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PAPER

Maternal obesity and pregnancy outcome: a study of 287 213 pregnancies in London†

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OBJECTIVE: To examine the maternal and foetal risks of adverse pregnancy outcome in relation to maternal obesity, expressed as body mass index (BMI, kg/m^2) in a large unselected geographical population.
DESIGN: Retrospective analysis of data from a validated maternity database system which includes all but one of the maternity units in the North West Thames Region. A comparison of pregnancy outcomes was made on the basis of maternal BMI at booking.
SUBJECTS: A total of 287 213 completed singleton pregnancies were studied including 176 923 (61.6%) normal weight (BMI 20–24.9), 79 014 (27.5%) moderately obese (BMI 25–29.9) and 31 276 (10.9%) very obese (BMI $\geq$ 30) women.
MEASUREMENTS: Ante-natal complications, intervention in labour, maternal morbidity and neonatal outcome were examined and data presented as raw frequencies and adjusted odds ratios with 99% confidence intervals following logistic regression analysis to account for confounding variables.
RESULTS: Compared to women with normal BMI, the following outcomes were significantly more common in obese pregnant women (odds ratio (99% confidence interval) for BMI 25–30 and BMI $\geq$ 30 respectively): gestational diabetes mellitus (1.68 (1.53–1.84), 3.6 (3.25–3.98)); proteinuric pre-eclampsia (1.44 (1.28–1.62), 2.14 (1.85–2.47)); induction of labour (2.14 (1.85–2.47), 1.70 (1.64–1.76)); delivery by emergency caesarian section (1.30 (1.25–1.34), 1.83 (1.74–1.93)); postpartum haemorrhage (1.16 (1.12–1.21), 1.39 (1.32–1.46)); genital tract infection (1.24 (1.09–1.41), 1.30 (1.07–1.56)); urinary tract infection (1.17 (1.04–1.33), 1.39 (1.18–1.63)); wound infection (1.27 (1.09–1.48), 2.24 (1.91–2.64)); birthweight above the 90th centile (1.57 (1.50–1.64), 2.36 (2.23–2.50)), and intrauterine death (1.10 (0.94–1.28), 1.40 (1.14–1.71)). However, delivery before 32 weeks' gestation (0.73 (0.65–0.82), 0.81 (0.69–0.95)) and breastfeeding at discharge (0.86 (0.84–0.88), 0.58 (0.56–0.60)) were significantly less likely in the overweight groups. In all cases, increasing maternal BMI was associated with increased magnitude of risk.
CONCLUSION: Maternal obesity carries significant risks for the mother and foetus. The risk increases with the degree of obesity and persists after accounting for other confounding demographic factors. The basis of many of the complications is likely to be related to the altered metabolic state associated with morbid obesity.
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Keywords: obesity; pregnancy; stillbirth

Introduction

Obesity is a significant health problem in the Western World with up to 40% of women in the UK being overweight.[1] Several studies have reported that maternal obesity is associated with increased risk of adverse pregnancy outcomes including gestational diabetes, infectious morbidity, postpartum haemorrhage, delivery of large-for-dates babies and, more recently, stillbirth.[2–12] However, many of the previous studies have lacked accurate quantification of such risks. This study develops other work especially regarding antenatal complications of pregnancy. The aim of this study was to examine pregnancy outcome in obese women compared to those of normal weight by reviewing a large number of singleton pregnancies using a validated database.[13] We

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aimed to test the hypothesis that obesity, as determined by maternal body mass index (BMI), is associated with adverse outcomes for mother and baby, and to quantify this risk after allowing for possible confounding factors.

Materials and methods

Data were derived from the St Mary's Maternity Information System database (SMMIS), including data from 1989 to 1997 inclusive. This is a clinical database recording maternity information from NHS hospitals within the geographical boundaries of the North West Thames Region and contains data on more than 80% of all deliveries in a region with a population of more than three and a half million. The database has been previously validated for commonly recorded variables including intrauterine death.[13]

BMI was calculated as weight at antenatal booking (kg), divided by height (m) squared. The subjects were divided into groups according to maternal BMI at booking using body mass groupings recommended by Abrams and Parker[14] (normal, BMI 20– < 25; moderately obese, BMI 25– < 30, and very obese, BMI > 30). In this study, women with BMI < 20 were considered underweight and were excluded from further analysis ($n = 38\ 182$).

The raw frequencies of the various outcomes of pregnancy in the maternal BMI groups were calculated and multiple logistic regression models were then constructed to examine the magnitude and significance of the independent effect of BMI. For each outcome variable, BMI (normal, moderately obese, very obese), maternal age, ethnic group, parity, history of hypertension and history of diabetes mellitus were included in all the models. All variables were categorical in the analytic models used. In addition, different factors of specific importance for each particular outcome were included as appropriate (see tables). The results are presented as frequencies by BMI group and odds ratios with 99% confidence intervals after adjustment for possible confounding factors. In order to correct for the influence of gestational age and foetal sex, birthweight was expressed as a delta value (the number of standard deviations by which the observed birthweight differed from the expected mean for sex and gestation). SAS Version 6.12 statistical analysis software was used. For the purposes of this study, pre-eclampsia was defined and documented as new onset hypertension and proteinuria, since the measurement of blood pressure alone may over represent the true prevalence, particularly if an inappropriate sized blood pressure cuff is used in obese subjects.

Results

Information on maternal BMI was available for 325 395 completed singleton pregnancies including 79 014 (24.3%) moderately obese and 31 276 (9.6%) very obese. The distribution of

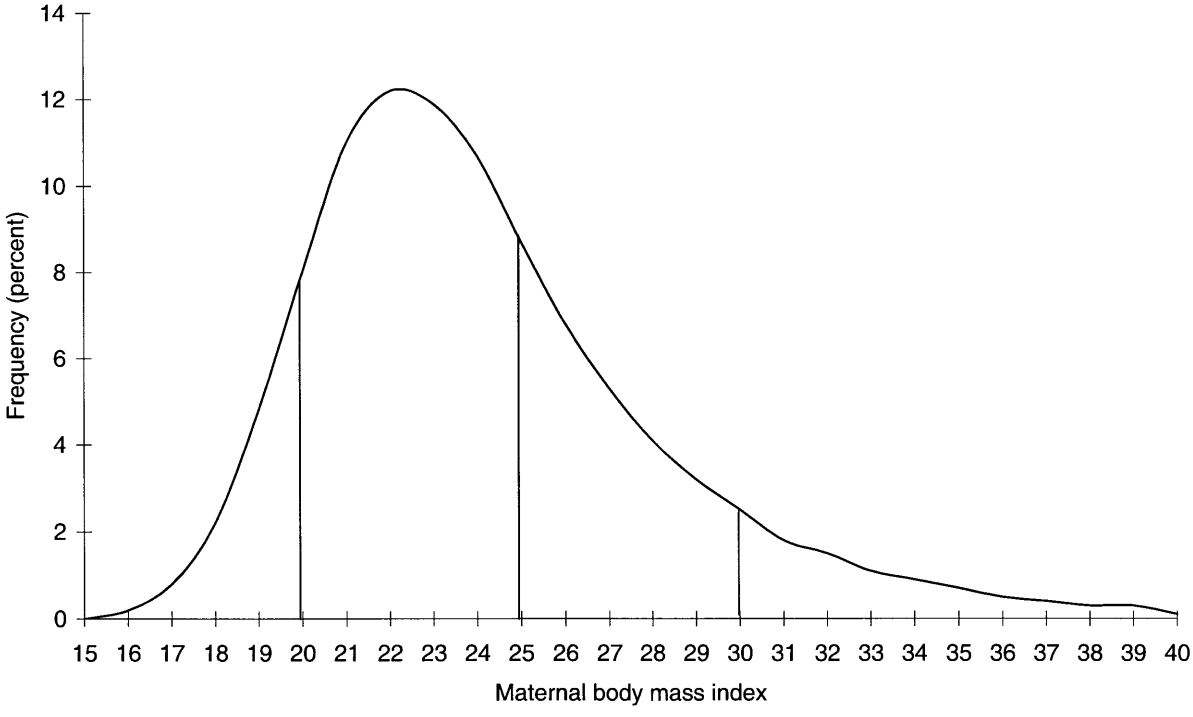


Figure 1 Frequency distribution of maternal body mass index at booking in 325 395 completed singleton pregnancies in North Thames (London).

Table 1 Demographic characteristics of women with normal and increased body mass index (BMI) expressed as frequency (%). Definitions of ethnic group are English Indian (from Indian sub-continent), Black (WI, Afro-Caribbean), Oriental (Far East Asian), Caucasian (Europid), Black (African; from African sub-continent), Mediterranean (South Mediterranean and Middle Eastern)

	BMI 20–24.9 (n = 176 923)	*BMI 25–29.9 (n = 79 014)*	*BMI 30+ (n = 31 276)*
Indian	12.2%	12.6%	10.2%
Black (WI)	2.3%	3.3%	4.4%
Oriental	1.6%	0.9%	0.4%
Caucasian	73.3%	70.3%	71.4%
Black (African)	2.2%	3.7%	4.2%
Mediterranean	1.7%	2.0%	2.1%
Other	3.5%	3.2%	4.0%
Para 0	47.2%	40.2%	33.5%
Para 1	33.8%	34.0%	34.6%
Para 2+	19.0%	25.8%	31.9%
Age (y ± s.d.)	28.3 ± 5.2	28.7 ± 5.3	28.8 ± 5.2
Diabetes mellitus	0.3%	0.6%	0.8%
Chronic hypertension	3.8%	6.6%	12.5%

BMI at booking is shown in Figure 1. Demographic characteristics of the maternal BMI groups are shown in Table 1.

The pregnancy outcome results by BMI group are summarised in Tables 2–5, with the proportion (%) of each risk factor or outcome being presented for each of the three maternal BMI groups. Odds ratios have been adjusted for the factors listed at the foot of the tables.

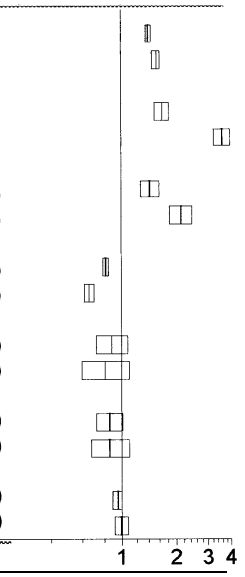
A history of diabetes mellitus and hypertension before the index pregnancy were more frequent in both the moderately and very raised BMI groups compared with women of normal BMI (Table 1). The odds of development of gestational diabetes and preeclampsia were increased in those with a raised BMI, even after allowing for demographic characteristics and histories of the above conditions (Table 2).

Induction of labour and delivery by caesarean section were both more common in obese women (Table 3). The frequency of both elective and emergency caesarean section was almost twice as high for very obese women as it was for women of normal BMI, which may have in part been related to the increased rate of induction of labour. The risk of postpartum haemorrhage rose with increasing BMI, and was about 30% more frequent in women with a moderately raised BMI and about 70% more frequent for women with very raised BMI compared with women of normal BMI. Although the assessment of postpartum haemorrhage is notoriously difficult the same increase in risk was demonstrated for blood loss of >1000 ml which would be considered clinically significant. All infectious morbidities were more prevalent in overweight women (Table 4).

Delta birthweight was positively associated with increasing maternal BMI at all gestational ages and the mean birthweight was significantly increased for the moderately

Table 2 Antenatal complications

	BMI Group (kg/m^2)	Proportion (%)	OR (99% CI)	Graphical representation of previous column
Late booking [2,8,9]	20-25	7.78		
	25-30	12.93	1.41 (1.37-1.46)	
	>30	13.54	1.56 (1.48-1.63)	
Gestational diabetes	20-25	0.75		
	25-30	1.70	1.68 (153-1.84)	
	>30	3.50	3.6 (3.25-3.98)	
Pre-eclampsia [2,6,10]	20-25	0.70		
	25-30	0.97	1.44 (1.28-1.62)	
	>30	1.43	2.14 (1.85-2.47)	
Anaemia [2,6,10]	20-25	10.27		
	25-30	9.20	0.82 (0.79-0.85)	
	>30	7.68	0.66 (0.62-0.70)	
Placenta praevia [2,6]	20-25	0.29		
	25-30	0.29	0.88 (0.72-1.08)	
	>30	0.28	0.81 (0.60-1.10)	
Placental abruption [2,3,6]	20-25	0.43		
	25-30	0.40	0.86 (0.72-1.01)	
	>30	0.43	0.86 (0.68-1.10)	
Breech presentation [2,6,9]	20-25	3.73		
	25-30	3.51	0.95 (0.89-1.00)	
	>30	3.71	0.99 (0.91-1.08)	



The proportion (percent) and the reslts of logistic regression analyses (odds ratio (99% confidence interval)), are tabulated for each variable according to BMI. (BMI 20–25 $n = 176\,923$, BMI 25–30 $n = 79\,014$, BMI > 30 $n = 31276$). All logistic regression analyses include ethnic group, parity, age and history of hypertension as confounding factors. Additional confounding factors are indicated by the superscript numbers (1 = abruption, 2 = gestational diabetes, 3 = pre-clampsia, 4 = placenta praevia, 5 = breech presentation, 6 = pre-existing diabetes, 7 = elective caesarean section, 8 = emergency caesarean section, 9 = delivery before 37 weeks gestation, 10 = smoking, 11 = induction of labour).

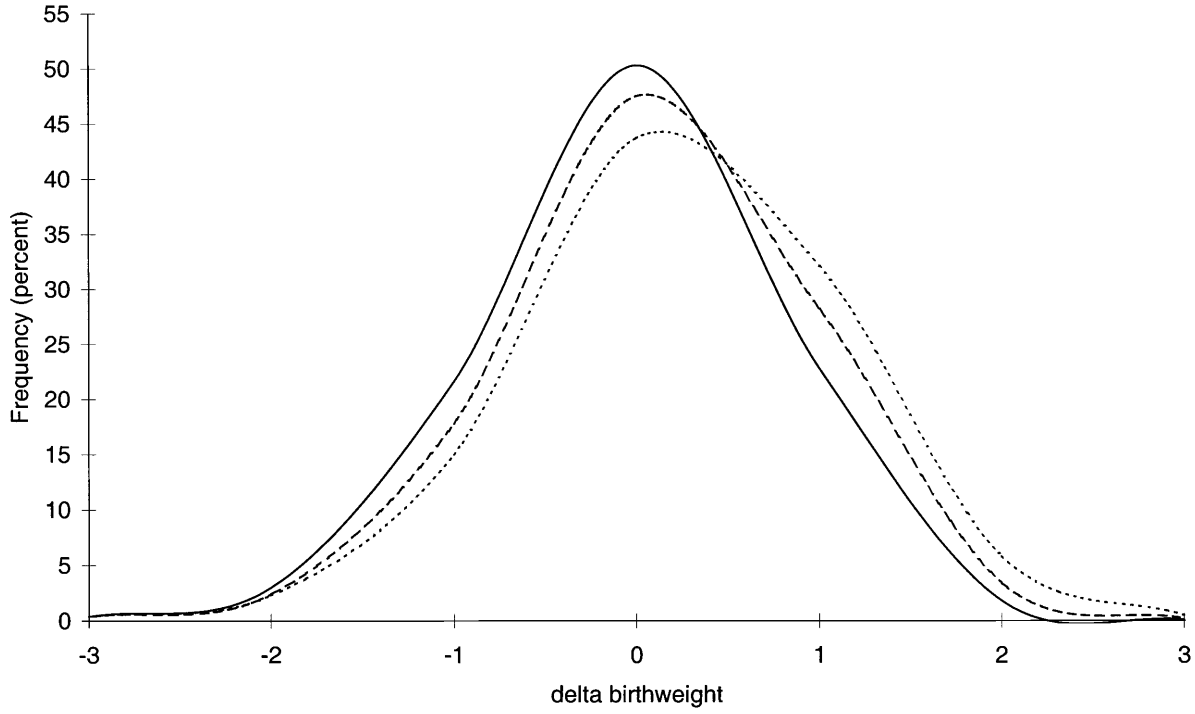


Figure 2 Birthweight distribution of infants by maternal body mass index (BMI) of the mother expressed as delta values (number of standard deviations by which the value differs from the expected mean for gestational age and foetal gender), showing the curve is shifted to the right with increasing BMI (BMI 20–24.9 (solid), BMI 25–29.9 (dashed) and BMI $\geq$ 30 (dotted)).

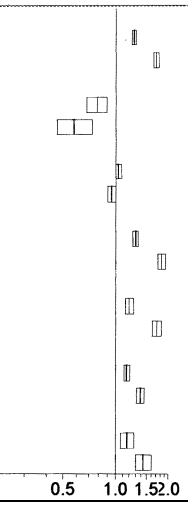
Table 3 Maternal complications

	BMI Group (kg/m^2)	Proportion (%)	OR (99% CI)	Graphical representation of previous column
Chest infection [2,6,7,8,10]	20-25	0.13		
	25-30	0.16	1.07 (0.81-1.41)	
	>30	0.28	1.34 (0.99-1.92)	
Genital tract Infection [2,6,7,8]	20-25	0.66		
	25-30	0.73	1.24 (1.09-1.41)	
	>30	0.76	1.30 (1.07-1.56)	
Wound infection [2,6,7,8]	20-25	0.39		
	25-30	0.59	1.27 (1.09-1.48)	
	>30	1.34	2.24 (1.91-2.64)	
Urinary tract infection [2,6,7,8]	20-25	0.69		
	25-30	0.84	1.17 (1.04-1.33)	
	>30	1.10	1.39 (1.18-1.63)	
Pyrexia of unknown origin [2,6,7,8]	20-25	1.002		
	25-30	1.29	1.19 (1.08-1.32)	
	>30	1.54	1.29 (1.13-1.48)	
Pulmonary embolism [2,6,7,8,10]	20-25	0.04		
	25-30	0.07	1.41 (0.91-2.19)	
	>30	0.08	1.48 (0.82-2.69)	
Prolonged post-natal stay [2,6,7,8,9]	20-25	20.35		
	25-30	21.08	1.00 (0.97-1.04)	
	>30	22.86	1.48 (0.82-2.69)	

The proportion (percent) and the results of logistic regression analyses (odds ratio (99% confidence interval)), are tabulated for each variable according to BMI. (BMI 20–25 n = 176 923, BMI 25–30 n = 79 014, BMI > 30 n = 31 276). All logistic regression analyses include ethnic group, parity, age and history of hypertension as confounding factors. Additional confounding factors are indicated by the superscript numbers (1 = abruption, 2 = gestational diabetes, 3 = pre-eclampsia, 4 = placenta praevia, 5 = breech presentation, 6 = pre-existing diabetes, 7 = elective caesarean section, 8 = emergency caesarean section, 9 = delivery before 37 weeks gestation, 10 = smoking, 11 = induction of labour).

Table 4 Fetal complications

	BMI Group (kg/m^2)	Proportion (%)	OR (99% CI)	Graphical representation of previous column
Induction of labour [2,3,8]	20-25	15.26		
	25-30	19.24	1.27 (1.23-1.30)	
	>30	24.65	1.70 (1.64-1.76)	
Breech delivery [2,9]	20-25	0.83		
	25-30	0.61	0.78 (0.68-0.89)	
	>30	0.46	0.58 (0.46-0.73)	
Operative vaginal delivery [2,6,12]	20-25	11.88		
	25-30	11.03	1.04 (1.00-1.08)	
	>30	9.08	0.95 (0.90-1.00)	
Emergency caesarean section [1,2,3,4,5,6]	20-25	7.83		
	25-30	10.25	1.30 (1.25-1.34)	
	>30	13.40	1.83 (1.74-1.93)	
Elective caesarean section [1,2,3,4,5,6]	20-25	4.05		
	25-30	5.64	1.20 (1.14-1.26)	
	>30	8.48	1.72 (1.62-1.83)	
Post partum haemorrhage [2,6,7,8]	20-25	10.38		
	25-30	13.19	1.16 (1.12-1.21)	
	>30	17.07	1.39 (1.32-1.46)	
Major Post partum haemorrhage [2,6,7,8]	20-25	1.35		
	25-30	1.83	1.17 (1.07-1.27)	
	>30	2.53	1.44 (1.30-1.60)	



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and very obese groups compared to the normal BMI group ($t=39.2$, $P<0.0001$ and $t=53.1$, $P<0.0001$ respectively, Figure 2). Similarly, the prevalence of large for gestational age babies (birthweight > 90th centile) was almost twice as high in the very obese compared to the normal group (Table 5), whereas obese women were at reduced risk of delivering a small for gestational age baby even after accounting for pregnancy complications such as pre-eclampsia. Table 5 also shows that the risk of stillbirth is increased in women with raised BMI and is significantly increased in those women with the highest BMI. The odds ratios adjusted for additional confounding variables reveal that raised BMI in itself conveys an increased risk of stillbirth.

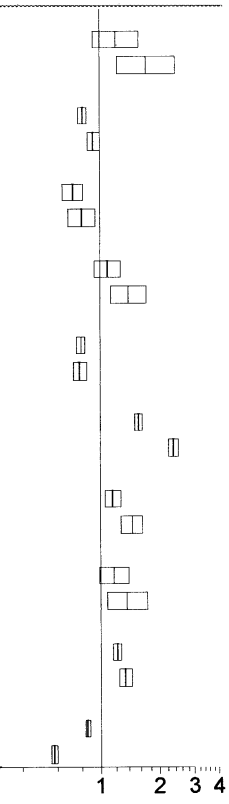
Discussion

This study has demonstrated that many adverse outcomes of pregnancy are associated with maternal obesity and has provided quantification of these risks. We have confirmed an increase in the previously reported complications of pregnancy in obese women such as gestational diabetes, induction of labour and wound infection. The risk of pre-eclampsia is positively associated with a raised BMI. This is an important observation because Reports on Confidential Enquiries into Maternal Death in the United Kingdom[15] show that hypertensive disease remains among the commonest causes of maternal death and has shown no great improvement over the last 20 y. Changes in thromboembolic disease associated with obesity were not significant; even in this large study there were not many events. The indications for thromboemblolic preventative therapy vary across the region and with time through the study period.

The increase in caesarean section rate among obese women persists even allowing for confounding variables such as abnormal presentation of the foetus and medical complications of pregnancy. The caesarean section rate for the very obese group was over 20% compared with nearer 10% for the normal-weight group in this series. The relevance of the raised caesarean section rate in this group is considerable because of their increased risk of associated complications, such as infectious morbidity. The causes of this increased operative delivery rate cannot be determined but include increased planned elective caesarean section for predicted macrosomia, maternal request or obstetrician request. The increase in emergency caesarean sections may in part have been a consequence of the increased rate of large for gestational age infants leading to disproportion during labour or it is possible that uterine contractility may be suboptimal in a subgroup of obese women, or there may be increased fat deposition in the soft tissues of the pelvis. The increased risk of postpartum haemorrhage in obese women, even after accounting for such predisposing factors as caesarean section may be explained by more bleeding from the

Table 5 Fetal complications

	BMI Group (kg/m^2)	Proportion (%)	OR (99% CI)	Graphical representation of previous column
Delivery after 42 weeks gestation [2,6]	20-25	0.13		
	25-30	0.17	1.21 (0.93-1.58)	
	>30	0.23	1.72 (1.23-2.42)	
Delivery before 37 weeks gestation [2,3,6,10]	20-25	5.55		
	25-30	5.33	0.82 (0.78-0.86)	
	>30	6.44	0.93 (0.87-1.00)	
Delivery before 32 weeks gestation [2,3,6,10]	20-25	0.79		
	25-30	0.86	0.73 (0.65-0.82)	
	>30	1.06	0.81 (0.69-0.95)	
Stillbirth [2,3,6,10]	20-25	0.4		
	25-30	0.53	1.10 (0.94-1.28)	
	>30	0.69	1.40 (1.14-1.71)	
Birthweight < 5th centile [2,3,6,10]	20-25	5.45		
	25-30	4.58	0.80 (0.76-0.84)	
	>30	4.76	0.79 (0.73-0.86)	
Birthweight > 90th centile [2,6]	20-25	9.03		
	25-30	13.41	1.57 (1.50-1.64)	
	>30	17.46	2.36 (2.23-2.50)	
Low APGAR score [1,2,6,9,10]	20-25	1.22		
	25-30	1.39	1.16 (1.06-1.28)	
	>30	1.82	1.45 (1.28-1.64)	
Very low APGAR score [1,2,6,9,10]	20-25	0.21		
	25-30	0.23	1.17 (0.99-1.40)	
	>30	0.27	1.36 (1.08-1.73)	
Admission to SCBU >24 hours [1,2,6,9,10]	20-25	5.14		
	25-30	5.52	1.22 (1.16-1.28)	
	>30	6.48	1.34 (1.25-1.44)	
Breast feeding [2,6]	20-25	3.73		
	25-30	3.51	0.86 (0.84-0.88)	
	>30	3.71	0.58 (0.56-0.60)	



The proportion (percent) and the results of logistic regression analyses (odds ratio (99% confidence interval)), are tabulated for each variable according to BMI. (BMI 20–25 $n=176\,923$, BMI 25–30 $n=79\,014$, BMI > 30 $n=31\,276$). All logistic regression analyses include ethnic group, parity, age and history of hypertension as confounding factors. Additional confounding factors are indicated by the superscript numbers (1 = abruption, 2 = gestational diabetes, 3 = pre-eclampsia, 4 = placenta praevia, 5 = breech presentation, 6 = pre-existing diabetes, 7 = elective caesarean section, 8 = emergency caesarean section, 9 = delivery before 37 weeks gestation, 10 = smoking, 11 = induction of labour).

relatively larger area of implantation of the placenta usually associated with a large for gestational age foetus.

Foetal macrosomia is more common in the obese non-diabetic mother compared to the lean mother with gestational diabetes.[16] The original Pedersen hypothesis[17] suggested that increased glucose concentrations in the diabetic mother led to foetal hyperglycaemia and hyperinsulinaemia causing increased foetal growth. Obesity is associated with maternal insulin resistance and foetal hyperinsulinaemia even in the absence of maternal diabetes.[18] Insulin resistant individuals have higher fasting plasma triglyceride levels and greater leucine turnover.[19,20] Amino acids are insulin secretagogues and an increased flux on amino acids could stimulate foetal hyperinsulinaemia. Triglycerides are energy rich and placental lipases can cleave triglyceride and transfer free fatty acids to the foetus.[21] The combination of an increased energy flux to the foetus and foetal hyperinsulinaemia may explain the increased frequency of large for gestational age infants seen in the obese non-diabetic women in this study.

Our study has also shown a small but significant increase in foetal death related to a raised maternal BMI, having made allowance for medical complications. The combination of rapid foetal growth induced by the endogenous hyperinsulinaemia in obese women and the functional limitations of the placenta to transfer sufficient oxygen to meet the requirements of the foetus, may lead to hypoxia and death in some cases. Unfortunately, because of known inconsistency in the recording of precise neonatal data, it has not been possible to determine whether there is also an increase in neonatal morbidity, but Lucas *et al*[22] have reported that the relative risk of neonatal death is greater in preterm infants born to obese mothers than to thin women, and suggested that this may be secondary to the altered metabolic milieu in obesity reducing the infant's ability to adapt

to postnatal life. Obese women in this study had a reduced risk of anaemia and placental abruption. The reason for the increased susceptibility to infectious disease is not described by these epidemiological data and may be a topic for further research.

Our findings are in agreement with, or have developed, previous studies. A study of 10 000 women in 1985 related ante-partum complications to varying degrees of maternal obesity.[5] The authors found a significant ($P < 0.05$) increase in hypertension, diabetes, thrombophlebitis and urinary tract infection. The largest study to date, from Sweden, investigated 167 750 women.[11] Pre-pregnancy weight was related to risk of adverse pregnancy outcomes. The authors found higher maternal weight before pregnancy was associated with increased risk of late foetal death although it protected against the delivery of small gestational age infant. The Swedish workers had better outcome data in terms of foetal death but less information on antenatal complications as we have demonstrated in this study.

There are potential criticisms of this study. First, the proportion of women who booked after 20 weeks gestation was only about 8% for the normal group and about 13% and 14% respectively for the moderately and very obese groups. Since BMI was calculated using weight at booking, and weight increases with advancing pregnancy, late bookers may potentially have artificially contributed to an increase in the numbers in the raised BMI groups. This effect is however likely to be of small magnitude and, if present, would actually tend to reduce the effects attributed to obesity. Indeed, the significance of results is not altered if the analyses are repeated after adjusting for gestation at booking. Another major study[11] used maternal memory of pre-pregnancy weight; we aimed to use a simple measure applicable in the ante-natal clinic to predict adverse pregnancy outcome. Our study did not include weight gain; as in other studies many women did not have weight measured twice, therefore weight gain may bias results. BMI is a better indicator of body composition than weight alone, being a more sensitive indicator of obesity in shorter women, more predictive of gestational diabetes and no less predictive than weight alone for any other outcomes.[23]

No information on social class was available for this study, however social class and obesity are related.[24] Some of the outcome variables studied here also have an association with social class, particularly low birth weight,[25] and breastfeeding.[26] It is noteworthy that in this instance, raised maternal BMI has been shown to be protective against low birth weight, but the complex relationship between social class and pregnancy outcome in conjunction with a raised BMI cannot be determined from this study. None of the centres in this study have a practice where obese women receive more diagnostic scrutiny or receive differential diagnostic practice. Whilst this is a possibility, we do not believe it has altered these results.

Greater understanding is needed of the pathophysiological link between obesity and the various adverse outcomes of pregnancy we have described before effective and safe management strategies can be devised. At present one can only advise that it would be sensible to attempt to achieve nearer normal weight before conception.

Acknowledgements

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