Impact of Maternal Obesity on Perinatal and Childhood Outcomes

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Maternal obesity is of major consequence, affecting every aspect of maternity care including both short- and long-term effects on the health of the offspring. Obese mothers are at a higher risk of developing gestational diabetes and pre-eclampsia, potentially exposing the foetus to an adverse intrauterine environment. Maternal obesity is linked to foetal macrosomia, resulting in increased neonatal and maternal morbidity. Foetal macrosomia is a result of a change in body composition in the neonate with an increase in both percentage fat and fat mass. Maternal obesity and gestational weight gain are associated with childhood obesity, and this effect extends into adulthood. Childhood obesity in turn increases chances of later life obesity, thus type 2 diabetes, and cardiovascular disease in the offspring. Further clinical trials of lifestyle and, potentially, pharmacological interventions in obese pregnant women are required to determine whether short- and long-term adverse effects for the mother and child can be reduced.

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Obesity: the scale of the problem

Obesity has become an epidemic throughout the world, and is considered one of the leading causes of death and disease in the industrialised world [1,2]. Maternal rates of obesity are no different to rates of obesity in the general population doubling over a decade [3]. In some parts of the UK, >20% of women of childbearing age are obese and the prevalence of obesity amongst pregnant women is spiralling, placing an unanticipated burden on health-care resources [4]. Maternal obesity is of major consequence in obstetrics and affects every aspect of maternity care including long-term effects on the future health of the offspring (Table 1). The purpose of this chapter is to highlight some of the key impacts of maternal obesity, both short and long term, on the offspring.

Short-term impact of maternal obesity on the offspring

Birthweight and foetal macrosomia

It is well recognised that birthweight is, in part, dependent on maternal nutrition [5,6], maternal pre-gravid weight [7,8] and weight gain during the pregnancy. [9]

Studies have shown positive correlations between maternal pre-pregnancy weight and gestational weight gain with the birthweight of the neonate and associated health risks [9]. The Institute of Medicine (IOM) published guidelines on recommended weight gain in pregnancy based on pre-pregnancy body mass index (BMI) [10] and several studies have examined the influence on maternal weight gain on birthweight. A Danish study investigated the association of maternal weight gain and birthweight in 2248 singleton term pregnancies and found that in normal-weight women there was an increased risk of birthweight <3000 g (OR 2.3 (1.5–3.7)) if the maternal weight gain in

<table>
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<tr>
<th>Obstetric complication</th>
<th>Increased risk associated with obesity</th>
<th>References</th>
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</thead>
<tbody>
<tr>
<td>Pre-eclampsia</td>
<td>RR 0.54% [0.27–0.80]</td>
<td>O’Brien et al., 2003 [48]</td>
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<tr>
<td>Gestational Diabetes</td>
<td>OR 2.14 [1.82–2.53] (overweight)</td>
<td>Ch SY et al., 2007 [16]</td>
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<td>OR 3.56 [3.05–4.21] (obese)</td>
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<td></td>
<td>OR 8.56 [5.07–16.04] (severely obese)</td>
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<tr>
<td>Preterm labour (nulliparous elective preterm delivery)</td>
<td>OR R 1.15 [1.03–1.27] (overweight)</td>
<td>Smith et al., 2007 [46]</td>
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<tr>
<td></td>
<td>OR 1.52 [1.31–1.77] (obese)</td>
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<tr>
<td></td>
<td>OR 2.13 [1.75–2.58] (severely obese)</td>
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<tr>
<td>Macrosomia</td>
<td>OR 2.1 [1.6–2.6]</td>
<td>Jolly et al., 2003 [22]</td>
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<tr>
<td>Adverse Perinatal Outcome</td>
<td>shoulder dystocia OR 2.9 [1.4–5.8]</td>
<td>Usha et al., 2005 [23]</td>
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<td></td>
<td>NNU admission OR 1.5 [1.09–2.3]</td>
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<td></td>
<td>Birth trauma (skin grazes, bruises, fractures, nerve palsies, muscle haematomas, cephalohaeatomas) OR 1.5 [1.1–2.1]</td>
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<tr>
<td>Miscarriage</td>
<td>RR 1.67 [1.25–2.25] (including all miscarriages &lt;20 weeks)</td>
<td>Metwally et al., 2008 [94]</td>
</tr>
<tr>
<td>Congenital anomalies</td>
<td>Neural tube defects RR 1.87 [1.82–2.15]</td>
<td>Stothard et al., 2009 [95]</td>
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<td></td>
<td>Spina bifida RR 2.24 [1.86–2.69]</td>
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<td>Cleft lip/palate RR 1.20 [1.03–1.40]</td>
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<td>Hydrocephaly RR 1.68 [1.19–2.36]</td>
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<td></td>
<td>Cardiovascular anomalies RR 1.30 [1.12–1.64]</td>
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Table 1
Summary of the complications and estimated risks associated with maternal obesity. (Values expressed OR = Odds ratio[95% confidence interval (CI)] or RR = relative risk[95% CI], obese versus lean unless otherwise stated).
higher risk of excessive weight gain in the antenatal period [13,14]. Given excessive weight gain in obese women is also associated with increased birthweight in neonates [12]. Obese women are at a higher risk of excessive weight gain in the antenatal period [13,14]. Given excessive weight gain in pregnancy per se is independently associated with birthweight and adiposity in the offspring, the neonate of lean women with excess gestational weight gain also may be at risk [11,15].

Macrosomia is known to be associated with diabetes in pregnancy. As highlighted in Chapter 5 of this book, given that obesity and pregnancy combined both confer a state of insulin resistance to the mother, it is not surprising that obese women are four times more likely and severely obese women are nine times more likely to develop gestational diabetes than lean women [16]. Evidence suggests that even modest fasting hyperglycaemia (4.2–5.6 mmol/l) is linearly associated with adverse outcome, such as increased birthweight and neonatal hypoglycaemia [17]. A study by Pettitt et al. assessed the risk of foetal macrosomia by maternal weight gain and glucose levels. Amongst their population of 80,000 mothers and newborns, they found that foetal macrosomia increased linearly with increasing maternal glucose levels, amongst women with a normal glucose tolerance test. Furthermore, they found that excessive gestational weight gain increased the risk of macrosomia across increasing levels of maternal glucose including gestational diabetes mellitus (GDM) [18].

Foetal macrosomia is linked not only to the absolute size of the foetus but also to the change in the actual body composition, with an increase in percentage fat and fat mass in the offspring of overweight and obese mothers [19]. Similar results were found in a study by Sewell et al., which demonstrated significant increases in percent body fat (11.6% (±4.7%) vs. 9.7% (±4.3%); p = 0.003) and fat mass (420 g (±220 g) vs. 380 g (±170 g); p = 0.01) in neonates of overweight/obese women versus lean/average weight women [20].

Furthermore, Catalano et al. have published a series of studies comparing the body composition analysis of infants to women with normal glucose tolerance (NGT) tests and GDM within 48 h of birth. There was no significant difference in the actual birthweight (GDM 3398 g (±550 g) vs. NGT 3337 g (±549 g), p = 0.26) or fat-free mass (GDM 2962 g (±405 g) vs. NGT 2975 g (±408 g), p = 0.74); however, there was a significant increase in fat mass (GDM 436 g (±206 g) vs. NGT 362 g (±198 g), p = 0.0002) and percentage body fat (GDM 12.4 g (±4.6 g), p = 0.0001) in the infants born to GDM mothers [21].

**Macrosomia, birth injuries and neonatal unit admission**

Foetal macrosomia contributes to significant challenges at delivery and in the immediate neonatal period. A UK-based cohort of 350,311 completed singleton pregnancies found macrosomia to be associated with prolonged labours, operative deliveries, shoulder dystocia, other birth traumas including nerve palsies and fractures, perinatal asphyxia and neonatal unit (NNU) admissions [22]. These findings have been replicated [23,24]. In a Welsh population-based cohort study of uncomplicated singleton term infants, they found adverse perinatal outcomes to be higher in the obese compared with the non-obese mothers including macrosomia (OR 2.1 (1.6–2.6)), shoulder dystocia (OR 2.9 (1.4–5.8)), NNU admission (OR 1.5 (1.09–2.3)) and birth trauma (including skin grazes, bruises, fractures, nerve palsies, muscle haematomas and cephalohematomas) (OR 1.5 (1.1–2.1)) Interestingly, mothers with pregnancy complications (e.g., diabetes) were excluded. Given that obesity has a strong association with conditions such as diabetes and pre-eclampsia, the adverse perinatal outcome of infants born to obese mothers is likely to be under-represented [23].

There is a well-established association between maternal obesity and neonatal admission to the NNU [25]. A recent study by Blomberg showed that the risk of adverse neonatal outcomes rises with increasing BMI regardless of the mode of delivery. Infants born to mothers of class 3 obesity had a twofold increased risk of skeletal birth injuries and respiratory distress. Furthermore, they had a threefold added risk of bacterial sepsis, convulsions, birth asphyxia, feeding difficulties and a fourfold increased risk of birth injuries to the peripheral nervous system and hypoglycaemia [26].

Obese mothers are less likely to labour spontaneously, more likely to have an induced labour, less likely to deliver vaginally and more at a risk of caesarean section [27,28]. This also impacts on higher rates of admission to the NNU for the management of transient tachypnea of the newborn secondary to retained lung fluid following caesarean section, especially a planned caesarean section [29].
Maternal obesity and breastfeeding

The benefits of breastfeeding to both the mother and baby are well established. The immediate postnatal period is thought to be crucial in programming long-term body weight and metabolism. It has been demonstrated that rapid weight gain in the neonatal period is a significant risk factor for developing obesity. Interestingly, it has been shown that for every increase in body weight by 100 g in the first week of life, even in babies born to non-obese mothers, the risk of obesity in adulthood rises by 28% [30].

The perinatal diet is thought to influence weight regulatory centres, by either promoting or reducing excessive weight gain. The findings of studies, however, are conflicting and confounding factors such as socioeconomic class and lifestyle factors may influence this. There is evidence, however, suggesting that infants who are exclusively breastfed are provided with a potential protective effect against obesity. Infants formula fed have been shown to exhibit faster weight gain than their breastfed counterparts and are at an increased risk of obesity in later life [31,32]. There are certain fatty acids (FA) contained in breast milk that are thought to possibly be protective against obesity. Such a FA supply, however, is supplied via the maternal diet, being found in meat and organic dairy products [33,34]. Conjugated linoleic acid (FA) isomers have been shown in animal studies to reduce fat gain and maintain insulin sensitivity in mice being given a high-fat diet. It is believed they increase energy expenditure and reduce body fat [35]. Furthermore, conjugated linoleic acid suppresses inflammation within the human body and promotes the production of natural anti-inflammatory agents [36].

In terms of obesity, there is evidence that maternal obesity is associated with reduced initiation, establishment and maintenance of breastfeeding. It is plausible that other confounding factors may influence this risk; however, this link still exists when corrected for age, parity, smoking status and educational level [37]. Maternal obesity is also a recognised risk factor for delayed lactogenesis that is compounded by an increased incidence of prolonged labour, operative deliveries and gestational diabetes that are all independently associated with poor lactogenesis [38-41].

Furthermore, in obese mothers, there is a mechanical difficulty in encouraging the infant to attach to heavier breasts and separation of the infant and mother soon after birth, due to higher risk of neonatal complications, with increased formula supplementation to treat neonatal hypoglycaemia [42].

Overall, it can be concluded that there are a number of factors which likely interplay and affect successful lactogenesis in obese mothers. The end result is of increased formula feeding, which may confer a risk of obesity to the neonate in childhood.

Maternal obesity and prematurity

Preterm birth/labour (PTL), defined as delivery under 37 weeks gestation, is the leading cause of neonatal morbidity and mortality, and this risk increases with decreasing gestation. Globally, an estimated 15 million babies are born prematurely annually, accounting for 11% of all live births. Approximately >1 million of these babies die as a result of morbidities related to prematurity. The concept of PTL is a complex one and clearly will be affected by a number of different factors involving the mother, foetus and intrauterine environment [26,43].

PTL can be either spontaneous or iatrogenic. Cohort studies have demonstrated that obese mothers are more at risk of iatrogenic delivery than spontaneous PTL [44,45]. A recent study by Cattingius et al. investigated the link between BMI category and preterm delivery by gestation. They found that all classes of preterm delivery (i.e., 22–36 weeks) increased with BMI and the risks related to obesity were highest for the extremely preterm infants (i.e., 22–27 weeks) [43]. Furthermore, a large retrospective record linkage study in Scotland investigated pregnancy outcomes for 187,290 women based on BMI. The study found there were statistically significant interactions between nulliparity and BMI for overall preterm deliveries, spontaneous preterm deliveries and elective preterm deliveries, with all p values <0.001. The risk of elective preterm delivery in nulliparous women increased with BMI in a linear fashion (BMI 25–29.9, OR 1.15 (1.03–1.27); BMI 30–34.9, OR 1.52 (1.31–1.77); BMI >35, OR 2.13 (1.75–2.58)). This association did exist but to a much lesser extent with parous women. A common
reason for planned preterm delivery within the nulliparous women with a BMI > 35 was pre-eclampsia, with 40.2% of women delivered being diagnosed with it, this compared to 18% in the multiparous group ($p < 0.01$). Spontaneous preterm delivery occurred less with increasing BMI (BMI < 20, OR 1.46 (1.32–1.62); BMI 25–29.9, OR 0.89 (0.73–0.99); BMI 30–34.9, OR 0.85 (CI 0.73–0.99); BMI > 35, OR 0.81 (0.63–1.04)). This protective effect against spontaneous preterm labour was stronger for multiparous women [46]. Overweight and obese mothers are known to be at an increased risk of maternal complications antenatally, including gestational diabetes, gestational hypertension, thromboembolism and pre-eclampsia [47]. Pre-eclampsia (PET) risk in pregnancy doubles for every 5–7 kg/m² increase in BMI [48]. The risk is doubled with a BMI of 26 and almost trebled when the BMI is > 30 compared to that of 21 [49]. Maternal and foetal complications arising from diabetes and pre-eclampsia often necessitate early delivery [50].

Finally, obesity is related with intrauterine growth restriction as well as macrosomia and if detected may warrant preterm delivery [49].

**Long-term impact of maternal obesity**

**Childhood obesity**

Maternal obesity has been linked to long-term adverse effects on the offspring [51–53]. Over the last 20 years, there has been a childhood epidemic of obesity [54]. It is now estimated that 42 million children under the age of 5 years are now overweight [55] and children born to obese mothers are more likely to be obese themselves [56]. Furthermore, childhood obesity has been identified as a risk factor for obesity in adolescent and adult life [57,58] independent of lifestyle, genetic and environmental factors [59]. As we have previously discussed, infants born to obese mothers are more at risk of being macrosomic and have increased body fat and fat mass compared to their average weight counterparts. In obese pregnancies, as a result of the in utero environment of relative maternal hyperglycaemia, the neonate is born hyperinsulinaemic and it has been hypothesised this hyperinsulinaemic state may exert long-term effects on the body by means of increasing adipocyte size and/ or number thus potentially contributing to childhood and adolescent obesity [60]. A study that measured amniotic fluid insulin levels, a surrogate marker of foetal insulin production, supported this hypothesis by demonstrating that higher levels of insulin correlated with adolescent obesity in infants born to diabetic mothers [60]. Additionally, a study amongst PIMA Indians showed that siblings born to mothers who concurrently were diabetic had a higher BMI between the ages of 9 and 24 years than those born to the same mothers who were not diabetic during that index pregnancy [61]. An American study interestingly found that for every 1-kg increase in the birthweight of term infants, there was approximately a 50% increased chance of obesity in these adolescents between the ages of 9 and 14 years [62]. Importantly, it is not only absolute BMI but also maternal weight gain in pregnancy which is relevant to the long-term risk of obesity in the offspring. A cohort study investigating the relationship of weight gain in pregnancy and pre-pregnancy maternal obesity found that a maternal weight gain of >7 kg increased the risk of adolescent obesity and abdominal obesity by 1.5-fold. Furthermore, this risk increased by fourfold if the mother had a pre-pregnancy BMI of >30 kg/m² compared with those of normal BMI [63].

The reasons for a potential long-term effect on childhood obesity is multifactorial and not yet fully understood, but one of the key proposed mechanisms may be through foetal programming when exposed to an unfavourable metabolic milieu in utero. Experimental evidence from both animal and human studies investigating the effects of a maternal high-fat diet on the offspring have shown that it leads to insulin resistance, hyperinsulinaemia and increased fat accumulation in the offspring [64–66]. Such effects are associated with the altered programming of “central reward pathways” leading to an increase in the rewarding nature of food and the preference for high-fat and saturated foods [67]. The hypothalamus is thought to be key to regulating body weight and metabolism. It is under hormonal control and leptin is thought to be one important hormone implicated in hypothalamic development. Leptin is derived from adipocytes and is a satiety hormone, which acts centrally in the arcuate nucleus of the hypothalamus to attenuate hunger and increase energy expenditure [68,69]. Furthermore, leptin contributes to the development of central pathways that regulate feeding and metabolism [68]. In
animal models, excess or an absence of leptin at crucial times during development, whether due to over- or under-nutrition, have been shown to interrupt the development of the circuit [70]. Animal studies have shown alterations to maternal diet during pregnancy or lactation can affect the magnitude and onset of the leptin surge, leading to long-term alternations in body weight regulation [71]. Babies carried by obese mothers may be exposed to high levels of leptin too soon and expose the immature hypothalamus prematurely to a leptin surge, to which it cannot respond appropriately [72]. If the hypothalamic centres are interrupted during their crucial period of development, the regions responsible for food intake may be altered [73] resulting in an excessive hunger state contributing to obesity [74]. The leptin theory of promoting obesity is clearly complex and leptin in humans is produced from other sources in addition to adipocytes [75].

The role of genetics in long-term offspring obesity

The underlying aetiology of obesity is multifactorial and complex. Both environmental and behavioural factors have contributed to the exponential rise in the prevalence of obesity since the early 1980s. These include abundance of cheap, processed food, enlarged portion sizes, widespread advertising encouraging the consumption of such foods and sedentary lifestyles [76]. Genetic factors have been implicated in rising obesity trends. The BMI is thought to be between 40% and 70% heritable, although <2% of the obesity-susceptible genetic loci have been identified [77]. Despite the role genetics may play in obesity, many of the identified obesity-susceptible loci have only small effects on BMI and obesity risk. An example to demonstrate this point is of the FTO gene. It is thought to have the most significant effect on BMI within the European population, yet it increases the BMI by only 0.26–0.66 units [77].

Given genetics may play a role in developing obesity in life, it would seem apparent that this risk is conferred by both the mother and father. Studies have found that paternal weight and diet can influence the likelihood of their offspring developing obesity and metabolic upset. This would seem a reasonable hypothesis given that obesity in humans affects the sperm production, increasing the likelihood of deoxyribonucleic acid (DNA) damage within the sperm [78]. Despite the possible risk the father might exert on his offspring in developing obesity and metabolic upset, the association between the birthweight of the offspring and risk of obesity is much stronger for the mother [79,80].

Sibling/sibling studies have been used in an attempt to separate out intrauterine events from shared environmental and genetic factors, and a recent study has shown an independent influence of maternal obesity and weight gain during pregnancy on offspring obesity, particularly among obese women [59]. Furthermore, the results from a study that compared body weights of children from sequential pregnancies, with mothers who underwent surgical intervention to combat obesity, showed that children born before the surgery were significantly heavier than those born after the surgery. This difference was noted at 12 and 21–25 years [81].

Childhood diabetes and cardiovascular disease

Childhood obesity leads to a range of metabolic disorders including type 2 diabetes mellitus (T2DM). There has been a surge of T2DM in childhood and adolescents in relation to obesity. In 1982, there were approximately 5% of adolescents diagnosed with of new onset T2DM, which subsequently increased to an estimated 45% in 1999 [82]. Moreover, there is growing evidence that obesity in childhood and adolescence is related not only to T2DM but also to a related wider spectrum of risk factors such as insulin resistance, glucose intolerance, hypertension and a dyslipidaemia characterised by high triglyceride and low high-density lipoprotein (HDL)-cholesterol [51,83–85]. Vulnerable children and adolescents therefore are thought to be at risk not only of developing T2DM and its sequelae but also to early stages of cardiovascular disease at much younger ages [86,87]. A few studies have demonstrated this link. A study of PIMA Indian children exposed to maternal diabetes and obesity showed that irrespective of birthweight, they were at an increased risk of developing T2DM [88] and hypertension [89]. Another study prospectively followed a cohort of children, aged from 6 to 11 years, who were born either large for gestational age (LGA) or average birthweight to mothers with and without gestational diabetes. These children had anthropometric measurements of blood pressure,
height and weight taken at 6, 7, 9 and 11 years. Biochemical blood testing, including plasma glucose levels, insulin and lipids levels, was taken at various intervals in the children from 6 to 11 years. The authors concluded that the offspring born LGA to diabetic mothers were at significant risk (50%) of developing features of the metabolic syndrome, with 15% meeting the official criteria. Furthermore, obesity amongst the 11-year-old children was a strong predictor of insulin resistance and a larger birthweight and diabetic mother may increase this risk. Lastly, an interesting finding was that the exposure of children to maternal obesity was as strong a predictor for developing the metabolic syndrome as was the LGA status [51].

In view of the potential association between childhood obesity with cardiovascular disease, a recent cohort analysis set out to investigate the relationship between maternal obesity in pregnancy and premature mortality from cardiovascular events in the adult offspring population. Birth records of 37,709 people within a stable northern Scottish population were used and linked to the General Register of Deaths, Scottish Morbidity record systems, Information and Services Division and NHS Scotland databases. The cause of death of the offspring and all admissions to a hospital for cardiovascular events were recorded. The study found there was a significant increase in all causes of mortality and in premature mortality in the offspring of overweight and obese mothers. The leading cause of death was cardiovascular, occurring in 24% of men and 13% of females, followed by cancer-related deaths. There were also significant associations between increased maternal BMI and cerebrovascular disease and peripheral artery disease in the offspring [52]. Whilst other factors, such as lifestyle and genetics, clearly contribute to adulthood obesity and cardiovascular death, these results highlight the danger of the growing epidemic of childhood obesity.

**Long-term cognitive function**

In addition to childhood obesity and its associated metabolic sequelae, maternal pre-pregnancy obesity has been implicated in lower childhood cognitive scores (intelligence quotient (IQ)) of the offspring. Animal studies have shown that the offspring of obese mothers, fed a high-fat diet, have marked elevations in pro-inflammatory cytokines within the brain [90] and morphological changes in the hippocampal neurons with shorter and decreased number of dendrites [91]. Cognitive skills, however, are difficult to accurately evaluate due to a variety of factors that influence them, with many potential confounders making comparisons between studies difficult. One recent study by Tanda et al. found that amongst healthy primary-aged school children, who were born at term, maternal pre-pregnancy obesity was associated with lower cognitive test scores by 2–3 points. Adjustments were made for confounding factors including intrauterine factors, family background and maternal and child factors. The estimate was repeated in this study, the second time excluding children who had any health condition that could affect school attendance, and found similar results [92]. By contrast, two European pregnancy cohorts — Avon Longitudinal Study of Parents and Children (ALSPAC) (UK) and Generation R (Netherlands) — found little consistent evidence of intrauterine effects of maternal pre-pregnancy overweight on offspring cognitive development and behavioural problems after adjustment of potential confounders [93]. Further studies with alternative methods to minimise the effects of confounding are required to clarify whether an adverse intrauterine environment such as obese pregnancy has a long-term effect on offspring cognitive function.

**Summary**

Maternal obesity creates a metabolically challenging environment for the developing foetus to grow and develop. The effects on the offspring are potentially multiple with both short- and long-term adverse effects. There is some evidence for programming mechanisms but even if most of the associations with long-term risk in the offspring were largely due to adverse lifestyles, there is a need to reverse obesity trends in women of reproductive age. If we cannot do so, we may be faced with more obesity, type 2 diabetes and premature death related to cardiovascular disease in the offspring with a potential exponential impact on future generations. Given that the prevalence of obesity is rising worldwide and the cost of treating it is soaring, educating young women and perhaps also pregnant
women in early pregnancy of the dangers of obesity is essential, in the hope of tackling the vicious cycle obesity is creating. Further trials in this area are also urgently needed.

**Practice Points**

- Pre-pregnancy counselling is important to optimise maternal health and hopefully to improve pre-pregnancy BMI.
- Women should be aware of the implications of obesity on the short and long-term health of their children.
- Until further evidence is available, obese women should aim to minimise weight gain to within limits set out by ISOM.
- Women of normal BMI should also be encouraged to limit weight gain in pregnancy, as excessive weight gain, irrespective of BMI, is positively associated with birthweight and adiposity.
- Breastfeeding infants of obese women should be actively supported and encouraged to limit the impact of maternal obesity on infant and childhood health.

**Research Agenda**

- Whether lifestyle or dietary intervention can influence the impact of maternal obesity on short and long-term health of the offspring including gestational diabetes, neonatal macrosomia and childhood obesity.
- A better prediction of adverse outcomes in obese pregnancies in order to streamline/target maternity care and potential intervention at those most at risk.
- Whether formula feeding ingredients can be optimised in such a way that imposes a lower risk of obesity on the neonate.
- National surveys dedicated to investigating the current understanding and beliefs of the actual health implications of maternal obesity and excessive weight gain in pregnancy that could help guide more effective interventional strategies.
- More studies are needed to evaluate further the possible effects of maternal BMI on offspring IQ.

**Conflict of interests**

The authors report no conflicts of interest.

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