Preventing childhood obesity: Early-life messages from epidemiology

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Abstract

The high rates of overweight and obesity, currently seen in young children, underline the urgent need for preventive strategies in early life, before excess weight is gained. However, alongside such practical considerations, a body of epidemiological evidence now links experience in fetal and early postnatal life to an individual's later risk of obesity – pointing to the importance and role of 'developmental influences', such as maternal obesity, excess gestational weight gain and short duration of breastfeeding, in the aetiology of childhood obesity. Differences in early experience are linked to lifelong differences in predisposition to gain weight and, as the associated differences in obesity risk are large, real benefits could be achieved by early intervention. Such messages, regarding the need for efforts to address these early influences to prevent obesity, are now embedded in national and international health policy. Whilst successful prevention initiatives are needed early in life, the most effective strategies may need to be focused even earlier in the life course, in the period before conception. Although the challenges of changing behaviour are considerable, such changes have the potential to impact both on the health of future mothers and future generations.

Keywords: childhood obesity, early life, life course, preconception, programming

Childhood obesity

The rapid increases in the prevalence of overweight and obesity, seen worldwide in recent decades, have made this a public health priority (Butland *et al.* 2007). In the UK, although there is some evidence to suggest a slowing rate of increase, rates remain very high. For example, the *Health Survey for England* (2015) showed that the majority of adults studied were overweight (41% of men, 31% of women) or obese (affecting a further 27% of men and women).

These are some of the highest levels of overweight and obesity seen in Western Europe (FAO 2013). These changes are not confined to adults; recent (2015/2016) data from the *National Child Measurement Programme* (*NCMP*) show that more than one in five children in England are now overweight or obese by the time they start primary school, with rates increasing to one in three by the end of Year 6. To put these statistics in context, using data from 1974, soon after the establishment of the British Nutrition Foundation (BNF), the *National Study of Health and Growth* found that less than 10% of English children aged 4–6 years were overweight and fewer than 2% were obese (Chinn & Rona 2001). As body composition 'tracks', such that children who

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are already overweight at school age are much more likely to remain overweight (Park *et al.* 2012), the current obesity rates predict poorer long-term health as well as greater likelihood of associated negative effects on quality of life, educational achievement and social discrimination (Williams *et al.* 2005; Park *et al.* 2012; Lobstein *et al.* 2015), for a sizeable part of the UK population. Furthermore, existing inequalities in the distribution of overweight and obesity, with greater rates found among disadvantaged groups, means that this future burden will not be evenly spread (NCMP 2016).

So how did this happen? The growing awareness of the problem of childhood obesity has led to widespread research efforts to answer this question and to devise preventive and treatment strategies for the future. Underpinning the secular trends in obesity are environmental changes: today's children are growing up in increasingly obesogenic environments that encourage and promote food consumption. Changes to the food environment include the ready availability of energy-dense snack foods (Kerr *et al.* 2009), provision of fast food and meals consumed outside the home (Bowman *et al.* 2004) and increased portion sizes (Young & Nestle 2002); at the same time, higher energy intakes may be matched by increasingly sedentary lifestyles. Such changes favour positive energy balance and are likely to be major influences on the rising numbers of children who gain excess weight. However, as only some children within a given population, living within the same setting, become overweight, it suggests that children differ in their responses to their environment. Understanding how they differ, and the extent to which these interactions predispose some children to gain excess weight from early life, may be central to understanding the aetiology of childhood obesity.

Epidemiology and early-life experience

The high rates of overweight and obesity seen in young children point both to an urgent need for preventive strategies and to the timing of such interventions – clearly, they are required in early life, before excess weight is gained. However, alongside these practical considerations, epidemiological evidence accrued over the past 30 years, which has enabled a new understanding of 'developmental influences' on lifelong health, places further emphasis on the importance of experience in early life (Hanson & Gluckman 2014). Much of this started with the innovative epidemiological studies of historical birth cohorts, published by David Barker and colleagues in the 1980s. Their studies provided evidence, for the first time, to link poorer fetal and infant growth to later risk of coronary heart disease, stroke and type 2 diabetes (Barker *et al.* 1989; Osmond *et al.* 1993). The associations were not explained by differences in adult lifestyle and, importantly, they have since been replicated in studies in different populations across the world (Stein *et al.* 1996; BNF 2013). Elucidation of the mechanisms that underpin these findings is now an international research effort, to understand the permanent 'programmed' effects on physiology and function linked to impaired growth, which determine individual vulnerability to these non-communicable conditions and result in lifelong differences in health. The epidemiological studies helped to transform the way that early-life experience is considered – with widespread impact both on the scientific research agenda through to the formulation of health policy. But perhaps the major change is the value now placed on ensuring that the nutrition of mothers and young children is prioritised, as essential not only for their own health but also for that of their children (Robinson 2015).

In the intervening decades, since the first historical cohort findings were published, new epidemiological and experimental studies have enabled exploration of the role of developmental influences on obesity and led to greater understanding of early-life factors that can impact on an individual's propensity to gain excess weight – a lifelong predisposition that will be a major contributor to their risk of obesity. Understanding such developmental effects will be essential to inform the design, in terms of timing and nature, of effective early-life interventions to prevent obesity in the future.

Early-life risk factors

A range of potential early-life influences on childhood obesity has been identified; as summarised in a number of systematic reviews (Monasta *et al.* 2010; Woo Baidal *et al.* 2016). Although much of the epidemiological evidence is observational, the number of studies and the consistency in their findings, together with experimental data, have established the role of early-life experience as a major influence on childhood obesity. This short review sets out some of the insights gained from epidemiological studies, which provide clues to the effects of early nutritional factors, and considers the potential impact on childhood obesity and how they might inform future preventive strategies.

Maternal obesity

Familial concordance in overweight and obesity is widely recognised. For example, among children in the UK *Millennium Cohort Study*, an increased risk of being overweight was already evident at 3 years of age if the child's mother was overweight [adjusted odds ratio 1.37 (95% CI, 1.18–1.58)] (Hawkins *et al.* 2009) and for children aged 7 years in the *ALSPAC* study the risk of being obese was increased fourfold among children born to an obese mother [adjusted odds ratio 4.25 (95% CI, 2.86–6.32)] (Reilly *et al.* 2005). A greater risk in later childhood was also observed in the *National Longitudinal Survey of Youth*, in which children were followed up when they were aged 2–3 years, 4–5 years and 6–7 years (Salsberry & Reagan 2005). At each age, maternal obesity was associated with an increased risk of overweight in the children but the risk increased substantially at older ages, suggesting that maternal obesity affects both the propensity to gain weight and the dynamics of childhood weight gain (Salsberry & Reagan 2005). There have been a number of studies of maternal obesity in recent years. Systematic review and meta-analysis of these data confirm the substantial differences in risk of offspring obesity; for example, maternal pre-pregnancy overweight/obesity has been shown to be associated with an increased risk of overweight or obesity in the offspring [in comparison with women of normal BMI: odds ratio (OR), 1.95; 95% CI, 1.77–2.13; and OR, 3.06; 95% CI, 2.68–3.49], respectively (Yu *et al.* 2013).

Apart from a genetic predisposition, part of the observed familial concordance is likely to be explained by a shared environment and patterns of behaviour, including common dietary habits (Fisk *et al.* 2011; Schrempft *et al.* 2016). However, there is now a wealth of experimental and other evidence suggesting that exposure to maternal obesity in fetal life has lifelong effects on the offspring's risk of obesity, which occur before such shared environmental influences act in postnatal life (Reynolds *et al.* 2015; Catalano & Shankar 2017). There are two lines of epidemiological evidence that are important. The first of these compares the effects of maternal obesity with paternal effects: direct effects of maternal obesity acting in fetal life would be expected to evident as stronger associations with offspring obesity risk, when compared with paternal effects. There is some evidence that this is the case (Murrin *et al.* 2012), although a systematic review of prospective cohort studies did not yield consistent findings (Patro *et al.* 2013) and further data are needed. The second type of study has compared children born to mothers before and after weight loss. For example, among children born to 113 obese mothers before and after undergoing weight loss surgery, the prevalence of overweight and obesity was 60% in those born before surgery compared with 35% in those who were born after (Kral *et al.* 2006). More recently, children born after maternal bariatric surgery have been shown to have improved cardiometabolic risk, as well as lower rates of obesity, when compared to siblings born before surgery – profiles that are carried into adulthood (Guénard *et al.* 2013). The studies suggest that reduction in maternal adiposity can prevent the transmission of adiposity to the offspring and provide strong support for a direct influence of maternal obesity acting on the intrauterine environment, with long-term effects on the offspring and their regulation of bodyweight. Maternal obesity is associated with insulin resistance, altered glucose and fatty acid availability and low-grade inflammation – each of which may affect the developing fetus (Godfrey *et al.* 2017). Experimental studies, together with the growing epidemiological evidence, now point to permanent 'programmed' effects of maternal obesity on metabolic control processes in the fetus, which include hypothalamic responses to leptin and subsequent effects on regulation of appetite and pancreatic β-cell physiology (Godfrey *et al.* 2017). Epigenetic modifications have been proposed as a key mechanism that links maternal obesity to a range of offspring outcomes. In recent years, significant progress has been made in understanding the role of epigenetic mechanisms, including DNA methylation, post-translational modification of histones and non-coding RNAs, that result in changes in gene function. Current evidence, from a range of international studies, is summarised in a recent review by Godfrey *et al.* (2017). Although findings from different studies have not always been consistent, there is now evidence across a number of cohorts to link maternal BMI and offspring DNA methylation. However, irrespective of the need for a better understanding of the mechanistic basis of the long-term effects of maternal obesity on the fetus, enough is already known to highlight concerns regarding the current prevalence of overweight and obesity among women of childbearing age (Butland *et al.* 2007) – both for their own health, and the health of their offspring.

Gestational weight gain

Although the optimal pattern and amount of weight gain in pregnancy are not known, there is a large body of evidence that links higher gestational weight gain

(GWG) to obesity risk in the offspring. For example, in a recent systematic review, excess GWG [defined either according to the US Institute of Medicine (IOM 2009) criteria or using specified cut-points] was associated with an increased risk of childhood overweight in 19 of the 21 studies that were considered (Woo Baidal *et al.* 2016). And the effect size is significant; Tie and colleagues estimated the risk of child overweight or obesity associated with excess GWG, compared with adequate GWG, was 11.4% and 8.8%, respectively (combined OR of excessive GWG and childhood overweight/obesity 1.33, 95% CI, 1.18–1.50) in a meta-analysis of 12 cohort studies (Tie *et al.* 2014). Most studies have relied on BMI to define overweight and obesity, which may be challenging in children as between-subject differences in fat-free mass may be an important component of variability in BMI (Wells *et al.* 2002). However, more recent evidence from studies that have examined direct measures of adiposity has yielded messages that are consistent with effects on BMI. For example, in the *Southampton Women's Survey* (*SWS*), children born to women with excess GWG (defined according to IOM categorisation) had a greater DXA-assessed fat mass at 4 and 6 years (increases of 4% and 10%, respectively), when compared with children whose mothers' weight gain in pregnancy was adequate (Crozier *et al.* 2010). Such differences in body composition, seen in children born to mothers who had excess GWG, need to be considered in the context that excess weight gain in pregnancy is prevalent and becoming more common (Muktabhant *et al.* 2015); in the *SWS* example, excess GWG affected almost half the women studied (Crozier *et al.* 2010). Notably, although excess GWG may be more common among obese women (Catalano & Shankar 2017), effects on offspring obesity have been observed that are independent of maternal pre-pregnant BMI (Godfrey *et al.* 2017). In terms of the timing of weight gain in pregnancy, there is some recent evidence to suggest that GWG in early pregnancy is key (Karachaliou *et al.* 2015). Fat accumulation represents a large part of maternal weight gain in this period; experimental evidence suggests that maternal dysmetabolism, with increased fuel supply to the fetus associated with excess early GWG may be important, with long-term effects on offspring appetite regulation, potentially via epigenetic mechanisms (Karachaliou *et al.* 2015).

There is considerable interest in the use of diet and exercise interventions to limit weight gain in pregnancy to improve a range of maternal and offspring outcomes. A Cochrane review has shown that interventions to change diet, physical activity or both combined are effective; the risk of excess GWG was reduced on average by 20% (average risk ratio 0.80, 95% CI, 0.73–0.87) in the reviewed studies (Muktabhant *et al.* 2015). A smaller number of trials have used diet and physical activity interventions to limit GWG with the specific aim of reducing risk of offspring obesity and, to date, findings of lifestyle interventions have been mixed (Tanvig *et al.* 2014; Horan *et al.* 2016). However, further data are needed. The potential of interventions to limit GWG has been highlighted by recent findings from the *UK Pregnancies Better Eating and Activity Trial* (*UPBEAT*) in the UK, which assigned obese pregnant women to a behavioural intervention [targeting maternal diet (glycaemic load, saturated fat intake) and physical activity] or to standard antenatal care (Poston *et al.* 2015). No differences were observed in the primary outcomes (gestational diabetes, large-for-gestational-age infants) but there were improvements in some secondary outcomes, including GWG. Furthermore, in a recent follow-up of 698 *UPBEAT* infants, when they were aged 6 months, lower subscapular skinfold thickness was found in the intervention group [z-score −0.26 SD (−0.49 to −0.02)]. Although differences in maternal diet were sustained at 6 months in the intervention group, mediation analysis suggested that the lower infant adiposity observed was partially mediated by changes in antenatal maternal diet and GWG, rather than postnatal diet (Patel *et al.* 2017).

Duration of breastfeeding

Over the past decade, much attention has been focused on the impact of different types and patterns of milk feeding in infancy on the regulation of energy balance in later life, and the possibility that breastfeeding promotion could be used as part of efforts to prevent obesity. A number of seminal reviews have examined the evidence, showing protective benefits of breastfeeding (Arenz *et al.* 2004; Owen *et al.* 2005). However, this remains contentious as there are particular challenges in the interpretation of observational infant feeding data that need to be highlighted. For example, in developed settings, there are concerns regarding confounding effects arising from differences in maternal education and social status that are linked both to differences in feeding practice and to differences in childhood diet (Robinson 2015). The most recent large review, published by Horta and colleagues in 2015, is therefore significant. They identified 37 new publications that could provide information on

the association between breastfeeding and prevalence of overweight or obesity; the meta-analysis included data from 105 studies (Horta *et al.* 2015). Individuals who had been breastfed were less likely to be overweight or obese [pooled OR: 0.74 (95% CI, 0.70–0.78)]; the association was slightly stronger in studies that provided information on exclusive breastfeeding. There were some differences in effect size between studies, and adjustment for confounding effects of socio-economic position attenuated the observed associations. However, the overall evidence was of protective effects of breastfeeding, observed even among the larger studies [pooled odds ratio: 0.81 (95% CI, 0.76–0.87)] (Horta *et al.* 2015).

Much of the current evidence for the protective effect of breastfeeding on offspring overweight is observational, and trials to allocate infants to different patterns/types of milk feeding would be unethical. However, if breastfeeding is causally linked to offspring body composition, protective benefits should be evident in interventions to promote breastfeeding prevalence, exclusivity and duration. A recent review of breastfeeding promotion interventions therefore provides important information (Giugliani *et al.* 2015). This showed that there were no significant differences in offspring bodyweight or length associated with breastfeeding promotion, but there were modest reductions in offspring BMI and/or weight-for-height z scores. The results are consistent with protective effects of breastfeeding and with effects described in an earlier meta-analysis of observational data (Owen *et al.* 2005).

One possible explanation for the observed differences in later obesity risk is that infants who are breastfed learn to control the amount of milk they consume in early life, which leads to effective self-regulation of energy intake that remains lifelong. Consistent with this possibility is the evidence that early introduction of solid foods is associated with greater odds of later obesity in formula-fed infants but not breastfed infants, suggesting that breastfed infants were able to reduce their milk consumption when provided with other food, whereas the formula-fed infants were not (Huh *et al.* 2011). It is also possible that there are effects of the mode of feeding, as bottle feeding itself could undermine an infant's ability to regulate milk consumption (Li *et al.* 2010). More recently, a growing appreciation of the complexity of breast milk and its content of a diverse range of bioactive components, including antimicrobial and anti-inflammatory factors, enzymes, hormones and growth factors, has suggested new mechanisms. It raises the possibility that differences in early exposure to the

bioactive constituents in breast milk could be linked to long-term differences in body composition, possibly via epigenetic processes or effects on the establishment of the gut microbiota in infancy (Robinson 2015; Hartwig *et al.* 2017). Whilst the mechanisms are yet to be elucidated, rapid advances in understanding the mechanistic basis of such programmed effects means that progress in this area is likely to be seen soon.

Importance of early-life nutrition and policy implications

The body of evidence, accrued over recent decades, which links experience in fetal and early postnatal life to an individual's later risk of obesity, highlights the need for early preventive interventions and, as the associated differences in risk of obesity are large, real benefits could be achieved. For example, in the *SWS*, five modifiable early-life risk factors (maternal obesity, excess GWG, smoking in pregnancy, low maternal vitamin D status, short duration of breastfeeding) were considered; half (52%) of children studied had at least two of these risk factors (Robinson *et al.* 2015). Positive graded associations were found between the children's number of these early-life risk factors and obesity outcomes in childhood. The differences were large: there was a fourfold increase in the risk of being overweight or obese at 6 years for the children who had four or more risk factors, when compared with the children who had none (Fig. 1), and a difference in fat mass between these groups of 47%.
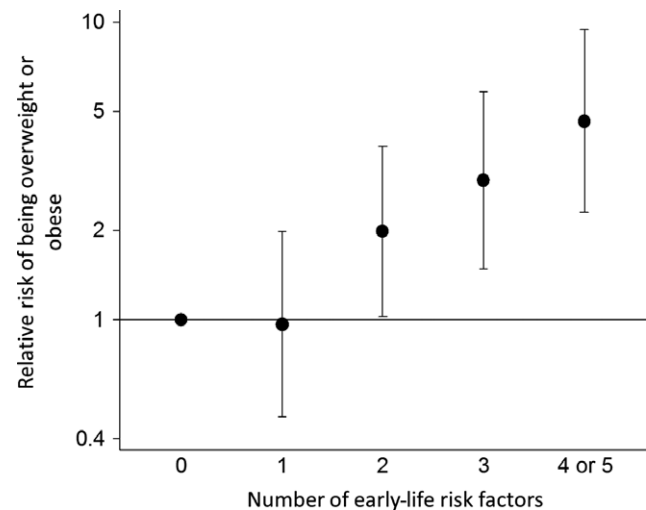
Figure 1 Relative risk (95% CI) of being overweight or obese at 6 years of age (International Obesity Task Force), according to number of early-life risk factors. Data adjusted for child's gestational age at birth, maternal height, education, parity and age at child's birth (Robinson *et al.* 2015).

Additionally, the associations were still evident after taking account of a range of potential confounding factors that included childhood level of physical activity and diet quality.

Over the past two decades, widespread recognition of the role and importance of early experience in the aetiology of obesity has developed, and this is now embedded in national and international health policy. For example, the life-course approach to obesity prevention is central to the World Health Organization's (WHO) *Commission on Ending Childhood Obesity* report, published in 2016 (WHO 2016). Whilst successful initiatives to prevent childhood obesity are clearly needed early in life, the most effective strategies may need to be focused even earlier in the life course, before conception. As data from experimental models suggest that the earliest stages of development are crucial, lifestyle interventions in mid-late pregnancy may be too late to be effective (Catalano & Shankar 2017). Preconception interventions offer huge potential to benefit offspring health but, as highlighted in recent reviews, to date, there has been a lack of trials focusing on the preconception period (Blake-Lamb *et al.* 2016; Reilly *et al.* 2017). The challenges of changing behaviour to achieve a healthy bodyweight before pregnancy may be considerable. Evidence from the *SWS* shows that there is little preparation for pregnancy in terms of changes in health behaviours and, notably, this is more marked among the most disadvantaged women (Inskip *et al.* 2009). Interventions and policies to encourage behaviour change need to include recognition of the influence of the wider social and economic environment, as well as consideration of how best to support individuals (Hanson *et al.* 2017). But, the impact of these changes could be significant both for future mothers and future generations.

Conflict of interest

No conflict of interests have been declared.

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