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Original Research

Association of Overweight and Obesity Development Between Pregnancies With Stillbirth and Infant Mortality in a Cohort of Multiparous Women

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OBJECTIVE: To identify the association of newly developed prepregnancy overweight and obesity with stillbirth and infant mortality.

METHODS: We studied subsequent pregnancies of mothers who were normal weight at fertilization of their first identified pregnancy, from a population-based cohort that linked birth registry with death records in Pennsylvania, 2003–2013. Women with newly developed prepregnancy overweight and obesity were defined as those whose body mass index (BMI) before second pregnancy was between 25 and 29.9 or 30 or higher, respectively. Our main outcomes of interest were stillbirth (intrauterine death at 20 weeks of gestation or greater), infant mortality (less than 365 days after birth), neonatal death (less than 28 days after birth) and postneonatal death (29–365 days after birth). Associations of both prepregnancy BMI categories and continuous BMI with each outcome were estimated by nonparametric targeted minimum loss-based estimation and inverse-probability weighted dose–response curves, respectively, adjusting for race–ethnicity, smoking, and other confounders (eg, age, education).

RESULTS: A cohort of 212,889 women were included for infant mortality analysis (192,941 women for stillbirth analysis). The crude rate of stillbirth and infant mortality in these final analytic cohorts were 3.3 per 1,000 pregnancies and 2.9 per 1,000 live births, respectively. Compared with women who stayed at a normal weight in their second pregnancies, those becoming overweight had 1.4 (95% CI 0.6–2.1) excess stillbirths per 1,000 pregnancies. Those becoming obese had 3.6 (95% CI 1.3–5.9) excess stillbirths per 1,000 pregnancies and 2.4 (95% CI 0.4–4.4) excess neonatal deaths per 1,000 live births. There was a dose–response relationship between prepregnancy BMI increases of more than 2 units and increased risk of stillbirth and infant mortality. In addition, BMI increases were associated with higher risks of infant mortality among women with shorter interpregnancy intervals (less than 18 months) compared with longer intervals.

CONCLUSION: Transitioning from normal weight to overweight or obese between pregnancies was associated with an increased risk of stillbirth and neonatal mortality.

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The obesity epidemic has affected millions of people worldwide, including 30% of adults in the United States.[1] Importantly, obesity is more prevalent in women of childbearing age compared with general population estimates. From 2007–2008 to 2015–2016, the prevalence of obesity among women aged 20–39 years increased from 31% to 36%.[2] Prepregnancy

obesity is a common high-risk obstetric condition with wide-ranging health effects,[3] including stillbirth[4] and infant death.[5]

Obesity is often defined through body mass index (BMI, calculated as weight in kilograms divided by height in meters squared), which is correlated with body fat percentage in women before fertilization.[6] However, obesity is a complex and heterogeneous disorder presenting with multiple subphenotypes. The effect of obesity subphenotypes such as with or without metabolic syndromes or other comorbidities, on adverse pregnancy outcomes can vary.[7,8] The duration that a woman is exposed to the obese phenotype may also affect her risk for obstetric and perinatal complications. For example, longer duration and greater severity of obesity are associated with higher cardiometabolic risk.[9,10] To date, however, there is little information regarding the timing of obesity onset relative to pregnancy outcomes.[11] Given this, we evaluated newly developed prepregnancy overweight and obesity as well as the change in BMI in relation to stillbirth and infant mortality.

METHODS

The Penn MOMS study, which included fetal death records and linked birth–infant death records in Pennsylvania from 2003 to 2013 (n=1,551,919 singleton pregnancies), was used to construct our analytic cohort. Unique identifiers were used to link pregnancies from the same women. In Pennsylvania, fetal death records before 2006 did not contain information to calculate BMI; therefore, women with stillbirth before 2006 (4,001 women; 5,168 pregnancies) were excluded. Details on data cleaning and linkage processes have been published previously.[12,13]

We established our analytic cohort by including women who had at least two pregnancies during the study period 2003–2013 and were a normal weight at the start of the first identified pregnancy (Fig. 1). Women with questionable data (illogical age or interpregnancy intervals) or prior twin gestations were excluded. We then limited our analytic sample to these women's second pregnancies. Overall, there were 212,889 pregnancies for the infant mortality analysis (2003–2013), and 192,941 pregnancies for the stillbirth analysis (2006–2013). This study was approved by the institutional review board at the University of Pittsburgh.

Outcomes of interest were stillbirth, which was defined as intrauterine death at 20 weeks of gestation or greater, and infant mortality, defined as the death of nonanomalous liveborn infants at less than 365 days. We further divided infant mortality into two groups based on the timing of death: neonatal death (less than 28 days) and postneonatal death (28 to less than 365 days).

Prepregnancy BMI was computed by using weight (kg) and divided by height (m) squared. Both weight and height were obtained from interviewing mothers before discharge from the hospital. We categorized BMI as underweight (less than 18.5), normal weight (BMI between 18.5 and 24.9), overweight (BMI between 25 and 29.9) or obese (30 or greater).[14] Women who were normal weight in the first-identified pregnancy in our cohort and became overweight or obese in their second pregnancy were considered to have newly developed overweight or obesity. Interpregnancy BMI changes were calculated as the difference in prepregnancy BMI units of first and second pregnancy.

We used causal diagrams to identify confounders (Appendix 1, available online at http://links.lww.com/AOG/B691).[15] We adjusted for maternal height and race–ethnicity, parity, as well as other characteristics during prior and current pregnancies including inter-pregnancy interval, maternal education, urban residence, percentage of black residents in census tract, prepregnancy diabetes, prepregnancy hypertension, smoking status, marital status, and payer status. In addition, we adjusted for prior pregnancy characteristics including gestational diabetes, gestational hypertension, smoking status during pregnancy, gestational age, birth weight, birth facility level of neonatal care, neonatal intensive care units admission, use of the Special Supplemental Program for Women, Infants, and Children, breast feeding, mode of delivery, Apgar score, stillbirth, and infant death. Inter-pregnancy interval, maternal height, gestational age, and birthweight were treated as continuous variables. Other confounders were categorized based on the groups listed in Table 1 and Appendix 2 (Appendix 2 is available online at http://links.lww.com/AOG/B691).

Information on maternal characteristics (race–ethnicity, age, education, marital status, smoking status), delivery payment insurance, prepregnancy diabetes or hypertension, gestational age at delivery, and level of neonatal care available in the birth facility were acquired from hospital discharge records. Neighborhood socioeconomic status (urbanity and the proportion of black residents) were computed based on the county-level federal information processing standards codes of the primary residence address. Inter-pregnancy interval was calculated as the number of months between delivery date of previous pregnancy and estimated fertilization date of current pregnancy. Roughly 20% of pregnancies were missing data

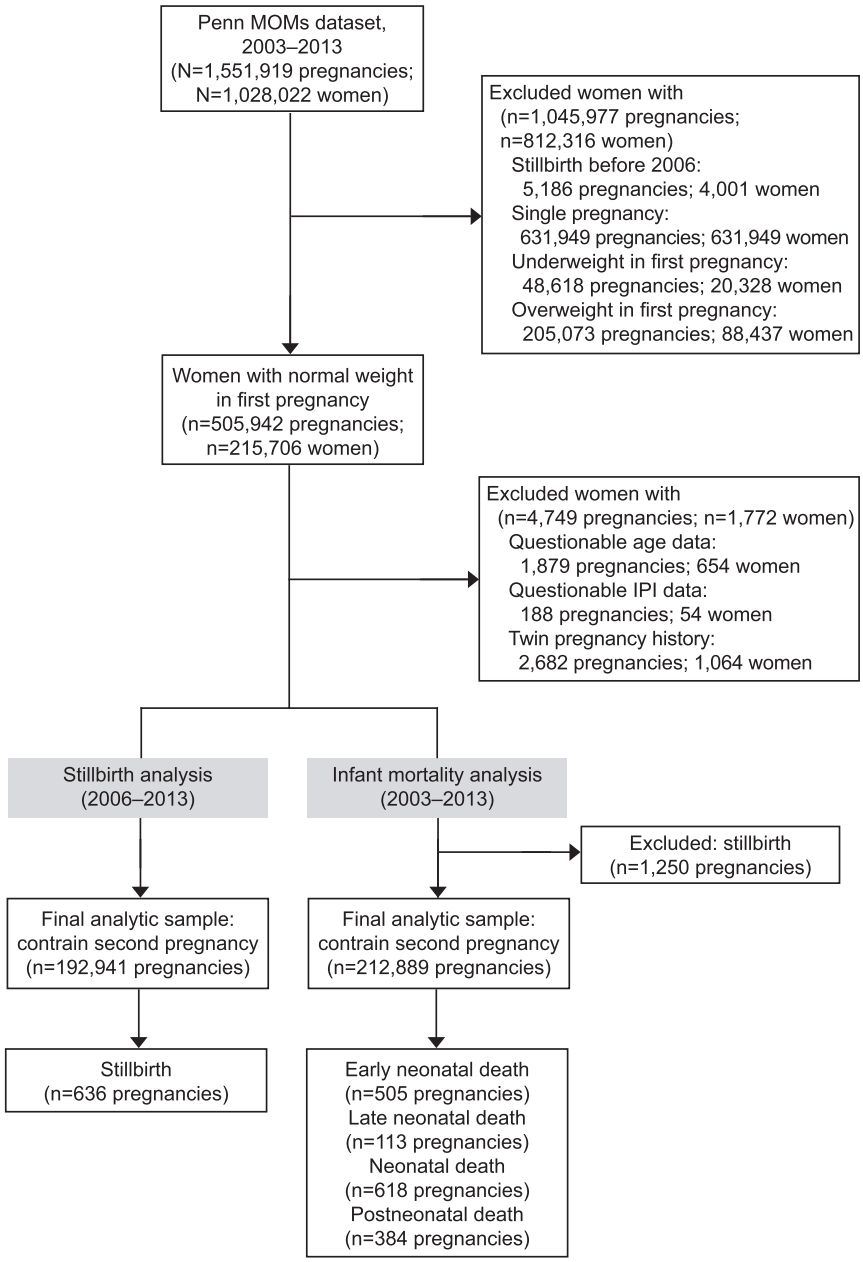

Fig. 1. Flow chart of analytic sample selection. IPI, interpregnancy interval.

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on variables of interest, which were imputed using a Markov chain Monte Carlo approach.[16] Details on variable collection and imputation have been previously provided.[12,13]

Our primary analysis aimed to determine the association between newly developed (or "incident") overweight and obesity before pregnancy and the risk of stillbirth and infant mortality. Nonparametric targeted minimum loss-based estimation[17] was used to estimate risk differences and risk ratios for the relation between categorical prepregnancy BMI (becoming underweight, becoming overweight, or becoming obese compared with remaining normal weight) and our outcomes of interest. We opted for targeted minimum loss-based estimation rather than standard regression because targeted minimum loss-based estimation is a "doubly robust" approach in that it combines a propensity score model for the exposure (BMI category) with a regression model for the outcome (stillbirth, infant mortality), thus providing two chances to adjust for potential confounding.[18] Furthermore, unlike standard regression, targeted minimum loss-based estimation enables use of nonparametric machine learning techniques that do not rely on

Table 1. Characteristics of Mothers Who Remained Normal Weight or Became Underweight, Overweight, or Obese in Their Second Pregnancy for Infant Mortality Analysis, Pennsylvania Birth Records, 2003–2013 (N=212,889)

Maternal Characteristics	Became Underweight (n=5,451)	Remained Normal Weight (n=160,001)	Became Overweight (n=39,011)	Became Obese (n=8,426)
Race–ethnicity				
Non-Hispanic white	3,948 (72)	125,246 (78)	27,068 (69)	5,288 (63)
Non-Hispanic black	652 (12)	15,527 (9.7)	6,163 (16)	1,834 (22)
Hispanic	483 (8.9)	11,089 (6.9)	4,151 (11)	1,051 (13)
Others	368 (6.8)	8,139 (5.1)	1,629 (4.2)	250 (3.0)
Age (y)				
Younger than 20	389 (7.1)	6,024 (3.8)	1,772 (4.5)	461 (5.5)
20–29	3,211 (59)	74,788 (47)	21,379 (55)	5,303 (63)
Older than 29	1,851 (34)	79,189 (50)	15,860 (41)	2,662 (32)
Education				
Less than high school	1,167 (21)	19,750 (12.3)	5,996 (15)	1,739 (21)
High school or equivalent	1,581 (29)	35,196 (22)	11,428 (29)	3,027 (36)
Some college	1,202 (22)	37,649 (24)	10,934 (28)	2,421 (29)
College graduate	1,501 (28)	67,406 (42)	10,653 (27)	1,239 (15)
Metropolitan area (no. of residents)				
More than 1 million	2,865 (53)	85,355 (53)	20,246 (52)	4,296 (51)
250,000–1 million	1,521 (28)	46,115 (29)	11,406 (29)	2,454 (29)
Less than 250,000	1,065 (20)	28,531 (18)	7,359 (19)	1,676 (20)
% black residents in neighborhood (tertile)				
Lowest (less than 1%)	1,874 (34)	57,283 (36)	12,728 (33)	2,459 (29)
Middle (1–7%)	1,781 (33)	59,299 (37)	12,668 (33)	2,376 (28)
Highest (greater than 7%)	1,796 (33)	43,419 (27)	13,615 (35)	3,591 (43)
Prepregnancy diabetes				
No	5,439 (99.8)	159,547 (99.7)	38,828 (99.5)	8,373 (99.4)
Yes	12 (0.2)	454 (0.3)	183 (0.5)	53 (0.6)
Prepregnancy hypertension				
No	5,431 (99.6)	159,187 (99.5)	38,653 (99.1)	8,234 (97.7)
Yes	20 (0.4)	814 (0.5)	358 (0.9)	192 (2.3)
Prepregnancy smoking (cigarettes/d)				
0	3,767 (69.1)	131,100 (82)	30,592 (78)	6,282 (75)
Less than 10	430 (7.9)	7,852 (4.9)	2,495 (6.4)	612 (7.3)
10–20	456 (8.4)	8,639 (5.4)	2,505 (6.4)	616 (7.3)
More than 20	798 (15)	12,510 (7.8)	3,419 (8.8)	916 (11)
Insurance				
Nonprivate	2,698 (50)	53,279 (33)	16,849 (43)	4,734 (56)
Private	2,753 (50)	106,722 (67)	22,162 (57)	3,692 (44)
Marital status				
Unmarried	2,423 (44)	45,542 (29)	15,342 (39)	4,349 (52)
Married	3,028 (56)	114,459 (72)	23,669 (61)	4,077 (48)
Interpregnancy interval (mo)				
Less than 18	3,341 (61)	101,450 (63)	26,480 (68)	6,145 (73)
18 or greater	2,110 (39)	58,551 (37)	12,531 (32)	2,281 (27)

Data are n (%).

unverifiable modeling assumptions (eg, linearity, additivity, no interaction) made in parametric regression. In our analyses, we used stacking[19,20] to combine several machine learning algorithms into one. We included the arithmetic mean, neural networks, multivariate adaptive regression splines, least absolute shrinkage and selection operator, generalized additive models, random forests, and gradient-boosted machines. These algorithms were used to estimate both the propensity score model for BMI category, and the

outcome models for stillbirth and infant mortality. To further triangulate this relationship between newly developed overweight and obesity before the pregnancy and risk of stillbirth and infant mortality, we conducted propensity score matching analysis (Appendix 3, available online at http://links.lww.com/AOG/B691). In brief, propensity score was estimated by fitting logistic regression with the same set of confounders aforementioned. Resulting propensity scores were used in 1:1 nearest neighbor matching within a caliper of 0.1 SDs of the logit propensity scores with replacement. Risk difference and risk ratios of the outcomes were estimated by generalized estimating equations accounting for matched pairs. All analyses were performed with the Matching and geepack packages in R. To examine the dose–response relation between the continuous interpregnancy BMI change and the risk of stillbirth and infant mortality, we further modeled interpregnancy BMI change using restricted cubic splines with four knots, and weighted by inverse of the propensity score to adjust for confounding.[21] We could not employ targeted minimum loss-based estimation because software routines are not currently available for continuous exposures. Knots for the restricted cubic splines were located at the 20th, 40th, 60th, and 80th percentiles of the distribution of BMI among the events.[22] These curves were also stratified by interpregnancy interval length. We adopted the commonly used cutpoints of less than 18 months to define short interpregnancy interval.[23]

RESULTS

Among 212,889 women who were normal weight in their initial pregnancy, most (75%) remained normal weight; 3%, 18%, and 4% became underweight, overweight, or obese, respectively, in their next pregnancy. The median interpregnancy interval was roughly 23 months (interquartile range: 24). Mothers with incident prepregnancy overweight or obesity were more likely than women who remained normal weight to be non-Hispanic black, younger, without college education, with prepregnancy diabetes and hypertension, smokers, and living in neighborhoods with higher percentages of black residents (Table 1). Women with incident overweight or obesity were more likely to have nonprivate insurance, shorter interpregnancy intervals, and be unmarried. Similar differences were observed in the smaller stillbirth cohort (Appendix 4, available online at http://links.lww.com/AOG/B691). During their previous pregnancy, women who became overweight or obese experienced more adverse pregnancy outcomes, had higher gestational weight gain, and lower prevalence of breast feeding (Appendices 2 and 5, available online at http://links.lww.com/AOG/B691).

The confounder adjusted association between BMI category change and the risk of stillbirth is provided in Table 2. Compared with women who remained normal weight in their second pregnancy, those who became underweight, overweight, or obese had 2.9 (95% CI 0.5–5.3), 1.4 (95% CI 0.6–2.1), or 3.6 (95% CI 1.3–5.9) excess stillbirth per 1,000 pregnancies, respectively. We did not find strong associations between changing prepregnancy BMI category and infant mortality or postneonatal mortality. However, women with newly developed obesity had 2.4 (95% CI 0.4–4.4) excess neonatal mortality events per 1,000 births compared with those women who maintained normal weight. A similar pattern was observed for risk ratios. Results from a matched PS analysis provided estimates that were similar to or larger than those obtained from targeted minimum loss-based estimation (Appendix 6, available online at http://links.lww.com/AOG/B691).

There was a U-shaped relation between interpregnancy BMI change for the risks of stillbirth and infant death, with the lowest risks at BMI change of 0–2 units (Fig. 2A and B). Risks of both outcomes rose sharply as BMI increased beyond 2-kg/m^2 (equivalent to an average interpregnancy weight gain of 5 kg for women with 160 cm height). These risks also increased when BMI decreased between two pregnancies. Although there were no meaningful differences in the stillbirth risk curve according to interpregnancy interval (Fig. 2C), the infant mortality risk curve varied according to interpregnancy interval (Fig. 2D). The association between BMI change and risk of infant death was slightly stronger for women with short interpregnancy intervals compared with those with a longer interpregnancy interval. For example, among women with short interpregnancy intervals, the risk of infant mortality increases by 2 per 1,000 live births with BMI changes of 2–6 units (from 5.8 [95% CI 4.9–6.9] to 7.8 [95% CI 6.5–9.4] per 1,000 live births). However, among women with long interpregnancy intervals, the risk of infant mortality increases by 1 per 1,000 live births with BMI changes of 2–6 units (from 3.7 [95% CI 3.1–4.4] to 4.7 [95% CI 3.9–5.6] per 1,000 live births). The curves for neonatal mortality showed similar patterns as the infant mortality curves, but no association was observed between interpregnancy BMI changes and postneonatal mortality (Fig. 3).

DISCUSSION

We found women at normal weight at initial pregnancy fertilization who became underweight, overweight, or

Table 2. Risk Difference and Ratios of Stillbirth and Infant Mortality by Prepregnancy Body Mass Index Category

BMI Category*	Event	Population at Risk	Unadjusted Risk†	Risk Difference (95% CI)‡§	Risk Ratio (95% CI)§
Stillbirth					
Became underweight	28	4,891	5.7	2.9 (0.5–5.3)	2.0 (1.3–3.1)
Remained normal weight	388	144,366	2.7	Reference	Reference
Became overweight	160	35,834	4.5	1.4 (0.6–2.1)	1.5 (1.2–1.8)
Became obese	60	7,850	7.6	3.6 (1.3–5.9)	2.3 (1.6–3.3)
Infant mortality					
Became underweight	30	5,451	5.5	−0.3 (−2.0 to 2.0)	1.0 (0.7–1.5)
Remained normal weight	719	160,001	4.5	Reference	Reference
Became overweight	189	39,011	4.8	−0.2 (−0.9 to 0.6)	1.0 (0.8–1.1)
Became obese	64	8,426	7.6	2.0 (−0.2 to 4.1)	1.4 (1.0–2.0)
Neonatal mortality					
Became underweight	19	5,451	3.5	0.4 (−1.3 to 2.2)	1.2 (0.7–2.0)
Remained normal weight	436	160,001	2.7	Reference	Reference
Became overweight	115	39,011	2.9	−0.7 (−0.7 to 0.5)	1.0 (0.8–1.2)
Became obese	48	8,426	5.7	2.4 (0.4–4.4)	1.8 (1.3–2.7)
Postneonatal mortality					
Became underweight	11	5,432	2.0	−0.4 (−1.5 to 0.6)	0.8 (0.4–1.6)
Remained normal weight	283	159,565	1.8	Reference	Reference
Became overweight	74	38,896	1.9	−0.7 (−0.6 to 0.4)	1.0 (0.7–1.3)
Became obese	16	8,378	1.9	−0.4 (−1.3 to 0.6)	0.8 (0.4–1.5)

BMI, body mass index.

* BMI category (kg/m^2): underweight (less than 18.5), normal weight (BMI between 18.5 and 24.9), overweight (BMI between 25 and 29.9), obese (30 or higher).

† Risk: per 1,000 live births (or pregnancies).

‡ Risk difference: per 1,000 live births (or pregnancies).

§ Covariates adjusted in the model: maternal race–ethnicity, height, parity, interpregnancy interval between current and previous pregnancy; variables of prior and current pregnancies: maternal age, education, urban residence, percentage of black residents, prepregnancy diabetes, prepregnancy hypertension, smoking status, marital status and insurance of current and prior pregnancy; variables of prior pregnancy: gestational diabetes, gestational hypertension, smoking status during pregnancy, gestational age, birth weight, birth facility level of neonatal care, neonatal intensive care unit admission, Women, Infants, and Children program usage, breast feeding, mode of delivery, Apgar score, stillbirth, and infant death.

obese before fertilization of the subsequent pregnancy had higher stillbirth and neonatal mortality rates than women who remained normal weight. We identified a U-shaped relation between interpregnancy BMI changes and risk of stillbirth and infant mortality. This association between BMI changes and infant mortality was stronger among the subgroup of women with short interpregnancy intervals than those with longer intervals. Our results suggest that becoming obese within an average of 2 years after pregnancy is associated with higher risk of stillbirth and neonatal mortality than remaining at a normal weight.

Three studies[24–26] examined maternal BMI in relation to stillbirth and infant mortality across multiple pregnancies. One study using Missouri vital records[25] found that compared with mothers staying at normal weight, normal weight mothers becoming overweight or obese had risks of stillbirth around 20% and 50% greater, respectively. The magnitudes of these associations are smaller than in our study. This difference may be explained by their adjustment for several variables (including preeclampsia and gestational diabetes in the second pregnancy) that are affected by prepregnancy BMI. Controlling for intermediates can result in overadjustment bias,[27] leading to potentially misleading estimates. Two other studies using a Swedish population-based cohort attempted to answer questions about interpregnancy weight change. Villamor et al[24] found an association between BMI changes and stillbirth in the overall analysis but not in the subgroup of women with BMIs less than 25 at first pregnancy. Cnattingius et al[26] found that among women whose BMI was less than 25 at first pregnancy, gaining more than 2 units of BMI increased risk of stillbirth, infant mortality, neonatal mortality, and postneonatal mortality. We had similar findings except for postneonatal mortality. Compared with their study, we adjusted for additional confounders (eg, characteristics from the prior pregnancy) which may provide less biased results. Our study also did not include women who were underweight in their first pregnancy. Although the literature does not

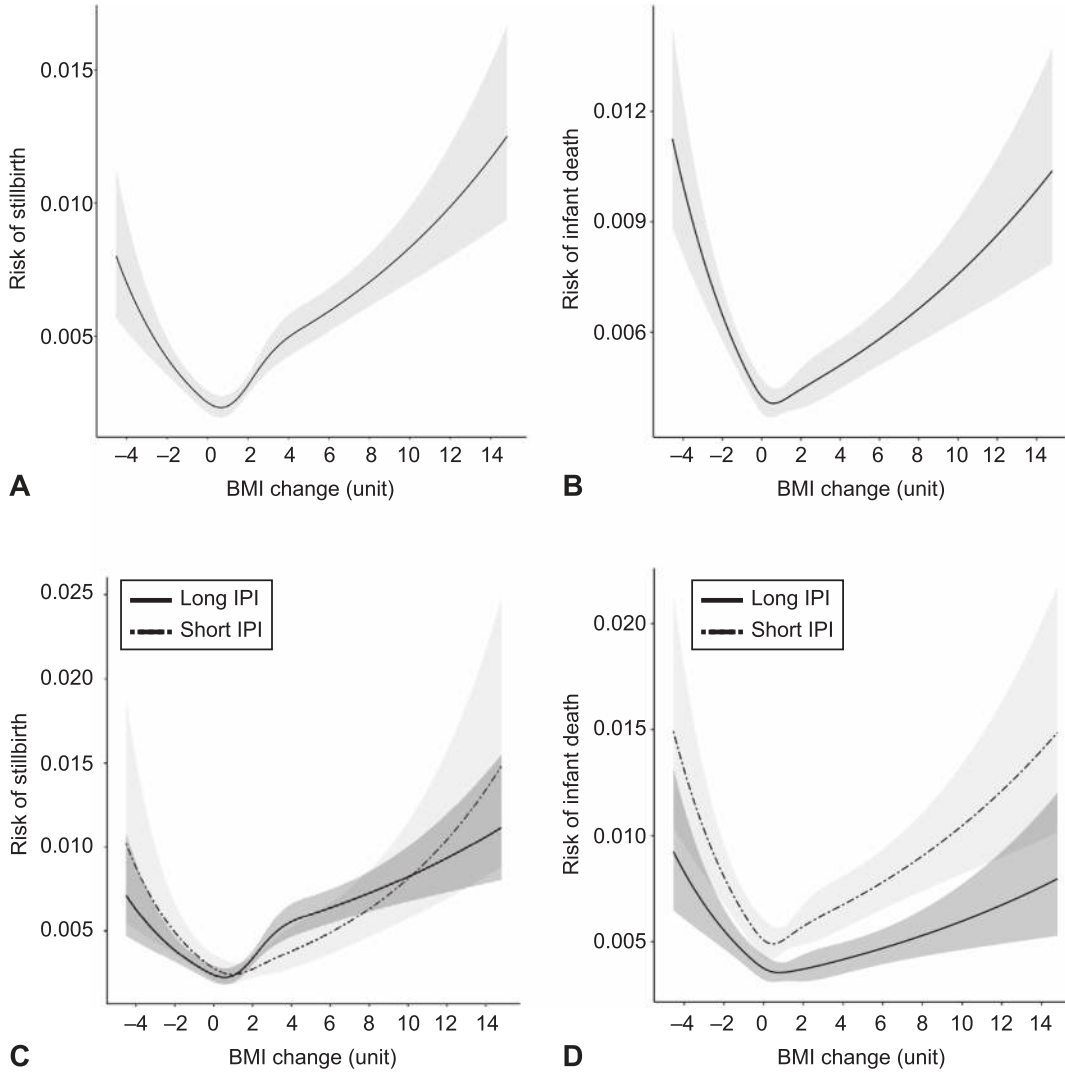

Fig. 2. Dose–response curve of interpregnancy body mass index (BMI) change with risk of stillbirth and infant mortality. Curve fitted by restricted cubic splines, with 4 knot and pointwise confidence bands constructed by bootstrap method. Risk presented as per 1,000 pregnancies (live births). Short interpregnancy interval (IPI) defined as IPI less than 18 months. Long IPI defined as IPI 18 months or more. Interpregnancy BMI change and risk of stillbirth (**A**), interpregnancy BMI change and risk of infant mortality (**B**), interpregnancy BMI change and risk of stillbirth by IPI (**C**), and interpregnancy BMI change and risk of infant mortality by IPI (**D**).

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examine underlying mechanisms of newly developed obesity, cumulative evidence in prepregnancy obesity suggests several plausible explanations including placental dysfunction, inflammation, and metabolic abnormalities.[28,29]

Contrary to previous literature,[24–26] we found increased risk of stillbirth among women who became underweight after being normal weight in a previous pregnancy. The plausible mechanisms remain unclear. Interpregnancy weight loss may affect changes in placental function–there is evidence suggesting a relation with lower placental weight.[30] However, these findings may also be affected by unmeasured risk factors such as underlying illness or psychosocial factors.

We found that interpregnancy interval modified the association between BMI increase and risk of infant death. Studies show that women start to gain weight 1 year after delivery[31] and late postpartum weight gain is not associated with gestational weight gain of prior pregnancy.[32] It is plausible that BMI increase among women with shorter interpregnancy intervals represents postpartum weight retention, whereas BMI increase among those with longer

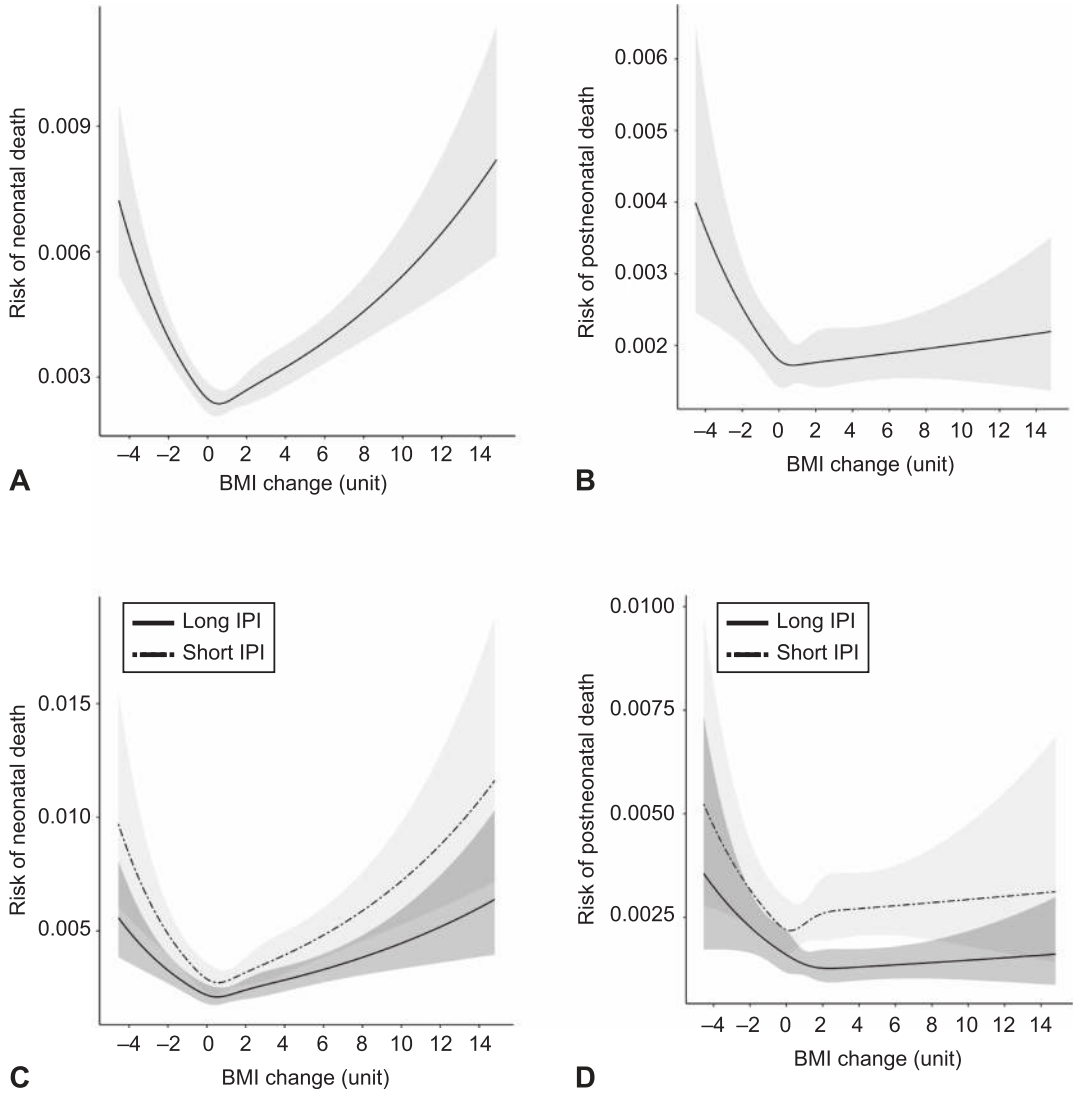

Fig. 3. Dose–response curve of interpregnancy body mass index (BMI) change with risk of neonatal and postneonatal mortality. Curve fitted by restricted cubic splines, with 4 knot and pointwise confidence bands constructed by bootstrap method. Risk presented as per 1,000 live births. Short interpregnancy interval (IPI) defined as IPI less than 18 months. Long IPI defined as IPI 18 months or more. Interpregnancy BMI change and risk of neonatal mortality (**A**), interpregnancy BMI change and risk of postneonatal mortality (**B**), interpregnancy BMI change and risk of neonatal mortality by IPI (**C**), and interpregnancy BMI change and risk of postneonatal mortality by IPI (**D**).

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interpregnancy intervals represents postpartum weight gain. These reasons for BMI changes may differentially affect the risk of infant death.

Our findings should be interpreted considering key limitations. Body mass index calculated from self-reported height and weight may result in misclassification.[33] However, previous studies analyzing this cohort showed that after accounting for misclassification, the relations between prepregnancy obesity and infant mortality were not meaningfully different.[12] In addition, BMI measurements were only available at the start of each pregnancy. We could not account for a history of high BMI in the women who were normal weight at first pregnancy nor understand whether weight change between pregnancies was because of postpartum weight retention or postpartum weight gain. Furthermore, to identify newly developed overweight or obesity, we restricted our analysis to women with two pregnancies and were normal weight before the first pregnancy. Therefore, our results estimated among multiparous women should be carefully generalized to other populations (eg, nulliparous women).

Reproductive history plays an important role in subsequent pregnancies, affecting both exposure and outcomes of interest.[34] Therefore, we adopted a reproductive life-based approach,[35] which considers potential effects from prior pregnancies. Unlike previous studies restricting analyses to women without adverse events in the first pregnancy,[24–26] we adjusted for pregnancy outcome and other characteristics of the prior pregnancy. Thus, we accounted for confounding of reproductive history without introducing selection bias.[27] In addition, we avoided adjusting for obstetric complications or any variables on the pathway from prepregnancy BMI to stillbirth or infant mortality of the second pregnancy.

Postpartum weight retention is a common reason for women to become overweight or obese; around 13–20% of women retain 5 kg or more of their prepregnancy weight 1 year after delivery.[36] Health care providers should inform women throughout pregnancy the importance of adequate gestational weight gain[37] and returning to prepregnancy weight. The intervention efforts may be best targeted to women whose BMI is at the upper limit of normal weight,[38] eg, optimizing diet during pregnancy.[39] Pregnancy presents a motivating opportunity for a healthy lifestyle; however, major physical and social role changes complicate potential interventions. It is important to consider how to tailor interventions to meet personal needs and how to translate results of trials into formal clinical guidelines.[40,41]

Different approaches for weight maintenance likely have different effects on reducing risk of adverse pregnancy outcomes. To develop effective interventions for weight maintenance to prevent stillbirth and infant mortality, future studies focusing on understanding the reasons for becoming obese are warranted.

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