3 yr). Early maturation with obesity has been associated with polycystic ovarian syndrome, insulin resistance and a higher risk of adult disease including metabolic syndrome and cancer.

The biological basis for the association between birth size, early pubertal development and increased adiposity has been attributed to developmental plasticity and rapid or compensatory growth in infancy and early childhood. Infants who have been growth restrained in utero tend to gain weight more rapidly during the postnatal period. Different developmental pathways are thought to be triggered by environmental events induced during sensitive or critical periods in the development. However, whether there are or not early critical periods in programming of obesity, is still a matter for debate.

References