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ORIGINAL ARTICLE

Long-term consequences of maternal overweight in pregnancy on offspring later health: Findings from the Helsinki Birth Cohort Study

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Introduction. **Obesity has reached epidemic proportions worldwide. Maternal obesity has consequences for the offspring's later health. Only few studies have focused upon the long-term consequences of maternal obesity on the offspring's later health.**
Methods. **A total of 13,345 men and women born in Helsinki during 1934–44 belonging to the Helsinki Birth Cohort Study were included in the study. Data on maternal weight and height in late pregnancy were available from hospital records. Using validated national registers we report on the following outcomes in relation to maternal BMI: death, cancer, coronary heart disease, stroke, and diabetes among the offspring.**
Results. **Maternal BMI was positively associated with each of the later health outcomes of the offspring. The associations were strongest for cardiovascular disease and type 2 diabetes. The association with type 2 diabetes was stronger in women.**
Discussion. **Our findings stress the importance of early prevention of overweight and obesity in women of child-bearing age.**

Key words: Cancer, cardiovascular disease, maternal obesity, offspring health, type 2 diabetes

Key messages

- Maternal BMI is positively associated with several adverse health outcomes in offspring in adult life.
- Higher maternal BMI is associated with an increased risk of cancer, cardiovascular disease, and type 2 diabetes among the offspring.
- Early prevention of overweight and obesity in women of child-bearing age is of major importance for the offspring's later health.

Introduction

Obesity has reached epidemic proportions in the developed world and is also a large health concern in the developing world. Within the European Union about one-third of women of reproductive age are overweight, and every fifth is obese (1–4). Maternal obesity is known to increase the risk of congenital defects and miscarriage (5–7). However, there is increasing evidence suggesting that maternal obesity also has long-term consequences for the offspring's later health (8,9). One plausible explanation for an association between maternal obesity, which provides adverse intrauterine experiences, and later health is *in utero* programming, which may work through environmental, genetic, and epigenetic mechanisms (10).

Early-life programming has traditionally been studied in relation to long-term health consequences of being born with a small body size (11–15). However, body size at birth is only a marker of intrauterine conditions, the long-term effects of which can in fact be seen without any influence on body size at birth (16,17). Many studies have shown associations between maternal obesity and obesity in the offspring (18,19). Fewer have focused upon other long-term health consequences of maternal obesity, mainly because there are not many cohorts with the necessary data. We have previously shown increased death rates from coronary heart disease in men who were thin at birth and whose mothers had a high body mass index (BMI) during pregnancy (20). In a recent study from Scotland maternal obesity in pregnancy was associated with an increased risk of premature death in adult offspring (9). Here we study associations between maternal pregnancy BMI and all-cause mortality, cancer mortality and incidence, cardiovascular morbidity and mortality, and diabetes in men and women from the Helsinki Birth Cohort Study (HBCS) born in 1934–44.

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Subjects and methods

The epidemiological part of the HBCS includes 13,345 men and women born as singletons in one of the two maternity hospitals in Helsinki during 1934–44, who also attended child welfare clinics in the city. Their birth and child welfare clinic records have been described in detail previously (21,22). The birth records included the mother's height and weight prior to delivery. BMI was calculated as weight (kg) divided by height^2 (m^2). The weight and length of the baby were recorded, and we calculated the ponderal index (birth weight/length^3).

Using the unique personal identification number assigned to each Finnish citizen, we identified hospital admissions and deaths from coronary heart disease and stroke among the men and women in the cohort between the years 1971 to 2010. We used International Classification of Diseases (ICD) codes 410-4 in ICD-8 and ICD-9 and I21-5 in ICD-10 to define coronary heart disease; we used 430-9 in ICD-8 and I60-9 in ICD-10 to define stroke. Cardiovascular disease was defined as coronary heart disease and stroke combined. All hospital admissions in Finland are recorded in the national hospital discharge register. All-cause mortality data were obtained from the national mortality register. We ascertained the occurrence of diabetes using the social insurance institution's register of people receiving medication for chronic diseases. In Finland, the costs of anti-diabetic drugs are partly reimbursed by the state subject to the approval of a physician who reviews each case history. Cancer incidence data were obtained from the Finnish cancer register until end of the year 2006. We assessed childhood socio-economic status from the occupation of the father, as recorded in the maternity, child welfare, and school records. Through Statistics Finland we obtained data on education, occupation, and taxable household income recorded in censuses.

The Ethics Committee at the National Public Health Institute and the Coordinating Ethics Committee of Helsinki and Uusimaa Hospital District approved the study.

Statistical methods

In all analyses we controlled for gender, year of birth, socio-economic status in childhood and adult life, educational attainment, and income.

Cardiovascular disease, coronary heart disease (CHD), and stroke: We analyzed the data using the Cox proportional hazards model, stratifying on year of birth, and also on sex in models in which men and women were analyzed together. Subjects were

Table I. Characteristics of the study cohort including maternal characteristics, neonatal measurements, adult socio-economic measures, and disease outcomes separately for men and women.

		Males ($n = 6975$)	Females ($n = 6370$)	Maternal BMI
Maternal measurements:				Correlation
Height (cm)	Mean (SD)	159.8 (5.7)	160.0 (5.6)	−0.115
Weight (kg)	Mean (SD)	67.0 (8.2)	67.0 (8.4)	0.819
BMI (kg/m^2)	Mean (SD)	26.2 (2.9)	26.2 (2.9)	1.000
Age (years)	Mean (SD)	28.3 (5.4)	28.5 (5.5)	0.221
Parity	Mean (SD)	1.9 (1.3)	2.0 (1.4)	0.152
Neonatal measurements:				
Length (cm)	Mean (SD)	50.6 (1.9)	49.9 (1.8)	0.188
Weight (g)	Mean (SD)	3468 (488)	3340 (460)	0.249
Gestational age (days)	Mean (SD)	279 (13)	280 (13)	0.099
Placental weight (g)	Mean (SD)	651 (120)	639 (119)	0.182
Socio-economic status				Mean
Upper middle	% (n)	17 (1211)	16 (1019)	25.9
Lower middle	% (n)	23 (1632)	24 (1513)	26.2
Laborer	% (n)	57 (3971)	57 (3649)	26.3
Unknown	% (n)	2 (161)	3 (189)	25.8
Adult measurements:				
Socio-economic status				
High official	% (n)	42 (2935)	36 (2318)	26.2
Low official	% (n)	23 (1610)	45 (2846)	26.2
Self-employed	% (n)	6 (403)	3 (161)	26.4
Laborer	% (n)	21 (1483)	6 (374)	26.3
Unknown	% (n)	8 (544)	11 (671)	26.1
Education				
Basic	% (n)	37 (2572)	42 (2683)	26.2
Upper secondary	% (n)	24 (1703)	22 (1371)	26.2
Lower tertiary	% (n)	21 (1464)	20 (1289)	26.2
Upper tertiary	% (n)	12 (834)	7 (473)	26.1
Unknown	% (n)	6 (402)	9 (554)	26.2
Income				
Lowest fifth	% (n)	14 (1006)	20 (1297)	26.2
Second fifth	% (n)	18 (1222)	16 (1023)	26.2
Third fifth	% (n)	18 (1279)	15 (964)	26.2
Fourth fifth	% (n)	18 (1259)	17 (1091)	26.1
Highest fifth	% (n)	17 (1171)	17 (1084)	26.3
Unknown	% (n)	15 (1038)	14 (911)	26.2
Disease outcomes:				
Death	% (n)	23 (1570)	10 (658)	
Cancer death	% (n)	5 (337)	4 (256)	
Cancer incidence	% (n)	11 (734)	12 (771)	
Coronary heart disease	% (n)	15 (1064)	5 (319)	
Stroke	% (n)	8 (554)	5 (320)	
Cardiovascular disease	% (n)	21 (1483)	9 (591)	
Diabetes	% (n)	7 (512)	4 (249)	

followed until the first of these events: death, migration from Finland, hospitalization for the condition, or reaching 1 January 2011 alive.

Cancer: As above, but subjects were followed until the first of these events: death, migration from Finland, cancer registration, or reaching 1 January 2007 alive. We also used the ICD code for the underlying cause to analyze deaths from cancer.

All causes: As above, but subjects were followed until the first of these events: death, migration from Finland, or reaching 1 January 2011 alive.

Diabetes: We analyzed the data using multiple logistic regression, controlling for sex and year of birth. Cases were those who received reimbursement for diabetes medication.

We analyzed the mother's BMI in late pregnancy as a predictor in two ways: first, as a continuous measure; second, in four groups of roughly equal size, designed to check for non-linearities in the association of mother's BMI with outcomes. The cut-points were 24, 26, and 28 kg/m^2.

The analyses were conducted in IBM SPSS Statistics version 21.

Results

Table I gives descriptive data for the cohort and shows how maternal BMI is correlated with other maternal and offspring measurements (all P values < 0.001).

Tables II (both sexes combined), III (males), and IV (females) show the cumulative incidence of the seven conditions we studied (all-cause mortality, cancer death, cancer incidence, CHD, stroke, cardiovascular disease, and diabetes) in the four groups according to maternal BMI. These data are also expressed as risk ratios relative to the group with the smallest maternal BMI, and as trends in risk per kg/m^2 of maternal BMI. These risks are shown in summary form in Figure 1. All risk estimates are adjusted for childhood socio-economic status, and adult socio-economic status, income, and education, each of which was strongly associated with the conditions we studied.

In both males and females all disease outcomes studied among the offspring were positively associated with maternal BMI. However, given the large number of cases, these were relatively modest effects, often only just statistically significant at the 5% level. The most statistically significant effect in males and females combined was with cardiovascular disease. Diabetes was more strongly associated with the mother's BMI in women than in men (P for interaction = 0.04). Further adjustment for birth weight produced little change in the risk estimates.

Discussion

Using data from Helsinki Birth Cohort Study (HBCS) we have studied long-term health outcomes in relation to maternal BMI during late pregnancy. HBCS is one of the largest birth cohorts still being followed up with register-based long-term health outcomes available. Maternal BMI was moderately positively associated with each of the studied seven later health outcomes in the offspring. These associations were independent of socio-economic measures obtained in childhood and adult life. They were strongest for cardiovascular disease and type 2 diabetes. The association with type 2 diabetes seemed stronger in women. Our findings stress the importance of early prevention of overweight and obesity in women of child-bearing age.

The underlying mechanisms explaining these findings are not fully recognized, but the over-nutrition hypothesis suggests that high maternal glucose, insulin, and free fatty acids—often a consequence of overweight and obesity—can result in permanent changes in the developing fetus, increasing its vulnerability to later obesity-related health outcomes (23,24). There are also other possible environmental, genetic, and epigenetic mechanisms (25).

The association between a non-optimal early environment and disease in later life is well documented. Many studies have shown associations between a small body size at birth and increased risk of non-communicable diseases, including CHD and type 2 diabetes (11–16). Within HBCS we have shown that high maternal BMI added to the effect of a small body size at birth on death rates from coronary heart disease. The highest standardized mortality ratio was seen in men who had the lowest ponderal index at birth and the highest maternal BMI. The lowest mortality

Table II. Cumulative incidence and risk of all-cause mortality, cancer death and incidence, cardiovascular disease, CHD, stroke, and diabetes according to mother's body mass index. Results for males and females combined.

	Mother's body mass index (kg/m^2)				
Outcome	≤24	to 26	to 28	>28	Trend per kg/m^2
All causes					
CI (%)	15.4	17.1	16.5	17.6	
HR	1.0 (baseline)	1.13	1.12	1.12	1.012 (0.997 to 1.028), $P = 0.1$
Cancer death					
CI (%)	4.1	4.5	4.1	5.1	
HR	1.0 (baseline)	1.12	1.01	1.22	1.013 (0.983 to 1.044), $P = 0.4$
Cancer incidence					
CI (%)	10.4	11.1	11.9	12.1	
HR	1.0 (baseline)	1.11	1.19	1.17	1.017 (0.998 to 1.036), $P = 0.08$
Cardiovascular disease					
CI (%)	14.4	15.7	15.8	16.5	
HR	1.0 (baseline)	1.10	1.13	1.13	1.026 (1.010 to 1.042), $P = 0.002$
Coronary heart disease					
CI (%)	9.7	10.1	10.8	11.1	
HR	1.0 (baseline)	1.03	1.13	1.13	1.030 (1.010 to 1.050), $P = 0.003$
Stroke					
CI (%)	5.7	7.1	6.7	6.8	
HR	1.0 (baseline)	1.28	1.22	1.18	1.026 (1.002 to 1.051), $P = 0.04$
Diabetes					
CI (%)	5.7	5.4	5.5	6.7	
OR	1.0 (baseline)	0.94	0.97	1.20	1.040 (1.013 to 1.068), $P = 0.004$

Hazard ratios include adjustment for mother's age and parity and for childhood and adult socio-economic status, education, and income.
CI = cumulative incidence; HR = hazard ratio; OR = odds ratio.

Table III. Cumulative incidence and risk of all-cause mortality, cancer death and incidence, cardiovascular disease, CHD, stroke, and diabetes according to mother's body mass index. Results for males.

	Mother's body mass index (kg/m^2)				
Outcome	≤ 24	to 26	to 28	> 28	Trend per kg/m^2
All causes					
CI (%)	21.5	22.8	21.9	23.5	
HR	1.0 (baseline)	1.10	1.08	1.10	1.010 (0.991 to 1.028), $P = 0.3$
Cancer death					
CI (%)	4.5	5.2	3.8	5.9	
HR	1.0 (baseline)	1.23	0.88	1.36	1.017 (0.978 to 1.058), $P = 0.4$
Cancer incidence					
CI (%)	9.3	10.3	11.1	12.0	
HR	1.0 (baseline)	1.15	1.23	1.30	1.025 (0.999 to 1.053), $P = 0.06$
Cardiovascular disease					
CI (%)	19.7	21.5	22.2	21.9	
HR	1.0 (baseline)	1.13	1.17	1.13	1.022 (1.003 to 1.041), $P = 0.03$
Coronary heart disease					
CI (%)	14.1	14.8	16.0	16.2	
HR	1.0 (baseline)	1.08	1.18	1.17	1.031 (1.009 to 1.054), $P = 0.006$
Stroke					
CI (%)	7.0	8.8	8.2	7.5	
HR	1.0 (baseline)	1.30	1.21	1.06	1.005 (0.974 to 1.037), $P = 0.8$
Diabetes					
CI (%)	8.0	7.0	6.9	8.0	
OR	1.0 (baseline)	0.89	0.86	1.02	1.015 (0.981 to 1.050), $P = 0.4$

Hazard ratios include adjustment for mother's age and parity and for childhood and adult socio-economic status, education, and income.
CI = cumulative incidence; HR = hazard ratio; OR = odds ratio.

ratio was seen in men with the highest ponderal index and the lowest maternal BMI. In a multivariate analysis the effects of ponderal index and mother's BMI on the risk of coronary heart disease were both strongly significant (21). The findings in our present study were similar but based upon a much larger study population, including females and other health outcomes. The findings in relation to CHD were statistically significant in males only, probably due to fewer cases of CHD among the females. The opposite was true for stroke. Higher maternal BMI during pregnancy was statistically significantly associated with stroke, but in females only. Similar findings were seen for maternal BMI and risk of type 2 diabetes. Our findings are consistent with the transmission of type 2 diabetes from the mother to her daughters being stronger than transmission to her sons.

Reynolds et al. recently showed that maternal obesity in pregnancy was associated with an increased risk of premature death in adult offspring, the hazard ratio being 1.35 (9). Most mothers in our study cohort were not obese by today's standard, and therefore we did not focus upon the obese group but upon the trend with BMI. Nevertheless we still observed a positive association between maternal BMI and offspring health decades later. These findings suggest that early preventive measure among girls and women could have far-reaching benefit in preventing non-communicable diseases in the next generation.

Table IV. Cumulative incidence and risk of all-cause mortality, cancer death and incidence, cardiovascular disease, CHD, stroke, and diabetes according to mother's body mass index. Results for females.

	Mother's body mass index (kg/m^2)				
Outcome	≤ 24	to 26	to 28	> 28	Trend per kg/m^2
All causes					
CI (%)	9.3	10.6	10.6	11.1	
HR	1.0 (baseline)	1.17	1.23	1.18	1.020 (0.992 to 1.049), $P = 0.2$
Cancer death					
CI (%)	3.8	3.6	4.5	4.2	
HR	1.0 (baseline)	0.98	1.17	1.04	1.007 (0.963 to 1.054), $P = 0.8$
Cancer incidence					
CI (%)	11.5	12.0	12.7	12.2	
HR	1.0 (baseline)	1.09	1.16	1.05	1.007 (0.981 to 1.034), $P = 0.6$
Cardiovascular disease					
CI (%)	9.2	9.1	8.8	10.4	
HR	1.0 (baseline)	1.04	1.03	1.15	1.035 (1.005 to 1.066), $P = 0.02$
Coronary heart disease					
CI (%)	5.3	4.7	5.1	5.3	
HR	1.0 (baseline)	0.91	1.03	1.01	1.025 (0.984 to 1.067), $P = 0.2$
Stroke					
CI (%)	4.3	5.2	5.0	6.0	
HR	1.0 (baseline)	1.26	1.22	1.42	1.059 (1.019 to 1.101), $P = 0.003$
Diabetes					
CI (%)	3.4	3.5	4.0	5.3	
OR	1.0 (baseline)	1.06	1.20	1.60	1.082 (1.036 to 1.130), $P < 0.001$

Hazard ratios include adjustment for mother's age and parity and for childhood and adult socio-economic status, education and income.
CI = cumulative incidence; HR = hazard ratio; OR = odds ratio.

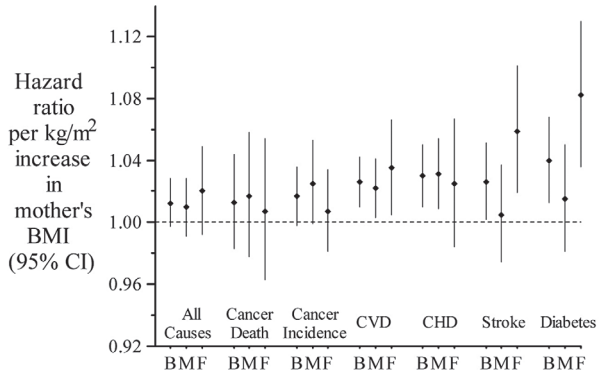

Figure 1. Hazard ratio per 1 kg/m^2 increase in maternal BMI for all-cause mortality, cancer mortality, cardiovascular disease, CHD, stroke, and type 2 diabetes incidence. B = both sexes; M = males; F = females.

There are limitations to our study. The HBCS is restricted to people who were both born and attended child-welfare clinics in the city of Helsinki. Most children and their parents attended these clinics, which were free, but attendance was voluntary. So the people in our study may not be representative of all people living in Helsinki. But at birth the distribution of fathers' occupations was similar to that in the city as a whole. The register data we used have been validated and found suitable for epidemiological studies. As expected, fewer women in the study than men had had CHD and cardiovascular events. So the statistical power of our observations on women is lower, but nevertheless each outcome is based on hundreds of events. Diabetes diagnosis was based upon use of medication, and therefore we cannot distinguish between type 1 and type 2 diabetes. Further people on diet treatment only will not be included. Mother's BMI reflects both weight gain during pregnancy and BMI before pregnancy. Weight gain in pregnancy results from an increase in fat, uterine, and breast tissue, and extracellular fluid, as well as from growth of the fetus and placenta. However, BMI in late pregnancy is highly correlated with BMI before pregnancy (26). Some of the children were born during World War II, and this could have impact on both maternal and child well-being and health.

The strengths of our study include the long follow-up period, and the reliable measurements of maternal body size and birth size obtained from hospital records. The outcomes were based on validated national registers and included both data on morbidity and mortality.

In conclusion, we have shown that increased maternal BMI in late pregnancy is an independent predictor of cardiovascular disease and type 2 diabetes among the offspring. Our study emphasizes the importance of maternal influences during pregnancy on later health. Strategies to raise awareness of the risks of overweight and obesity among women of child-bearing age are required.

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Declaration of interest: The authors report no conflicts of interest.

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