Original Contribution

High Maternal Body Mass Index in Early Pregnancy and Risks of Stillbirth and Infant Mortality—A Population-Based Sibling Study in Sweden

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In a population-based case-control study, we investigated whether familial confounding influenced the associations between maternal overweight/obesity and risks of stillbirth and infant mortality by including both population and sister controls. Using nationwide data from the Swedish Medical Birth Register (1992–2011), we included all primiparous women with singleton births who also had a sister with a first birth during that time period. We used logistic regression analyses to calculate odds ratios (and 95% confidence intervals) adjusted for maternal age, height, smoking habits, education, and time period (5-year groups) of child's birth. Body mass index (BMI) was calculated as weight (kg)/height (m)^2. Compared with population controls with a normal BMI (18.5–24.9), stillbirth risk increased with increasing BMI (BMI 25–29.9: odds ratio (OR) = 1.51 (95% confidence interval (CI): 1.21, 1.89); BMI 30–34.9: OR = 1.77 (95% CI: 1.24, 2.50); BMI ≥35: OR = 3.16 (95% CI: 2.10, 4.76)). The sister case-control analyses revealed similar results. Offspring of obese women (BMI ≥30) had an increased risk of infant mortality when population controls were used (OR = 2.41, 95% CI: 1.83, 3.16), and an even higher risk was obtained when sister controls were used (OR = 4.04, 95% CI: 2.25, 7.25). We conclude that obesity in early pregnancy is associated with increased risks of stillbirth and infant mortality independently of genetic and early environmental risk factors shared within families.

body mass index; familial confounding; infant mortality; neonatal mortality; postneonatal mortality; sibling-design studies; stillbirth

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

Editor's note: An invited commentary on this article appears on page 106.

The body mass index (BMI; weight (kg)/height (m)^2) of the general population has increased worldwide during recent decades, and in 2008 there were an estimated 300 million obese (BMI ≥30) women in the world (1). In Sweden, the prevalences of overweight (BMI 25–<30) and obesity (BMI ≥30) in early pregnancy (usually the first trimester) increased from 20% and 6%, respectively, in 1992 to 25% and 13%, respectively, in 2013 (2). In the United States in 2011–2012, 53% of gravid women were reported to be overweight or obese in early pregnancy (3).

Maternal obesity is associated with increased risks of gestational diabetes, large-for-gestational-age birth, preterm birth (<37 weeks' gestation), and congenital malformations (4–8). Risks of stillbirth and infant mortality also increase with maternal overweight and obesity (9–13). However, the possible influence of genetic or early environmental factors has not been accounted for in previous studies.

Twin and family studies show that heritability plays a substantial role in determining BMI (14). Genetic factors may also influence risks of stillbirth and infant mortality, and there is a recurrence risk of stillbirth in successive pregnancies (15). Fetal growth restriction is one of the main causes of stillbirth (16), and there is a strong familial aggregation of small-for-gestational-age births (17, 18). Preterm birth is a major cause of infant mortality (9) and has been shown to be clustered in families (19, 20). Quantitative genetic analyses show that maternal genetic factors are of importance for preterm birth (21). Congenital anomalies are commonly seen in both

stillbirths and infant mortality (22, 23) and may be caused by genetic factors (24).

Full sisters have approximately 50% of their genomes in common and are usually brought up together. Thus, results from a study of risks related to maternal BMI within sister pairs discordant for the outcome would be controlled for familial factors (i.e., shared genetic and early environmental factors) by design. In this population-based study, we investigated associations between BMI in early pregnancy and risks of stillbirth and infant mortality using 2 reference groups: population controls and sister controls.

METHODS

Data sources

In this study, data from nationwide Swedish registries were linked using the individually unique personal registration number assigned to all Swedish residents (25). The Swedish Medical Birth Register was established in 1973 and includes information collected prospectively during pregnancy, delivery, and the neonatal period (26). Information on early-pregnancy BMI has been included in the Register since 1992. The Cause of Death Register, which provides information on dates and causes of death in Sweden, was used to identify cases of infant mortality (27). Information about the mother's highest achieved educational level was retrieved from the Swedish Register of Education (28). The Multigeneration Register includes data on all subjects born in 1932 and later (index persons) and their first-degree relatives (parents, full and half siblings, and children) and was used to identify full sisters of mothers included in the Medical Birth Register (29).

Study population

From 1992 through 2011, there were 2,038,185 births recorded in the Swedish Medical Birth Register. Because parity influences both BMI and rates of stillbirth and infant mortality (9, 30, 31), we included only births to primiparous mothers ($n = 871,330$). We excluded births to women without a primiparous sister ($n = 495,669$), births to women not born in Sweden (as their full sisters were less likely to be identified in the Multigeneration Register ($n = 148,766$)), and twin births (singletons and twins differ with respect to risk factors and rates of stillbirth and infant mortality ($n = 23,227$)). Consequently, the study population was restricted to 203,668 singleton births to primiparous women who also had a primiparous sister with a singleton birth between the beginning of 1992 and the end of 2011 (Figure 1).

Outcomes

Stillbirth was defined as fetal death occurring at or after 28 completed weeks of gestation. In 2008, the definition of stillbirth in the Medical Birth Register was changed from 28 weeks or later to 22 weeks or later, and we decided to use the same stillbirth definition throughout the study period (i.e., intrauterine deaths occurring at ≥28 weeks). Therefore, in the analysis of stillbirth, we excluded 726 births (stillbirths and live births) that took place at 22–27 weeks' gestation. Infant mortality was defined as the death of a liveborn infant during the first year of life. Analyses of infant mortality were based on all live births occurring from 22 completed weeks' gestation onward; 563 stillbirths were excluded, of which 36 occurred at 22–27 weeks' gestation (Figure 1). Neonatal mortality was defined as infant mortality occurring within the first 27 completed
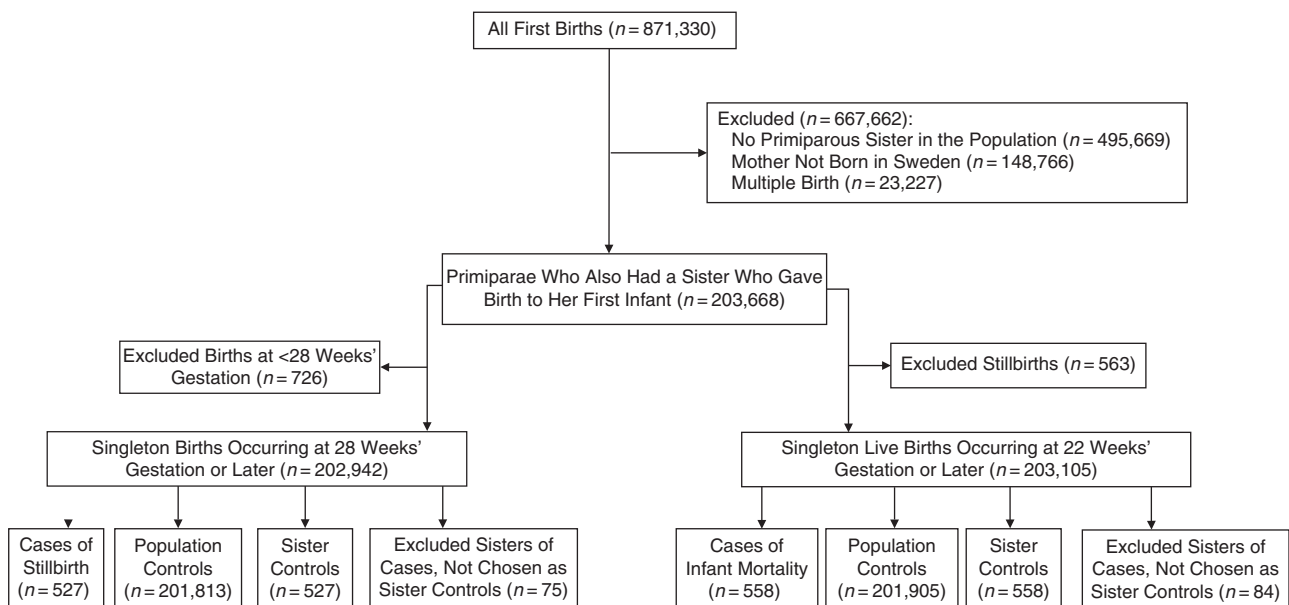

Figure 1. Selection of participants for a study of associations between early-pregnancy body mass index and risks of stillbirth and infant mortality using 2 reference groups: population controls and sister controls. The study population included all singleton first births taking place in Sweden during 1992–2011 to mothers born in Sweden who had a sister who also gave birth to her first child during that time period.

days after birth, and postneonatal mortality was defined as infant mortality occurring 28–364 completed days after birth.

Study design

We selected 2 control groups: population controls and sister controls, both having their first singleton birth during the study period. If a case mother had more than 1 suitable sister control, the sister who gave birth closest in time to the case mother was selected as the control (Figure 1). For the stillbirth case-control study, we identified 527 cases of stillbirth, 201,813 population controls, and 527 sister controls with live births at 28 weeks' gestation or later (Figure 1). For the case-control analyses of infant mortality, we identified 558 cases of infant mortality, 201,905 population controls, and 558 sister controls for which infants in both control groups survived the first year of life. Of the 558 cases of infant mortality, there were 390 neonatal deaths and 168 postneonatal deaths.

Exposures

Information on maternal height and weight was collected at the first antenatal visit, which occurs within the first trimester for more than 90% of Swedish women (26). Height was self-reported, while weight was measured by the midwife. BMI was calculated as weight in kilograms divided by height in meters squared. The World Health Organization's classification was used to categorize BMI as underweight (BMI <18.5), normal-weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), obese grade 1 (BMI 30–34.9), or obese grade 2–3 (BMI ≥35) (32). Self-reported information on smoking in early pregnancy was also collected at the first antenatal visit. Information on maternal age at delivery was calculated from the mother's birth date (included in the personal registration number) and the date of delivery. Information on the mother's highest level of education attained (by December 31, 2011) was retrieved from the Education Register. Maternal age, height, smoking, and educational level and period of child's birth (in 5-year groups) were categorized as presented in Table 1.

The study protocol was approved by the Research Ethics Committee at Karolinska Institutet (Stockholm, Sweden).

Statistical analyses

In order to control for familial confounding (i.e., genetic and family environmental factors), we compared results from case-control analyses using population controls and sister controls. If an association is seen using population controls but not when using sister controls, the association is confounded by familial factors. We used unconditional logistic regression for the population case-control analyses of associations between maternal BMI and risks of stillbirth and infant, neonatal, and postneonatal mortality. The dependency in the data (due to the fact that all mothers had a sister in the population) was handled with a generalized estimating equations model. In the analyses of case-control sister pairs, we used conditional logistic regression. Odds ratios and 95% confidence intervals were calculated, and results were adjusted for maternal age, height, smoking, education, and time period (5-year groups) of child's birth. In the analyses of infant mortality,

Table 1. Distribution of Maternal Characteristics for Cases of Stillbirth, Population Controls, and Sister Controls Among Singleton Births in Sweden, 1992–2011

| | Stillbirth Cases | | Controls | | | |
| | | | Population | | Sisters | |
	No.	%	No.	%	No.	%
Total	527	100.0	201,813	100.0	527	100.0
Maternal body mass index[a]						
<18.5	14	2.7	4,675	2.3	15	2.9
18.5–24.9	244	46.3	120,962	59.9	308	58.4
25–29.9	120	22.8	37,398	18.5	102	19.4
30–34.9	38	7.2	10,198	5.1	24	4.6
≥35	26	4.9	3,832	1.9	13	2.5
Missing data	85	16.3	24,748	12.3	65	12.3
Maternal age, years						
≤24	143	27.1	52,569	26.1	153	29.0
25–29	188	35.7	80,862	40.1	195	37.0
30–34	137	26.0	53,081	26.3	139	26.4
≥35	59	11.2	15,301	7.6	40	7.6
Maternal height, cm						
≤154	14	2.7	2,712	1.3	6	1.1
155–164	171	32.5	59,112	29.3	173	32.8
165–174	246	46.7	105,518	52.3	262	49.7
≥175	46	8.7	21,326	10.6	56	10.6
Missing data	50	9.5	13,145	6.5	30	5.7
Smoking in early pregnancy						
Yes	71	13.5	18,939	9.4	66	12.5
No	414	78.6	172,818	85.6	441	83.7
Missing data	42	8.0	10,056	5.0	20	3.8
Education, years						
≤9	42	7.9	10,944	5.4	44	8.4
10–11	88	16.7	28,448	14.1	87	16.5
12	155	29.4	56,414	28.0	137	26.0
13–14	77	14.9	30,129	14.9	89	16.9
≥15	161	30.6	74,994	37.2	165	31.3
Missing data	4	0.8	884	0.4	5	1.0
Time period of child's birth						
1992–1996	118	22.4	46,089	22.8	133	25.2
1997–2001	145	27.5	47,283	23.4	117	22.2
2002–2006	149	28.3	56,757	28.1	159	30.2
2007–2011	115	21.8	51,684	25.6	118	22.4

[a] Weight (kg)/height (m)^2.

there were too few sister controls in the obesity grade 2–3 category (BMI ≥35) for analysis (*n* = 8); therefore, all women with obesity (BMI ≥30) were collapsed into 1 category. In analyses of neonatal and postneonatal mortality, underweight mothers were excluded because of small numbers
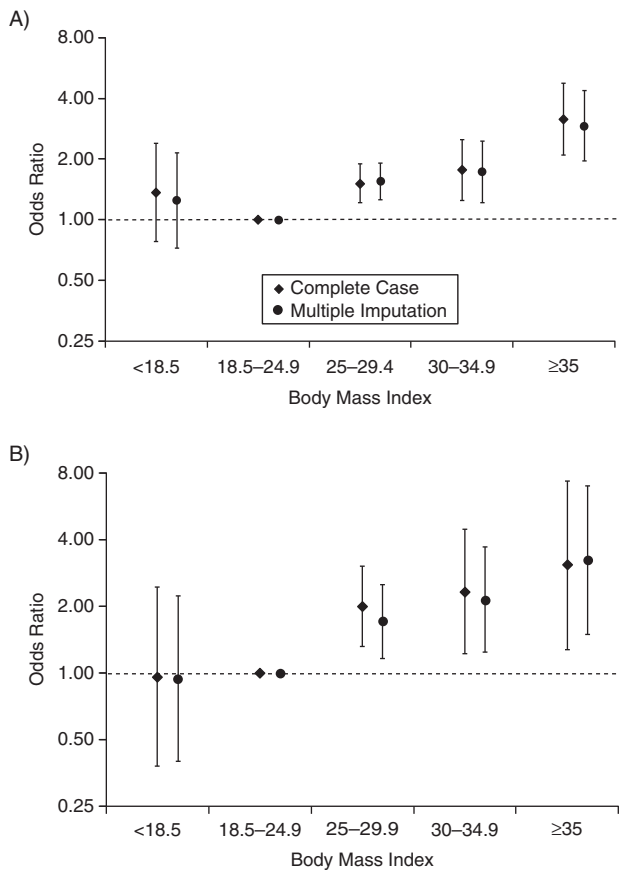
Figure 2. Risk of stillbirth in singleton first births according to early-pregnancy body mass index (weight (kg)/height (m)^2) among mothers born in Sweden who gave birth during 1992–2011, derived using population controls (A) and sister controls (B). Odds ratios were estimated using complete covariate information ($n = 174{,}199$ in the population control analysis and $n = 889$ in the sister control analysis) and after multiple imputation of missing values. Odds ratios were adjusted for maternal age, maternal height, smoking, education, and time period (5-year groups) of child's birth. Bars, 95% confidence intervals.
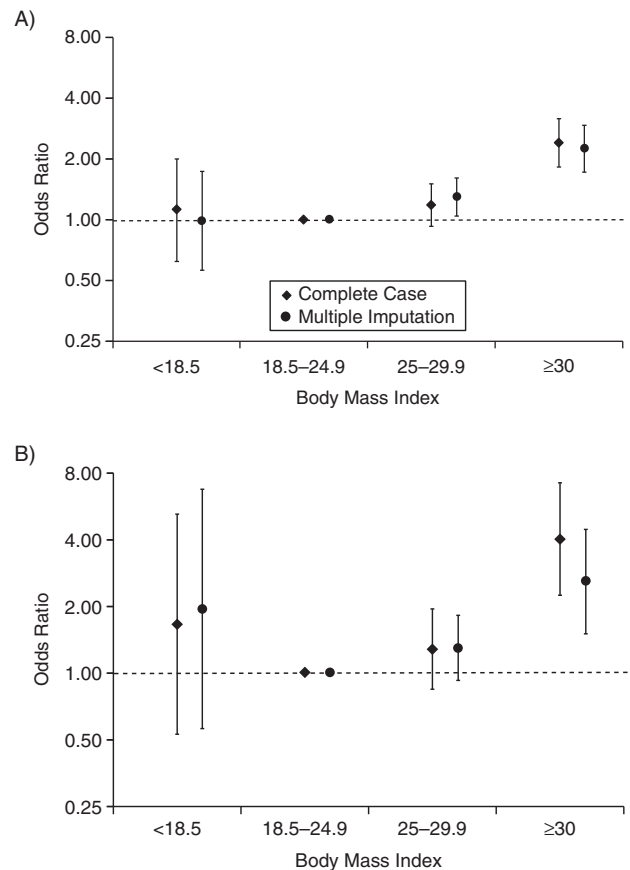

Figure 3. Risk of infant mortality in singleton first births according to early-pregnancy body mass index (weight (kg)/height (m)^2) among mothers born in Sweden who gave birth during 1992–2011, derived using population controls (A) and sister controls (B). Odds ratios were estimated using complete covariate information ($n = 174{,}141$ in the population control analysis and $n = 917$ in the sister control analysis) and after multiple imputation of missing values. Odds ratios were adjusted for maternal age, maternal height, smoking, education, and time period (5-year groups) of child's birth. Bars, 95% confidence intervals.

(7 cases of neonatal mortality and 5 cases of postneonatal mortality).

Because data on maternal BMI were missing for up to 19% of participants, we also performed analyses using multiple imputation. Missing values for BMI, maternal height, smoking, and education were imputed by means of ordinal logistic regression, using available information on maternal age, time period of child's birth, stillbirth, and/or infant mortality. Ten data sets were created and were analyzed with the same logistic regression models as in the complete-case analyses. Parameter estimates and covariance matrices were analyzed with the MIANALYZE procedure in SAS (SAS Institute, Inc., Cary, North Carolina). These estimates were used in sensitivity analyses, and the results were compared with those of the complete-case analyses (Figures 2 and 3).

We also performed trend tests with BMI included in the analyses as a continuous variable. *P* values for neonatal mortality

and postneonatal mortality are presented. SAS software, version 9.4, was used for all analyses.

RESULTS

Among the stillbirth case mothers, rates of overweight (BMI 25–29.9), obesity grade 1 (BMI 30–34.9), and obesity grade 2–3 (BMI ≥35) were higher than corresponding rates among population controls and sister controls. The stillbirth case mothers were slightly older and were more often smokers than population controls and sister controls (Table 1).

Among the infant mortality case mothers, 17.6% were overweight and 12.9% were obese. Corresponding rates were 18.5% and 6.9%, respectively, in population control mothers and 20.6% and 7.0%, respectively, among sister control mothers (Appendix Table 1). Smoking during pregnancy

Table 2. Maternal Body Mass Index in Early Pregnancy and Risks of Neonatal Mortality and Postneonatal Mortality Derived Using Both Population Controls and Sister Controls, Sweden, 1992–2011

| | Population Control Analyses | | | | Sister Control Analyses | | | |
| | Crude | | Adjusted[a] | | Crude | | Adjusted[a] | |
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
				Neonatal Mortality[b]				
Maternal BMI[c]								
18.5–24.9	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
25.0–29.9	1.23	0.93, 1.61	1.20	0.90, 1.59	1.17	0.73, 1.88	1.14	0.67, 1.96
≥30.0	2.35	1.72, 3.21	2.52	1.83, 3.47	4.00	2.05, 7.81	4.86	2.33, 10.14
P for trend	<0.0001		<0.0001		<0.0001		<0.0001	
				Postneonatal Mortality[d]				
Maternal BMI[e]								
18.5–24.9	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
25.0–29.9	1.08	0.70, 1.67	1.12	0.72, 1.75	1.53	0.80, 2.92	1.76	0.83, 3.74
≥30.0	2.24	1.38, 3.61	2.03	1.20, 3.42	2.45	1.02, 5.87	2.41	0.82, 7.09
P for trend	0.0006		0.0028		0.0068		0.0150	

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

[a] ORs were adjusted for maternal age, maternal height, smoking, education, and time period (5-year groups) of child's birth.

[b] Neonatal mortality was defined as death occurring during the first 27 completed days of life ($n = 381$ cases, $n = 197,228$ population controls, and $n = 381$ sister controls).

[c] BMI was defined as weight (kg)/height $(m)^2$. Women with a BMI below 18.5 were excluded because of very low numbers of cases and sister controls ($n = 7$ cases, $n = 2$ sister controls, and $n = 4,677$ population controls).

[d] Postneonatal mortality was defined as death occurring within the first year of life, after the first 27 completed days of life ($n = 160$ cases, $n = 197,228$ population controls, and $n = 160$ sister controls).

[e] Women with a BMI below 18.5 were excluded because of very low numbers among cases and sister controls ($n = 5$ cases, $n = 5$ sister controls, and $n = 4,677$ population controls).

was equally common among case mothers and their sisters (13%), as compared with 9.4% among population controls.

In adjusted analyses, we found positive associations between maternal BMI and stillbirth risks, both when using population controls (Figure 2A) and when using sister controls (Figure 2B). Compared with normal weight, the stillbirth risk increased with BMI in the population case-control analyses (overweight: odds ratio (OR) = 1.51 (95% confidence interval (CI): 1.21, 1.89); obesity grade 1: OR = 1.77 (95% CI: 1.24, 2.50); obesity grade 2–3: OR = 3.16 (95% CI: 2.10, 4.76)) (Figure 2A). In the sister case-control analyses, corresponding odds ratios and 95% confidence intervals were similar (overweight: OR = 2.00 (95% CI: 1.32, 3.03); obesity grade 1: OR = 2.33 (95% CI: 1.22, 4.46); obesity grade 2–3: OR = 3.08 (95% CI: 1.28, 7.40)) (Figure 2B). We found no statistically significant association between being underweight (BMI <18.5) in early pregnancy and stillbirth risk, either when using population controls (OR = 1.36, 95% CI: 0.78, 2.39) or when using sister controls (OR = 0.96, 95% CI: 0.38, 2.45). The estimates from the multiple imputation analyses were similar to those from the complete-case analyses (Figures 2A and 2B).

In analyses of BMI and infant mortality, we also found a similar risk pattern regardless of whether we used population controls (Figure 3A) or sister controls (Figure 3B) as the reference group. In the population case-control analyses, infants of obese women (BMI ≥30) had a more than doubled risk of infant death (OR = 2.41, 95% CI: 1.83, 3.16) compared with

infants of normal-weight women. Infant mortality risk was not increased among infants of overweight women (OR = 1.18, 95% CI: 0.93, 1.50) or underweight women (OR = 1.12, 95% CI: 0.62, 2.00) (Figure 3A). In the sister case-control analyses, infants of obese women had a 4-fold higher risk of infant death (OR = 4.04, 95% CI: 2.25, 7.25) than infants of normal-weight women. The odds ratios for infant mortality were 1.29 (95% CI: 0.85, 1.95) for infants of overweight women and 1.67 (95% CI: 0.53, 5.24) for infants of underweight women (Figure 3B). The estimates from the multiple imputation analyses showed results similar to those of the complete-case analyses (Figure 3A and 3B).

Finally, we estimated risks of neonatal and postneonatal mortality, respectively (Table 2). Most infant deaths occurred during the neonatal period, and the association between maternal BMI and risk of neonatal mortality was similar to that in the analyses of BMI and infant mortality. Compared with infants of normal-weight mothers, the adjusted risk of neonatal mortality was higher in infants of obese mothers, both when using population controls (OR = 2.52, 95% CI: 1.83, 3.47) and when using sister controls (OR = 4.86, 95% CI: 2.33, 10.14). Compared with infants of normal-weight mothers, the risk of postneonatal mortality was doubled among infants of obese mothers in the crude analyses. In the adjusted analyses, although point estimates were similarly elevated, a statistically increased risk was achieved only when population controls were used (Table 2).

DISCUSSION

In this large population-based study, we found that risk of stillbirth increased with overweight and increasing obesity, both when using population controls as the reference group and when using sister controls. Moreover, in the analyses of infant mortality, we found that obesity but not overweight in early pregnancy was associated with increased infant mortality risk irrespective of control group. These findings indicate that the associations between BMI and stillbirth and infant mortality are not confounded by familial factors.

Many earlier studies found an association between maternal overweight/obesity and risks of stillbirth and infant mortality (9, 10, 16, 33). In a large meta-analysis, a dose-response association between BMI and stillbirth was shown, with an increased relative risk of 24% per 5-unit increase in BMI (34). Maternal weight gain between the first and second pregnancies also increases risks of stillbirth and infant mortality in second-born offspring (31, 35). Weight loss in overweight or obese women during pregnancy seems to decrease the risk of gestational diabetes, large-for-gestational-age birth, and neonatal mortality (31, 35, 36). These studies strengthen the possibility of a causal relationship between maternal overweight/obesity and risks of stillbirth and infant mortality.

The mechanisms behind the associations between maternal BMI above the normal range and stillbirth and infant mortality are probably multifactorial. Inflammation and infection have been associated with stillbirth, and obesity may contribute to dysregulation of inflammatory responses and increase the risk of infection, leading to fetal loss (37). Adipose tissue in obese individuals mainly releases proinflammatory cytokines (e.g., tumor necrosis factor α, interleukin-6, leptin), while adipose tissue in lean individuals secretes antiinflammatory adipokines (adiponectin, transforming growth factor β, interleukin-10) (38). Histopathological investigations of placentas have shown a higher prevalence of both acute and chronic placental inflammation among stillbirths (39, 40). Genetic studies have shown an association between stillbirth and heritable thrombophilias. One example is a common association between stillbirth, fetal growth restriction, and placental abruption and 4G/4G homozygosity for the hypofibrinolytic plasminogen activator inhibitor type 1 gene (*PAI-1*) (41, 42). Proinflammatory cytokines may also be responsible for the obesity-related increased risk of spontaneous extremely preterm birth, which is a main cause of infant mortality (5, 9). Further, maternal obesity increases the risk of preeclampsia, which may increase risks of stillbirth and infant mortality through fetal growth restriction and preterm birth (43, 44). Obesity is also a risk factor for gestational diabetes, which increases risks of antepartum stillbirth (45), large-for-gestational-age birth, and neonatal infant mortality due to asphyxia (9, 46, 47). Moreover, congenital anomalies, which may cause stillbirth and infant mortality, are also more common in pregnancies of obese women (7, 9, 23).

Strengths of this study include the large population-based study base. The study base included more than 870,000 births to primiparous women, recorded in reliable health registries. The subsidized health-care system in Sweden gives all mothers equal access to antenatal and delivery care free of charge. We were able to perform linkage between sisters' data using individually unique Swedish personal registration numbers and the Multigeneration Register. We were also able to control for additional important confounding factors, such as smoking, education, and maternal age.

Limitations of this study included a lack of statistical power to use finer BMI categories in the analyses of infant mortality and neonatal and postneonatal mortality. The mothers included in our study might have had a more stable family background than the general population, as we only included full sisters who gave birth during a limited time period. However, the same inclusion criteria were applied to both sibling and population controls, and our focus was to compare associations between maternal BMI and stillbirth and infant mortality when cases were compared with either sibling or population controls, rather than generalizability. Maternal BMIs were calculated from self-assessed height and from weight measured at the first antenatal care visit. Self-assessed height tends to be overestimated (48), and differences in time of weight measurement in early pregnancy might under- or overestimate BMI in early pregnancy. However, these data were recorded prospectively and would be unlikely to lead to systematic misclassification of BMI between cases and controls. We had up to 19% missing information on BMI in our data, and the rates of missing values were higher among cases than among sister or population controls. We used multiple imputation to assess the risk of selection bias due to missing data, and found that the estimates from the multiple imputations were similar to those from the complete-case analyses (Figures 2 and 3).

Although the sibling case-control study design controls for factors shared by the siblings, including genetic and early environmental factors, it may be sensitive to bias due to nonshared confounding (49). Moreover, if the sibling pairs differ more with regard to the confounders than with regard to the exposure of interest, a spurious association due to nonshared confounding bias may occur. In our study, the largest differences between the sisters were seen in BMI and not in the other confounders (Table 1 and Appendix Table 1), so there seems little reason to assume the presence of confounding bias. However, this does not include unmeasured confounding bias.

In conclusion, we found that obesity in early pregnancy is associated with increased risks of stillbirth and infant mortality independently of genetic and early environmental risk factors shared within families.

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Appendix Table 1. Distribution of Maternal Characteristics for Cases of Infant Mortality, Population Controls, and Sister Controls Among Singleton Births in Sweden, 1992–2011

| | Infant Mortality Cases | | Controls | | | |
| | | | Population | | Sisters | |
	No.	%	No.	%	No.	%
Total	558	100.0	201,905	100	558	100
Maternal body mass index[a]						
<18.5	12	2.2	4,677	2.3	7	1.3
18.5–24.9	268	48.0	120,918	59.9	335	60.0
25–29.9	98	17.6	37,389	18.5	115	20.6
≥30	72	12.9	14,017	6.9	39	7.0
Missing data	108	19.4	24,904	12.3	62	11.1
Maternal age, years						
≤24	183	32.8	52,582	26.0	174	31.2
25–29	201	36.0	80,872	40.1	213	38.2
30–34	127	22.8	53,134	26.3	122	21.9
≥35	47	8.4	15,317	7.6	49	8.8
Maternal height, cm						
≤154	7	1.3	2,714	1.3	12	2.2
155–164	185	33.2	59,118	29.3	161	28.9
165–174	241	43.2	105,463	52.2	292	52.3
≥175	48	8.6	21,314	10.6	57	10.2
Missing data	77	13.8	13,296	6.6	36	6.5
Smoking in early pregnancy						
Yes	74	13.3	18,916	9.4	75	13.4
No	416	74.6	172,783	85.6	457	81.9
Missing data	68	12.2	10,206	5.1	26	4.7
Education, years						
≤9	48	8.6	10,928	5.4	58	10.4
10–11	106	19.0	28,472	14.1	87	15.6
12	155	27.8	56,453	28.0	152	27.2
13–14	68	12.2	30,168	14.9	83	14.9
≥15	174	31.2	75,002	37.2	173	31.0
Missing data	7	1.3	882	0.4	5	0.9
Time period of child's birth						
1992–1996	176	31.5	46,073	22.8	145	26.0
1997–2001	128	22.9	47,301	23.4	143	25.6
2002–2006	147	26.3	56,798	28.1	144	25.8
2007–2011	107	19.2	51,733	25.6	126	22.6

[a] Weight (kg)/height (m)^2.