

# Prenatal Experience And Childhood Obesity

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## **Abstract**

There is growing interest in the role of early experience in the aetiology of obesity. In particular, prenatal life may be of importance as a 'critical period' when the risk of development and persistence of overweight and obesity is increased. At present developmental influences on obesity in humans are poorly understood, although there is increasing evidence of links between prenatal factors and adiposity in childhood. These factors include maternal obesity, excessive weight gain in pregnancy, gestational diabetes, and maternal smoking. A predisposition to gain excess weight in childhood may in part be the consequence of influences acting during fetal life.

There is growing interest in the role of experience in early life in the risk of becoming overweight or obese. Attention has been focused on prenatal life as a ‘critical period’ when the risk of development and persistence of overweight and obesity is increased [1]. Although at present developmental influences on obesity are poorly understood, there is increasing evidence that maternal factors that influence the intrauterine environment are associated with offspring body composition. This paper examines some of the epidemiological evidence that links prenatal experience to body composition in postnatal life.

## Maternal obesity

Maternal obesity is a strong predictor of overweight and obesity in children [2, 3]. This was clearly shown in a recent systematic review and meta-analysis that examined the association between pre-pregnancy maternal obesity and child overweight and obesity; children of mothers who were obese prior to pregnancy were three times more likely to be overweight or obese (OR 3.06; 95% CI 2.68-3.49;  $p=0.001$ ) when compared with children born to mothers who had a normal body mass index (BMI) [3]. The meta-analysis findings were consistent with the results from seven of the eight other studies included in the systematic review but not pooled into the meta-analysis. For example in the National Longitudinal Survey of Youth, children aged 6-7 years whose mothers were obese ( $BMI \geq 30\text{kg/m}^2$ ) prior to pregnancy were almost three times more likely to be overweight (OR 2.89, 95% CI: 2.02, 4.15) [4]. In this study the association between maternal and child obesity became more marked with increasing age of the children. The authors suggest that the effect of maternal obesity on childhood overweight is the result of an early persistent propensity to gain excess weight, that is perpetuated as the child ages [4].

A problem in the interpretation of mother-child correspondence in obesity is the potential effects of the shared postnatal environment, and the impact of common patterns of diet and physical activity on risk of gaining excess weight. An important consideration therefore in understanding direct effects of maternal obesity on the offspring comes from the comparison with the effects of paternal obesity. Direct effects of maternal obesity acting in intrauterine life should result in a relatively greater correspondence in BMI between mother and child, when compared with that of father and child. However, a meta-analysis of seven prospective cohort studies found that the association between offspring obesity and pre-pregnancy adiposity of the mother and father is not consistent across studies [5]. There is some evidence of a stronger maternal effect [6, 7], but few studies have made formal comparisons of the magnitude of effects, and the evidence is not conclusive [8-10].

An important study that may provide insights into the direct effects of maternal obesity was published by Kral and colleagues in 2006 [11]. In this study, the prevalence of obesity was compared in children aged 2 to 18 years who were conceived and born to 113 obese mothers before or after they had weight loss surgery [11]. The prevalence of overweight and obesity among 45 children born before maternal surgery was 60%, compared with a prevalence of 35% among 172 children who were born after surgery. These data suggest that obesity surgery had prevented the transmission of obesity to the offspring, and provide strong support for a direct influence of maternal obesity acting on the intrauterine environment, that has long-term effects on the offspring and their regulation of body weight.

## Weight gain in pregnancy

The optimal pattern of weight gain in pregnancy is not known. There is considerable observational evidence that links greater gestational weight gain to increased adiposity in the offspring [12-16]. For example, among 1044 mother-child pairs studied in Project Viva, child overweight at the age of 3 years was associated with greater gestational weight gain (OR 1.30, 95% CI: 1.04, 1.62 for each 5 kg weight gained) [17].

Much of the evidence of a link between gestational weight gain and offspring adiposity has been found when gestational weight gain is considered using the US Institute of Medicine categories of recommended weight gain, which define appropriate weight gain according to maternal BMI. Compared with women who gained adequate weight, the offspring of women who gain excess weight are at an increased risk of obesity [12, 13]. In two meta-analyses, the odds of overweight in the offspring were 1.33 (95 % CI 1.18–1.50) [16] and 1.38 (95% CI: 1.21–1.57) [14] for children born to mothers who had gained excess weight. In another meta-analysis, the risk of overweight in the offspring was 1.4 times greater (95% CI: 1.23–1.59) [13]; the relationships were similar when stratified by life stage, suggesting that excess gestational weight gain influences offspring obesity over the short- and longer-term.

Although there are difficulties in the interpretation of BMI in childhood [18], two recent UK studies that used direct measures of body composition assessed by dual-energy X-ray absorptiometry have shown consistent associations. Greater adiposity was found in children aged 9 years in the ALSPAC cohort [19] and children aged 6 years in the Southampton Women’s Survey [20], who were born to mothers who had gained excessive weight in pregnancy.

The association between inadequate gestational weight gain and risk of obesity is less clear. While there is some evidence that a U-shaped association exists, such that inadequate gestational weight gain may also be associated with increased risk of later obesity [20, 21], other evidence has shown a reduced risk [13, 14], in that the offspring of women who gained inadequate gestational weight were at a decreased risk of obesity (relative risk [RR]: 0.86; 95% confidence interval [CI]: 0.78–0.94) [13]. Less is known about the timing of GWG, although current evidence points to high rates of gestational weight gain during early- and mid-pregnancy as being important for later risk of child overweight and obesity [12].

Excessive gestational weight gain is common [20], and even in the US where the IOM guidelines are promoted [17]. There is also some evidence that the prevalence is increasing [22]. However, much of the evidence that links gestational weight gain to offspring adiposity and overweight is observational; follow-up of intervention trials will be needed to determine the impact of interventions to prevent excess weight gain in pregnancy on these outcomes [23].

## Maternal glycemia and gestational diabetes

A proposed mechanism to explain the link between maternal obesity and excess weight gain is that the fetus is overnourished (the ‘fetal overnutrition’ hypothesis), due to exposure to high maternal plasma concentrations of glucose, free fatty acids and amino acids [1, 9]. In postnatal life, there may be permanent consequences of prenatal overnutrition, that include effects on appetite control,

neuroendocrine function and energy metabolism – with lifelong consequences for the offspring’s ability to regulate energy balance and body weight [9].

Consistent with this possibility, fasting glucose concentration among diabetic women has been shown to be a strong predictor of fat mass in the offspring [24]. Gestational diabetes is associated with greater adiposity in the offspring at birth [25] and in childhood [26, 27]. Evidence of the effects of diabetes on the intrauterine environment has come from a study of Pima Indian mothers and children. Among children who were born to women who developed diabetes, BMI was greater in those who were born after the diagnosis, when compared with their siblings who were born before [28]. Notably, there were no associations with a diagnosis of paternal diabetes, which highlights the importance of the effects of the intrauterine environment, rather than genetic effects, on the development of postnatal body composition.

Part of the association between gestational diabetes and childhood obesity may be explained by maternal obesity [29-31]. In a recent systematic review, crude odds ratios for the relationship between gestational diabetes mellitus and childhood overweight or obesity ranged from 0.7 to 6.3 [29]. However, when pre-pregnancy obesity has been adjusted for, the association between gestational diabetes mellitus and childhood overweight and obesity was either no longer present [29, 31] or with reduced odds ratios ranging from 1.6 to 2.3 [30].

Importantly, an effect of maternal glycemia on offspring body composition may not be restricted to women who have gestational diabetes. For example, Hillier and colleagues [32] showed in a follow-up study of 9,439 women with normal glucose tolerance at initial screening, that increasing maternal glycemia was associated with a greater risk of obesity in the children, even amongst children of normal birthweight. Furthermore, in children born to women who fulfilled the criteria for gestational diabetes, the relationship between maternal glycemia and offspring obesity was lost if the mother received treatment.

## **Maternal smoking**

Observational studies show a consistent link between maternal smoking in pregnancy and an increased risk of offspring overweight and obesity. Two recent meta-analyses have shown that the risk of overweight and/or obesity among children born to mothers who smoked in pregnancy was 1.50 (95% CI 1.36, 1.65) [33] and 1.52 (95% CI 1.36, 1.70) [34]. Although smoking is associated with a range of sociodemographic and lifestyle factors, such that smokers tend to have less healthy diets and lifestyles, the risk estimate was little changed by adjustment for the effects of confounding influences [33]. Maternal smoking during pregnancy has also been found to have a higher risk on child overweight and obesity in comparison to paternal smoking, highlighting the importance of the prenatal exposure. The offspring of mothers who smoked in pregnancy had an increased risk of overweight (1.33; 95% CI 1.23, 1.44) and obesity (1.60, 95% CI 1.37, 1.88) in comparison to paternal smoking (overweight 1.07, 95% CI 1.00, 1.16 and obesity 1.23, 95% CI 1.10, 1.38) [35].

The mechanisms to explain the effect of smoking on risk of overweight in the offspring are not understood, but may result from fetal exposure to nicotine which has long-term effects on the control of food consumption and other appetitive behaviours [36]. It does not appear to be mediated by differences

in the pattern of growth of children exposed to maternal smoking in pregnancy [36]. A number of lines of evidence suggest that smoking may be causally linked to overweight in children, most notably the evidence of a dose-response effect that has been demonstrated in some studies [26]. However studies of the effects of smoking cessation in pregnancy have not shown consistent effects on overweight in the offspring [36]. Future studies are needed to elucidate the role of confounding factors in this association [33].

## Summary

The prevalence of obesity in childhood is increasing, and is recognised as a major health problem. This has led to widespread research efforts to understand the factors involved in its aetiology, including the role of the ‘obesogenic’ environment, and the importance of unhealthy dietary behaviours and patterns of physical activity [37]. However, despite the undoubted importance of these factors in contributing to gains in adiposity, it is important to recognise that not all children within a population become overweight. Understanding variations between individuals in the way that they interact with their environment, and the extent to which these interactions predispose some children to gain excess weight from early life, will be key to developing future strategies to prevent obesity. In children, prenatal life may be a critical period when the long-term regulation of energy balance is permanently ‘programmed’ [7]. Importantly, increases in the risk of overweight and obesity in childhood that have been linked to differences in prenatal experience are large [38]. Although the mechanisms that link prenatal experience to development of obesity are not fully understood, there are implications of the existing findings that could be disseminated, as it is already evident that obesity in today’s mothers is linked to obesity in their children. Maternal obesity is a modifiable risk factor, and policies and interventions are needed that support young women and encourage them to have a healthy body weight before pregnancy - both for their own health as well as that of their children.

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## Jenna Hollis



Dr Jenna Hollis is a postdoctoral researcher at the *Rowett Institute of Nutrition and Health* and *University of Aberdeen* in Scotland. Jenna has worked in research for 7 years and is passionate about obesity prevention, public health nutrition, and women's and children's health. She graduated from the University of Newcastle (Australia) with a Bachelor of Nutrition and Dietetics (Honours I) in 2009, and a PhD in Nutrition and Dietetics in July 2014. Her PhD examined the effectiveness of a 12-month health professional-led intervention on dietary and physical activity behaviour change to prevent weight gain in mid-age Australian women.

Prior to relocating to the United Kingdom, Jenna carried out research at the University of Newcastle, University of Canberra and Hunter New England Local Health District Population Health Unit (Australia) across numerous nutrition and physical activity studies including the 40-Something Study, Physical Activity for Everyone (PA4E1) RCT, SHED-IT, and the Healthy Habits RCT. She has undertaken additional training in biostatistics and epidemiology. Jenna has presented her research at both international and national conferences, and has 6 peer reviewed papers and a further 2 under-review.

After completing her PhD, Jenna moved to Scotland in October 2014 to join the *Rowett Institute of Nutrition and Health* as a Research Fellow with the Public Health Nutrition Research Group. Her position is funded by the *Rural and Environment Science and Analytical Services Division (RESAS)* programme of the Scottish Government. Her research focuses on i) evaluating simplified methods of monitoring the progress of the diet of the population towards healthy eating targets such as the Eatwell Plate, and ii) assesses the relationships between diet, food availability and affordability using the Kantar Worldpanel Survey of household food purchases across Scotland.

Jenna recently obtained an Endeavour Research Fellowship, funded by the Australian Government, to investigate the dietary and lifestyle predictors of maternal and child health and the developmental origins of health and disease in cohorts of women and children from the United Kingdom and Australia. She will commence the fellowship in November 2015 at the MRC Lifecourse Epidemiology Unit at the University of Southampton.

## Siân Robinson



Siân Robinson is Professor of Nutritional Epidemiology at the MRC Lifecourse Epidemiology Unit at the University of Southampton, UK. Her research addresses the contribution of nutrition across the lifecourse to inequalities in adult health, with a particular focus on early life influences on growth, development and later function, and effects on sarcopenia and ageing. Her main interests are in body composition in childhood and in older adults, in understanding how normal variations in dietary behaviour impact on these outcomes, and the extent to which nutrition in early life conditions adult responsiveness to diet and lifestyle. She is responsible for the nutritional components of the large UK cohorts run by the MRC Lifecourse Epidemiology Unit and is a Research Area Lead for the NIHR Southampton Biomedical Research Centre.

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