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Pre-pregnancy maternal obesity, macrosomia, and risk of stillbirth: Apopulation-based study

Chioma A. Ikedionwu[a], Deepa Dongarwar[a,b], Korede K. Yusuf[c], Sahra Ibrahimi[c], Abraham A. Salinas-Miranda[d], Hamisu M. Salihu[a,e,*]

[a] Center of Excellence in Health Equity, Training and Research, Baylor College of Medicine, Houston, TX, USA
[b] Office of the Provost, Baylor College of Medicine, Houston, TX, USA
[c] College of Nursing & Public Health, Adelphi University, Garden City, NY, USA
[d] Harrell Center, College of Public Health, University of South Florida, Tampa, FL, USA
[e] Department of Family and Community Medicine, Baylor College of Medicine, Houston, TX, USA

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ABSTRACT

Objective: An examination of the synergistic effects of maternal obesity and macrosomia on the risk of stillbirth is lacking. The purpose of this study was to determine the association between fetal macrosomia, maternal obesity, and the risk of stillbirth.

Methods: This retrospective cross-sectional study used the CDC's Birth Data and Fetal Death Data files for 2014–2017 [n = 10,043,398 total births; including 48,799 stillbirths]. The exposure was fetal macrosomia stratified by obesity subtypes (I–III). The outcome was the risk of stillbirth. We also controlled for potential and known confounding factors in adjusted models. Adjusted Relative Risks (ARR) were estimated with log-binomial regression models.

Results: The rate of stillbirth was higher among macrosomic infants born to mothers with obesity compared to those without (6.55 vs. 0.54 per 1000 total births). After controlling for confounding, women with obesity types II and III were at increased risk for stillbirth [Obesity II ARR = 2.37 (2.07−2.72); Obesity III ARR = 9.06 (7.61−10.78)].

Conclusions for practice: Obesity-related fetal overgrowth is a significant risk factor for stillbirth, especially among women with type II and type III obesity. This finding highlights the need for more effective clinical and public health strategies to address pre-pregnancy obesity and to optimize gestational weight gain.

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Introduction

Fetal macrosomia is a well-documented complication of pregnancy occurring in approximately 9 in 100 births, both in the United States and globally [1,2]. While commonly defined as birth weight greater than 4000 g, a cut-off of 4500 g may also be observed throughout the literature [3–6]. Irrespective of this variation, a grading scale has been established to further delineate among macrosomic phenotypes. Infants weighing between 4000 g and 4499 g are defined as type I, 4500 g – 4999 g are type II, and ≥ 5000 g are designated type III [7]. As birth weight increases among macrosomic infants, so does the risk of adverse birth events [1,8]. Fetal macrosomia can lead to a variety of complications in the intrapartum and post-partum periods including the risk of operative delivery, future development of obesity, and stillbirth [8–10].

With a global rate of 2.6 million cases per year, stillbirth remains a major problem and obstetrics concern [11]. Fetal death has several consequences for parents, families, and society at large. Major risk factors include a history of stillbirth, socioeconomic factors, and maternal obesity [12–15]. Obesity has also been recognized as a major public health concern exhibiting a frequency increasing to epidemic proportions, including among the pregnant population [16–18]. While a number of factors may contribute to the development of fetal macrosomia, pre-pregnancy obesity and significant weight gain during pregnancy remain the most significant [19]. Consequently, infants born to mothers with obesity are at increased risk of shoulder dystocia, fetal asphyxia, admission to neonatal intensive care unit, and stillbirth [20–22].

The connection between maternal obesity, fetal macrosomia, and overall increased probability of adverse outcomes has been

* Corresponding author at: Center of Excellence in Health Equity, Training and Research, Baylor College of Medicine, 3701 Kirby Drive, Suite 600, Houston, TX, 77098, USA.
E-mail address: Hamisu.salihu@bcm.edu (H.M. Salihu).

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well-documented in the literature. However, few studies have examined the risk of stillbirth, specifically among macrosomic infants born to mothers with obesity. In this study, we hypothesize that fetal macrosomia associated with maternal obesity presents a higher risk of stillbirth than macrosomia among infants born to mothers who are not obese. We also hypothesize that there is a dose-response relationship between obesity severity and the risk of stillbirth among macrosomic infants.

Materials and methods

This was a cross-sectional study using the Birth Data and Fetal Death Data which were made publicly available by the Centers for Disease Control and Prevention (CDC). The data were obtained from the National Vital Statistics System (NVSS), and the National Center for Health Statistics (NCHS) is responsible for collecting and disseminating the data for vital events like births, deaths, marriages, divorces and fetal deaths. The information in the Birth Data is abstracted from the birth certificates filed in the vital statistics offices of each state in the US and contains information on all births. The Fetal Death dataset provides information regarding all fetal deaths or stillbirths, which is defined as spontaneous intrauterine death of a fetus at 20 weeks' gestation or later [23]. Both data files include information regarding maternal socio-demographic and health characteristics. However, information regarding maternal pre-pregnancy BMI was available in both data sets only for the years 2014 – 2017. Therefore, we utilized data from those years to conduct this study [n = 10,043,398].

Study variables and sample

Only the variables in both the Birth Data and the Fetal Death datasets were included in the study. These include birth weight, gestational age, plurality, mother's age, race, nativity, maternal education, pre-pregnancy BMI, birth facility, prenatal care, delivery method, and birth attendant. Given the lack of longitudinal fetal growth measures in the birth certificates for the calculation of fetal weight by gestational age percentiles prior to fetal death, we had to use the official birth weight $\geq$ 4000 g to measure fetal macrosomia [24–26]. We further identified three phenotypes of fetal macrosomia based on the recommended classification in the literature [7]: Phenotype 1 = 4000–4499 grams; Phenotype 2 = 4500 – 4999 g; and Phenotype 3 = $\geq$ 5000 g. Maternal pre-pregnancy BMI was categorized in the data set as follows: Underweight: <18.5, Normal: 18.5–24.9, Overweight: 25.0–29.9, Obesity type I: 30.0–34.9, Obesity type II: 35.0–39.9 and Obesity type III: $\geq$ 40.0 [27]. We included mothers characterized as "Normal" and those characterized as "Obese". Mother's age was categorized as: Under 20 years; 20−29 years; 30−39 years; and $\geq$40 years. Maternal race was classified as Non-Hispanic White, Non-Hispanic Black, Hispanic, or Others. Mother's nativity was categorized into "born in US" and "born outside US". Highest educational attainment was grouped as "less than high school" and "high school graduate, GED completed or higher". Birth facility included categories of "Hospital" and "others" which comprised of freestanding birth center, home, and clinic/doctor's office. Birth attendants were sub-grouped into "medical professionals" and "others". We

Table 1
Socio- demographic information comparing women with and without obesity.

	Normal BMI		Women with Obesity		Prevalence of obesity	p-value
	n = 6,386,944	% = 63.6	n = 3,656,454	% = 36.4		
Maternal Age						<0.0001
<20 years	425,465	6.7%	142,942	3.9%	25.1 %	
20−29 years	3,127,342	49.0%	1,903,549	52.1%	37.8 %	
30−39 years	2,658,700	41.6%	1,490,194	40.8 %	35.9 %	
> = 40 years	175,437	2.7%	119,769	3.3%	40.6 %	
Race						<0.0001
NH-White	3,599,652	56.4%	1,755,992	48.0%	32.8 %	
NH-Black	679,241	10.6%	703,456	19.2%	50.9 %	
Hispanic	1,305,532	20.4%	950,661	26.0%	42.1 %	
Others/Missing	802,519	12.6%	246,345	6.7%	23.5 %	
Mother's nativity						<0.0001
Born in US	4,804,181	75.2%	3,052,599	83.5%	38.9 %	
Born outside US	1,568,814	24.6%	596,761	16.3 %	27.6 %	
Missing	13,949	0.2%	7094	0.2%	33.7 %	
Education						<0.0001
Less than high school	806,857	12.6%	534,244	14.6%	39.8 %	
High school graduate, GED completed or higher	5,506,419	86.2%	3,085,203	84.4%	35.9 %	
Unknown	73,668	1.2%	37,007	1.0%	33.4 %	
Birth facility						<0.0001
Hospital	6,249,432	97.8%	3,626,017	99.2%	36.7 %	
Others	137,233	2.1%	30,324	0.8 %	18.1 %	
Unknown	279	0.0%	113	0.0%	28.8 %	
Prenatal care						<0.0001
1st trimester	4,847,782	75.9%	2,741,131	75.0%	36.1 %	
2nd or 3rd trimester	1,285,523	20.1%	782,933	21.4%	37.9 %	
No prenatal care/Missing	253,639	4.0%	132,390	3.6%	34.3 %	
Delivery method						<0.0001
Vaginal	4,813,219	75.4%	2,152,791	58.9%	30.9 %	
C-Section	1,570,738	24.6%	1,501,777	41.1%	48.9 %	
Missing	2987	0.0%	1886	0.1%	38.7 %	
Birth attendant						<0.0001
Medical professionals	6,327,971	99.1%	3,630,790	99.3%	36.5 %	
Other	55,082	0.9%	23,734	0.6%	30.1 %	
Unknown	3891	0.1%	1930	0.1%	33.2 %	

restricted the study to singletons within the gestational age of 20–42 weeks.

Statistical analysis

The study was performed using publicly available de-identified data and was approved as exempt by the Institutional Review Board of Baylor College of Medicine. All statistical analyses were performed using R (version 3·5·1) and RStudio (Version 1·1·423). Pearson's chi-squared tests were conducted to identify differences in proportions. First, we calculated the prevalence of pre-pregnancy maternal obesity by socio-demographic and birth characteristics. Next, we calculated the rate of stillbirth per 1000 total births among various socio-demographic and birth characteristics. We then calculated stillbirth rates per 1000 births among all categories of maternal BMI sub-groups stratified by fetal macrosomia and its phenotypes. We used log-binomial modeling to compute adjusted relative risks for the association between macrosomia phenotypes and stillbirths among mothers with and without obesity [28]. Utilizing the stepwise regression technique, maternal age, race, nativity, and educational qualification were then adjusted for. These covariates were chosen based on previous literature on the topic [29,30]. All tests of hypotheses were two-tailed with a type 1 error rate at 5%.

Results

The analysis included a total of 10,043,398 singleton births from the years 2014–2017. Of these, 3,656,454 mothers had obesity (36.4 %). Among them, obesity type I accounted for 55.0 %, obesity type II was 26.5 %, and obesity type III was 18.5 %. Table 1 displays the socio-demographic characteristics of mothers with obesity versus those without. The prevalence of obesity was lowest among adolescent mothers (25.1 %) and most frequent among women ≥ 40 years of age (40.6 %). The prevalence of obesity was most pronounced among Black (51 %) and Hispanic women (42 %). The frequency of obesity was significantly higher among US-born (about 39 %) compared to non-US born mothers (about 28 %). Women with less than high school education were more likely to have obesity than those with at least high school level of education. The prevalence of obesity tended to be relatively higher in established hospitals and among births attended by well-qualified medical professionals. Obese mothers were more likely to have documented prenatal care visits. Approximately half of the women who underwent cesarean delivery had obesity compared to less than one-third among those who delivered vaginally.

In the entire study sample, 48,799 counts of stillbirth were documented, equating to a rate of 5 per 1000. Table 2 outlines the differences in the rate of stillbirth based on socio-demographic features. The greatest risk for stillbirth was maternal age ≥ 40 years. For all other socio-demographic groups, mothers with missing information were by far the group with the most elevated risk for stillbirth. The most pronounced stillbirth rate was in mothers with missing mode of delivery information at approximately 270 per 1000 – more than 50 times the rate of stillbirth for the entire study sample. The lowest stillbirth rate in those with missing information was observed among mothers with missing prenatal care information at 17 per 1000 which was more than three times that of the entire study sample. Hence, missingness of socio-demographic and delivery information was a marker of stillbirth.

Table 2
Socio- demographic information comparing women experiencing live and stillbirth.

	Live Birth		Stillbirth		Risk of stillbirth per 1000 total births	p-value
	n = 9,994,599	% = 99.5	n = 48,799	% = 0.5		
Maternal Age						<0.0001
<20 years	565,079	5.7%	3328	6.8%	5.85	
20–29 years	5,007,313	50.1%	23,578	48.3%	4.69	
30–39 years	4,129,406	41.3%	19,488	39.9%	4.70	
> = 40 years	292,801	2.9%	2405	4.9%	8.15	
Race						<0.0001
NH-White	5,349,401	53.5%	6243	12.8%	1.17	
NH-Black	1,381,930	13.8%	767	1.6%	0.55	
Hispanic	2,243,126	22.4%	13,067	26.8%	5.79	
Others/Missing	1,020,142	12.6%	27,726	6.7%	26.46	
Mother's nativity						<0.0001
Born in US	7,817,997	78.2%	38,783	79.5%	4.94	
Born outside US	2,157,826	21.6%	7749	15.9%	3.58	
Missing	18,776	0.2%	2267	4.6%	107.73	
Education						<0.0001
Less than high school	1,333,573	12.6%	7528	14.6%	5.61	
High school graduate, GED completed or higher	8,554,097	86.2%	37,525	84.4%	4.37	
Unknown	106,929	1.1%	3746	7.7%	33.85	
Birth facility						<0.0001
Hospital	9,827,561	98.3%	47,888	98.1 %	4.85	
Others	166,675	2.1%	882	0.8 %	5.26	
Unknown	363	0.0%	29	0.1%	73.98	
Prenatal care						<0.0001
1st trimester	7,555,331	75.6%	33,582	68.8%	4.43	
2nd or 3rd trimester	2,059,848	20.6%	8608	17.6%	4.16	
No prenatal care/Missing	379,420	3.8%	6609	13.5%	17.12	
Delivery method						<0.0001
Vaginal	6,918,531	69.2%	47,479	97.3%	6.82	
C-Section	3,072,515	30.7%	0	0.0%	0.00	
Missing	3553	0.0%	1320	2.7%	270.88	
Birth attendant						<0.0001
Medical professionals	9,911,635	99.2%	47,126	96.6%	4.73	
Other	77,875	0.8 %	941	1.9%	11.94	
Unknown	5089	0.1%	732	1.5%	125.75	

The rate of macrosomia in the entire study sample was 8.1 %. A total of 405,022 fetuses among mothers with normal BMI were classified as macrosomic, yielding a rate of 6.3 %. In contrast, among mothers with obesity, a total of 409,916 fetuses or 11.2 % were diagnosed as macrosomic. The distribution of macrosomia phenotypes among mothers with normal BMI was: Grade 1 = 362,550 or 89.5 %; Grade 2 = 39,262 or 9.7 %; and Grade 3 = 3210 or 0.8 %. Among mothers that had obesity, the distribution of macrosomia phenotypes was as follows: Grade 1 = 338,917 or 82.7 %; Grade 2 = 61,483 or 15.0 %; and Grade 3 = 9516 or 2.3 %. Fig. 1 illustrates the distribution of stillbirth across maternal BMI categories by fetal macrosomia phenotypes. The rate of fetal macrosomia was highest among mothers with obesity and was most pronounced for Grade III fetal macrosomia for which the stillbirth rate was nearly 8-fold as high compared to that in mothers with normal BMI (45.18 versus 5.92). Among women with BMI $\geq$ 30.0, the stillbirth rate was consistently greater with increment in the severity of the obesity (type III > type II > type I).

Table 3 shows the unadjusted and adjusted risk ratio estimates for the association between fetal size at birth and stillbirth stratified by obesity status. Among normosomic fetuses, the risk for stillbirth was only increased among women with obesity type I and type II. By contrast, fetuses identified with macrosomia and born to women with obesity as a general condition experienced about 30 % elevation in the adjusted risk for stillbirth when compared to those of women without obesity (RR = 1.31; 95 % Confidence Interval (CI): 1.22–1.42). However, the risk of stillbirth among mothers with obesity was only elevated for fetal macrosomia Grade 2 and Grade 3 phenotypes. Within each fetal macrosomia phenotype category, the risk for stillbirth increased in a dose-response pattern with ascending severity of obesity (type III > type II > type I).

Discussion

In this large, nationally representative cross-sectional study, we found that with increasing maternal BMI, the severity of macrosomia and the rates of stillbirth increased. The risk of stillbirth among macrosomic infants born to mothers with obesity was about 30 % higher compared to macrosomic infants born to mothers with normal weight. We also observed a dose-response relationship between obesity severity and the risk of stillbirth among macrosomic infants. This dose-response pattern remained for all the macrosomic phenotypes. It is important to note that among obesity type 1 mothers, macrosomia was protective against stillbirth. This intriguing finding was primarily observed as a significantly reduced risk of stillbirth in infants with macrosomia phenotype 1. Further studies are required to elucidate plausible mechanisms or establish whether this was a random error.

Prior studies have determined that obesity in mothers portends a worse prognosis in pregnancy and is associated with a number of adverse outcomes, including fetal macrosomia and stillbirth [15,31,32]. In a retrospective cohort study among nulliparous singletons births, Battacharya et al. documented rates of macrosomia and stillbirth that were twice as high in women with obese and morbidly obese BMIs than in those with normal BMI [33]. Similarly, our study demonstrated a rate of macrosomia that was nearly double in those who had obesity versus those who did not. A

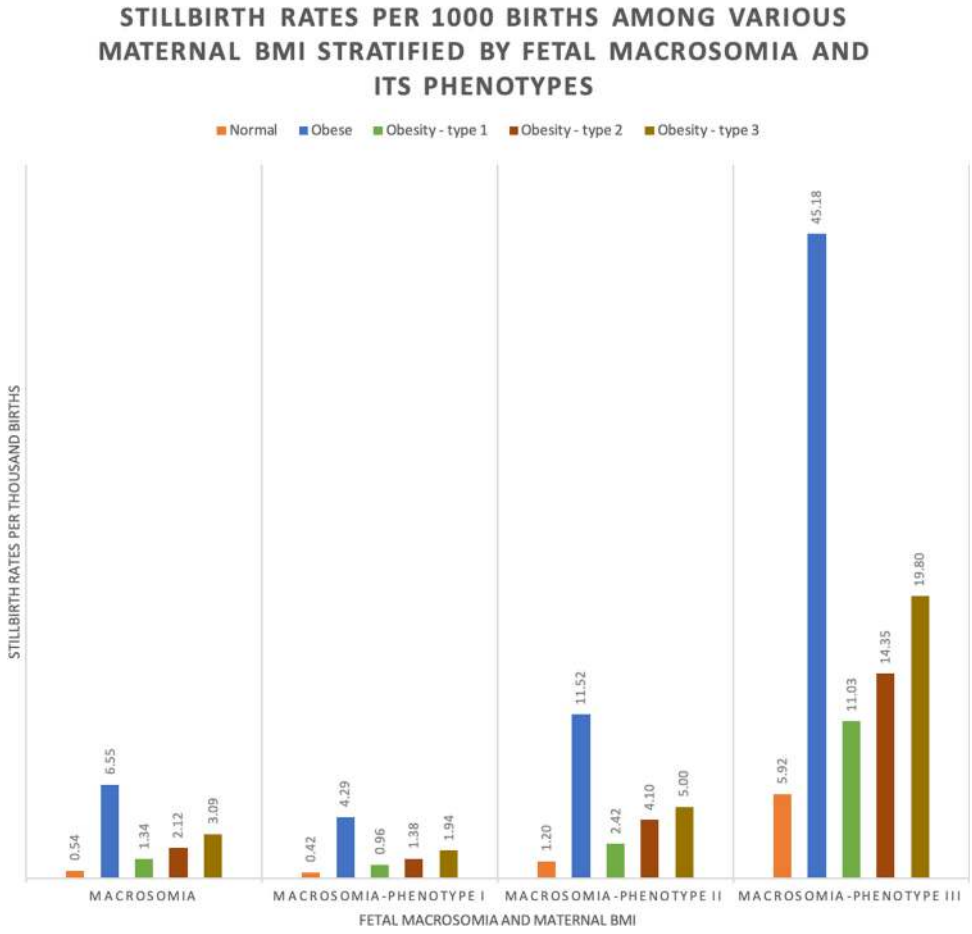

Fig. 1. Stillbirth rates per 1000 total births among various maternal BMI stratified by fetal macrosomia and its phenotypes.

Table 3
Association between maternal obesity and stillbirth among infants with fetal macrosomia, stratified by macrosomia phenotypes.

	Normal Birthweight		Macrosomic		Macrosomia Phenotype 1		Macrosomia Phenotype 2		Macrosomia Phenotype 3	
	Unadjusted RR	Adjusted RR	Unadjusted RR	Adjusted RR	Unadjusted RR	Adjusted RR	Unadjusted RR	Adjusted RR	Unadjusted RR	Adjusted RR
Maternal BMI Classification										
Normal BMI	reference	reference	reference	reference	reference	reference	reference	reference	reference	reference
Women with Obesity (All Types)	0.73(0.64–0.84)	0.92(0.80–1.05)	1.35(1.25–1.45)	1.31(1.22–1.42)	1.06(0.93–1.20)	1.07(0.94–1.22)	1.40(1.22–1.61)	1.32(1.15–1.52)	1.66(1.46–1.90)	1.50(1.31–1.71)
Obesity - type I	0.57(0.49–0.67)	0.72(0.61–0.85)	0.89(0.81–0.98)	0.87(0.79–0.96)	0.76(0.65–0.89)	0.77(0.65–0.90)	0.92(0.77–1.10)	0.87(0.72–1.04)	1.04(0.87–1.25)	0.96(0.80–1.15)
Obesity - type II	1.62(1.21–2.15)	2.12(1.58–2.83)	2.47(2.16–2.83)	2.37(2.07–2.72)	1.91(1.50–2.43)	1.95(1.53–2.48)	2.72(2.14–3.45)	2.58(2.03–3.29)	2.70(2.14–3.40)	2.24(1.76–2.83)
Obesity - type III	8.02(5.11–12.60)	9.79(6.09–15.74)	10.44(8.82–12.37)	9.06(7.61–10.78)	8.78(6.43–11.98)	8.44(6.15–11.57)	9.64(6.99–13.29)	7.41(5.28–10.39)	10.84(8.35–14.07)	8.51(6.50–11.15)

Note: For the adjusted models, the following variables were loaded: maternal age, race, nativity and educational qualification.

meta-analysis conducted by Chu et al. reported about a doubling of the odds (unadjusted OR: 2.07, 95 % CI: 1.59–2.74) of stillbirth across eight countries among mothers with obesity compared to mothers of normal weight [20]. Even though we also observed an increased risk of stillbirth in our study, our crude estimate was lower than what was described by Chu et al. The lower risk might be due to our study population, which included both multiparous and nulliparous women. Only one of the nine studies included in the meta-analysis had multiparous women. Another difference between our study population and those included in the meta-analysis is that we focused on a high-risk subgroup, macrosomic infants, and stratified our analyses by macrosomia phenotypes.

Although a biological pathway for the relationship between obesity and an increased risk of stillbirth in macrosomic fetuses has not been established, there are suggested mechanisms to explain the association between obesity and stillbirth. One that may explain our findings is that obesity in pregnancy increases the risk for gestational diabetes mellitus and hypertensive disorders, both of which are known to be risk factors for stillbirth [12,34]. Many studies have suggested that the association between obesity and stillbirth is not quite explained by gestational diabetes mellitus or hypertensive disorders [19]. However, when considering the prevalence of these undiagnosed conditions, we speculate that they may serve as contributing factors. This is plausible because the etiology of 50 % of all stillbirths remain unexplained [34,35]. It has also been suggested that women with normal weight may be better able to perceive decreased fetal movements than women with obesity, which would engender more timely medical treatment [12]. This mechanism may apply irrespective of whether the fetus is macrosomic or not.

The main limitation of our study is the possibility of residual confounding since we did not control for potential factors such as parity, substance abuse, alcoholism, and smoking as well as other inherent maternal conditions that may be associated with maternal obesity and stillbirth. For instance, in a case-control study, Varner et al. found a significant association between smoking and illicit drug use to stillbirth [36]. Because data on these covariates were not available for the same period as the Birth Data and Fetal Death Data, we could not adjust for these factors. Another important limitation of our study is the lack of direct fetal growth measures that capture fetal macrosomia before the occurrence of the fetal death. It should be noted that fetal weight among stillborn fetuses maybe affected by loss of mass due to maceration or conversely increase weight due to hydrops fetalis or other lethal congenital conditions. Given the lack of fetal weight measures before birth in this dataset, we consider the next best measure the official birth weight recorded in the birth certificates. We recommend that future studies consider prospective assessments of fetal weight. We also observed that mothers with missing information on socio-demographic characteristics had the most elevated risk for stillbirth. We believe that the missing data are unlikely to be random and may result from a high degree of misclassification. For instance, mothers missing mode of delivery information (i.e., the group with the greatest stillbirth rate) are likely to have had vaginal birth as it is implausible that cesarean section will be undocumented. This implies that the true estimate of stillbirth in women who had vaginal delivery was underestimated. Nevertheless, we do not expect the missingness of this data to affect our findings because none of the variables with missing data were key study variables. We had no missing data on maternal obesity, macrosomia, or stillbirth. Nevertheless, our research has some notable strengths, including the large sample size and the evaluation of stillbirth among various maternal BMI sub-groups at the phenotypic levels of macrosomic infants. This dose-response relationship observed may provide more insights into understanding the etiology and the mechanisms of stillbirth.

In summary, we observed that maternal obesity increases the risk of stillbirth among macrosomic infants. Preconception counseling should reinforce the need for optimal pre-pregnancy BMI. Weight loss in women with obesity should be encouraged, as this may help reduce adverse fetal outcomes.

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Declaration of Competing Interest

The authors have no financial disclosures to declare and no conflicts of interest to report.

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