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Fetal heart rate and motor development in overweight and obese pregnant women

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Abstract

Objective—To determine the relationship between maternal prepregnancy body mass index (BMI) and fetal cardiac and motor activity and integration during the second half of pregnancy.

Methods—Longitudinal data were collected from 610 nonsmoking women with normally progressing pregnancies at three gestational periods (24, 30–32, and 36 weeks) across eight cohorts studied between 1997 and 2013. Fifty minutes of fetal heart rate and motor activity data were collected at each period via actocardiography in a laboratory setting. Data were digitized and analyzed using customized software. Standard BMI categories were computed from maternal prepregnancy weight and height. Participants were stratified into normal weight (n=401, 65.7%), overweight (n=137, 22.5%), or obese (n=72, 11.8%).

Results—Fetuses of obese women showed lower heart rate variability and fewer accelerations relative to fetuses of normal weight women. Fetuses of both obese and overweight women exhibited more vigorous motor activity than fetuses of normal weight women. Cardiac–somatic integration was reduced in both obese and overweight groups. Findings differed by gestational age at assessment.

Conclusions—Excess maternal prepregnancy weight in overweight and obese women alters the normal trajectory of fetal cardiac and motor development and their integration, with effects amplified as pregnancy progresses.

Keywords

Fetal heart rate; Fetal motor activity; Maternal overweight; Obesity and pregnancy; Prepregnancy body mass index

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Conflict of interest

The authors have no conflicts of interest.

1. Introduction

Increasing attention has been given to the effects of maternal obesity on pregnancy outcomes. Most extant literature focuses on how excess weight before and during pregnancy affects maternal health including metabolic and inflammatory processes [1–3]. Fewer studies focus on ramifications for the developing fetus; those that do typically evaluate the association between maternal weight and increased risk for adverse birth outcomes such as stillbirth [4] and both fetal growth restriction and macrosomia [5,6]. While birth weight reveals some information about disruption to the intrauterine milieu, it is an imperfect instrument to infer functional consequences for fetal neurodevelopment.

The knowledge that the expression and maturational trajectories of fetal cardiac and motor parameters reflect the developing nervous system has been well established in studies of fetuses in normally developing pregnancies, at risk pregnancies, and fetuses compromised by congenital conditions [7–11]. The aim of the present study was to examine the associations between maternal prepregnancy body mass index (BMI) and fetal neuromaturation indexed by measures of fetal heart rate, motor activity, and their integration. The study utilized prospectively collected data generated from a series of longitudinal cohort studies of pregnant women who were monitored on three occasions during the second half of pregnancy.

2. Materials and methods

The study sample was based on 773 enrollments across eight longitudinal cohorts of maternal–fetal pairs collectively known as the Johns Hopkins Fetal Development Project. Data collection was conducted within the Prenatal Diagnosis and Treatment Center at the Johns Hopkins Hospital, located in Baltimore, from June 19, 1997, to February 27, 2013. Individual cohort results in relation to primary aims unrelated to BMI are detailed elsewhere [12,13]; consistency in the basic data collection protocol for each cohort enabled aggregation. Pregnancies with congenital malformations (e.g. cleft palate) or conditions with known neurodevelopmental consequences (e.g. trisomy 21) were excluded (n=10). Twenty-three participants did not complete the study protocol resulting in a final sample of 740 eligible cases. The study was approved by the Johns Hopkins Institutional Review Board and women provided informed consent to participate.

Participants were nonsmoking women with normally progressing, singleton pregnancies without pre-existing medical complications known to complicate pregnancy (e.g. pregestational diabetes). Pregnancy dating was based on early detection (4.8 ± 1.5 weeks) followed by clinical confirmation (8.0 ± 2.3 weeks). Maternal medical and pregnancy history was recorded at intake and followed through delivery. BMI—weight in kilograms divided by height in meters squared—was calculated based on self-reported maternal prepregnancy weight. Categories were defined as: normal weight (18.5–24.9), overweight (25–29.9), or obese (≥ 30).

The final sample included a subset of siblings (n=197) generated from 106 women. Of these, 91 participated twice, 14 participated three times, and 1 with each of four pregnancies. We have previously shown significant correlations within sibling pairs for cardiac and motor

measures [14]. To alleviate statistical concerns regarding non-independence of observations, only the first pregnancy was included for women who participated more than once, resulting in 634 maternal–fetal pairs. A case of fetal ventricular septal defect brought the sample to 633 maternal–fetal pairs. Women who were underweight prior to pregnancy ($n=21$; BMI <18.5) and those with unreported weight ($n=2$) were excluded from the analysis, resulting in a final analytic sample of 610 maternal–fetal pairs.

Maternal–fetal data were based on the three gestational periods with the greatest commonality across cohorts. This includes an initial visit at 24 weeks and a later one at 36 weeks. All cohorts included data collection midway between these periods, at either 30 or 32 weeks. These gestational age periods are labelled G1 (24 weeks), G2 (30–32 weeks), and G3 (36 weeks). There is some variation in actual dates at testing for each period; comprehensive information about the cohorts is available elsewhere [14].

Prenatal visits were scheduled at 1:00 PM or 3:00 PM to control for potential diurnal or postprandial effects. At each visit, data were collected during a 50-minute monitoring period using an MT-325 fetal actocardiograph (Toitu, Tokyo, Japan). This monitor detects fetal movement and fetal heart rate (FHR) using a single-wide array transabdominal Doppler transducer. Data were digitized and analyzed off-line (GESTATE; James Long Company, Caroga Lake, NY, USA). FHR data underwent error rejection procedures based on moving averages of acceptable values as needed. Variable extraction included FHR, FHR variability (standard deviation of each 1-minute epoch of fetal heart rate averaged over time), and number of accelerations defined as excursions in fetal heart rate greater than or equal to 10 bpm for a minimum of 15 seconds. Fetal movement data represent raw voltage values generated from the actograph calibrated by multiplying by a conversion factor and scaled from 0 to 100 in arbitrary units (arb. unit). Fetal motor activity was computed as the total time moving—a product of the number of movements and their average duration—and level of vigor, represented by mean movement amplitude. The relation between fetal motor activity and fetal heart rate (cardiac–somatic coupling) was calculated as the proportion of discrete fetal movement bouts—defined as commencing when an actograph signal attained an amplitude of 15 units and ending with a cessation of signal for at least 10 seconds—associated with excursions in fetal heart rate ≥ 5 bpm over baseline within 5 seconds before the start of a movement or within 15 seconds after the start of a movement, consistent with previously developed criteria [15].

Sample characteristics were compared using general linear modeling or the χ^2 test where appropriate. Hierarchical linear modeling was used to test differences in the trajectory of fetal neurobehavioral development from the first to third gestational period by prepregnancy BMI group. Contrast estimates to test pairwise comparisons between prepregnancy BMI groups were specified and the level of statistical significance was set at $P<0.05$. Maternal education, race/ethnicity, and parity were identified as potential confounders and were entered as covariates. Analyses were performed using SAS version 9.2 (SAS Institute Inc, Cary, NC, USA).

3. Results

Of 610 healthy pregnant women, 401 (65.7%) were of normal weight, 137 (22.5%) were overweight, and 72 (11.8%) were obese based on prepregnancy BMI. Table 1 presents sociodemographic characteristics by maternal prepregnancy BMI group. Enrollment criteria resulted in a medically low-risk sample. The small number of women that developed gestational diabetes ($n=20$) or pregnancy-induced hypertension ($n=10$) was retained in the analysis. Women who were overweight or obese were significantly more likely to be African American or Asian ($P<0.001$) multiparous ($P=0.001$), have fewer years of education ($P<0.001$), report lower gestational weight gain ($P<0.001$), and to have been delivered by cesarean ($P=0.003$).

Offspring characteristics are presented in Table 2. Fifty-one percent of the fetuses were female. Infants that were delivered preterm ($n=39$; most [$n=32$] delivered at 35–36 weeks) or were growth restricted ($n=7$) were not excluded from the analysis. Neonatal outcomes were not associated with BMI category (Table 2).

3.1. Fetal heart rate

Signal loss resulted in the exclusion of, on average, 4.7% of fetal heart rate data in the 50-minute recording for normal weight women, 5.7% for overweight women, and 7.2% for obese women. Compared with normal weight women, signal loss was significantly higher for both overweight (5.7% vs 4.7%; $P=0.002$) and obese (7.2% vs 4.7%; $P<0.001$) women. Data are presented in Figures 1a and 1b. At G1, there were no differences in FHR, FHR variability, or number of accelerations by prepregnancy maternal BMI category (P range = 0.233 to 0.779) with the exception of a trend level for more accelerations in fetuses of obese women relative to normal weight women ($\beta = -0.77$, $SE = 0.45$, $t = -1.70$, $P=0.089$).

However, differences among groups in both rate of change and level emerged later in pregnancy. Fetuses of women with prepregnancy weight in the obese category showed a trend for less decline in FHR relative to both fetuses of normal and overweight women ($\beta = 3.77$, $SE = 2.15$, $t = 1.75$, $P=0.080$) between G2 and G3 (not shown). Fetal heart rate variability (Figure 1a) showed a less steep rate of development commencing at G2 for fetuses of obese women relative to those of normal weight ($\beta = -0.75$, $SE = 0.37$, $t = -2.06$, $P=0.040$) such that by G3 variability was significantly lower in the obese group ($P=0.024$). Differences between fetuses of overweight and normal weight women were not significant (P range = 0.261 to 0.609). A similar pattern of results was exhibited for accelerations. Figure 1b shows less gain in the number of accelerations per 50-minute interval in fetuses of obese women from G1 to G2 compared with normal weight ($\beta = -1.32$, $SE = 0.57$, $t = -2.31$, $P=0.022$) and overweight ($\beta = -1.26$, $SE = 0.65$, $t = -1.95$, $P=0.052$) groups. From G2 to G3, fetuses of obese women continued to show less gain in the number of accelerations relative to fetuses of normal weight women ($\beta = -1.31$, $SE = 0.62$, $t = -2.11$, $P=0.035$), but not compared with overweight women ($P=0.281$). By G3, fetuses of obese women exhibited significantly fewer accelerations relative to normal weight ($P=0.001$) and overweight groups ($P=0.025$).

3.2. Fetal motor activity

As with cardiac measures, no differences in fetal motor behavior were detected by maternal weight category at G1 (P range = 0.123 to 0.946) with the exception of a trend increase in movement vigor among fetuses of overweight women relative to the normal weight group ($\beta = -0.49$, $SE = 0.26$, $t = -1.88$, $P = 0.061$). Later in gestation, whereas differences in fetal cardiac measures were detected only in the obese category, motor development differed in both overweight and obese groups compared with the normal weight group. Figure 2a shows movement vigor (i.e. amplitude); the rate of change from G1 to G2 was significantly different between fetuses of women who were obese and those of normal weight ($\beta = 0.91$, $SE = 0.37$, $t = 2.46$, $P = 0.014$). By G2, fetuses of overweight ($P = 0.003$) and obese women ($P = 0.002$) showed significantly higher movement vigor compared with fetuses of normal weight women. Greater movement vigor in fetuses of overweight ($P = 0.020$) and obese ($P = 0.001$) women persisted at G3, although slopes from G2 to G3 were similar across BMI groups (P range = 0.582 to 0.832). Total fetal movement (not shown) showed no differences in the rate of change over time among BMI groups (P range = 0.145 to 0.788). At G2 fetuses of overweight and at a trend level of obese women, showed greater activity compared with fetuses of normal weight women ($P = 0.006$ and $P = 0.070$, respectively). Significantly higher motor activity among fetuses of overweight ($P = 0.007$) and obese ($P = 0.001$) women was also observed at G3.

3.3. Cardiac–somatic coupling

As with the other measures, cardiac–somatic coupling did not differ among groups at G1 (P range = 0.532 to 0.744). However, fetuses of women who were obese showed less increase in coupling from G1 to G2 relative to fetuses of normal weight women ($\beta = -0.04$, $SE = 0.02$, $t = -2.20$, $P = 0.028$); the rate of change was not significantly different between fetuses of normal weight and overweight women ($P = 0.112$, see Figure 2b). At G2, fetuses of overweight ($P = 0.012$) and obese ($P = 0.016$) women showed significantly less coupling than fetuses of normal weight women. At G3, fetuses of overweight women continued to show significantly less coupling ($P = 0.006$), but the significance level for fetuses of obese women declined to a trend level ($P = 0.059$). No slope differences were found from G2 to G3 (P range = 0.599 to 0.801).

3.4. Additional analyses

Both gestational diabetes ($n = 20$) and intrauterine growth restriction ($n = 7$) were not excluded from the sample but can have independent adverse effects on fetal neurodevelopment. Models for each fetal variable were re-run excluding these cases. This subset of women was disproportionately obese relative to the larger sample (51.9% normal weight, 22.2% overweight, and 25.9% obese; $\chi^2 = 5.41$, $P = 0.020$). Models revealed that although some associations with obesity and overweight were marginally reduced, the pattern of findings remained the same. Gestational weight gain was also examined as a separate factor and was entered as a predictor in all models. Gestational weight gain contributed no additional explanatory power above and beyond prepregnancy BMI (P range = 0.180 to 0.968).

4. Discussion

Maternal prepregnancy BMI categorized as overweight and obese was associated with dysregulated and delayed trajectories of fetal neurobehavior from mid-gestation to near term. Group differences in fetal cardiac and motor activity were not yet evident at the earliest assessment period (i.e. 24 weeks). However, by 36 weeks, fetuses of obese women showed decreased FHR variability and fewer accelerations. Differences emerged earlier for motor activity such that fetuses of both obese and overweight women moved more vigorously and were more active by the second assessment. Fetuses of women with higher prepregnancy BMI also showed less cardiac–somatic integration—a marker of maturation of the central nervous system [16,17] and predictive of postnatal infant neurobehavior [18–20].

Mechanisms of action for maternal excess prepregnancy weight on the fetus remain under speculation. A recent animal model suggests that a high fat diet in the pregnant female promotes production of leptin, insulin, and lipids that reach the fetal compartment and induce alterations in placental vasculature resulting in fetal hypoxia [21]. Similar alterations to the maternal endocrine milieu and placental function are noted in clinical populations of diabetic pregnant women [22,23].

Whereas differences in fetal cardiac measures, when detected, were observed only in obese women, differences in motor development were observed in both the overweight and obese groups. This suggests that motor disruptions in the fetus may be more sensitive to maternal metabolic and endocrine outputs. It is also possible that increased fetal motor activity may be a means of fetal self-regulation to a hyperglycemic environment, thereby mediating the relationship between maternal diabetes and infant birth weight [24].

Despite observations of associations between maternal BMI and fetal neuromaturation, no effects on infant outcomes were observed in this low-risk sample. This suggests that effects on fetal neuromaturation can occur in the absence of alterations to growth or the timing of delivery. This observation further supports the position that reliance on overly blunt outcomes such as birth weight and Apgar scores may obscure more subtle consequences exerted by an obesogenic intrauterine milieu.

The study is limited by reliance on maternal self-reporting of prepregnancy weight and height. This introduces nonrandom bias as people tend to report that they weigh less and are taller [25]. Nonetheless, measurement error in self-reporting would reduce our ability to detect significant associations and suggests that the actual associations may be stronger than detected here. Although the sample constitutes a relatively sociodemographically advantaged group, rates of overweight and obesity (22.5% and 11.8%, respectively) were comparable to national reports [3]. A sample with higher incidence of comorbid pre-existing or pregnancy conditions, such as excess gestational weight gain coupled with obese prepregnancy BMI, might be expected to show stronger associations.

In summary, findings demonstrate that maternal overweight and obesity during pregnancy is not without consequence for the developing fetal nervous system. These results have implications for weight management recommendations in women of childbearing age and clinical applications related to pregnancy management.

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Figure 1.

Unadjusted fetal heart rate variability (a) and heart rate accelerations (b) from the first to the third gestational period by maternal BMI category. Numbers by gestational period were 481, 535, and 506, respectively. Fetuses of obese women show decreased heart rate variation at G3 (36 weeks of gestation), indexed both continuously and episodically.

Figure 2.

Unadjusted fetal motor vigor (a) and cardiac-somatic coupling (b) from the first to the third gestational period by maternal BMI category. Numbers by gestational period were 481, 532, and 504, respectively, excluding select movement outliers at G2 and G3. Increased movement vigor is observed among fetuses of both overweight and obese women earlier in gestation, at G2 (30–32 weeks of gestation), as is decreased cardiac-somatic coupling. Differences were also observed again at G3 (36 weeks of gestation).

Table 1Maternal characteristics by prepregnancy body mass index (n=610).^{a,b}

Characteristic	Normal weight BMI 18.5–24.9 (n=401)	Overweight BMI 25.0–29.9 (n=137)	Obese BMI 30 or greater (n=72)	P value
Maternal age, y	30.9 ± 4.6	31.2 ± 5.1	31.1 ± 5.4	0.74
Education, y	16.9 ± 2.2	16.0 ± 2.3	15.7 ± 2.4	<0.001
Race/ethnicity (n=607) ^c				<0.001
White (n=474)	327 (69.0)	96 (20.2)	51 (10.8)	
African American (n=86)	37 (43.0)	32 (37.2)	17 (19.8)	
Asian (n=47)	35 (74.5)	8 (17.0)	4 (8.5)	
Nulliparous	291 (72.6)	83 (60.6)	39 (54.2)	0.001
Prenatal complications ^d	35 (8.7)	15 (10.9)	11 (15.3)	0.21
Gestational weight gain, kg	15.9 ± 4.7	15.2 ± 6.1	12.2 ± 7.9	<0.001
Cesarean delivery	101 (25.2)	56 (40.9)	20 (27.8)	0.003

^aBMI calculated as weight in kilograms divided by height in meters squared.^bData are presented as mean ± SD or number (percentage).^cThree cases of race/ethnicity were unreported.^dIncludes preterm labor and delivery, gestational diabetes, or hypertensive disorders.

Table 2Neonatal outcomes by prepregnancy body mass index.^{a,b}

Characteristic	Normal weight BMI 18.5–24.9 (n=401)	Overweight BMI 25.0–29.9 (n=137)	Obese BMI 30 or greater (n=72)	P value
Fetal sex, no. male	202 (50.4)	61 (44.5)	38 (52.8)	0.40
Gestational age at delivery, wk	39.2 ± 1.7	39.0 ± 1.5	39.4 ± 1.5	0.22
Birth weight, g	3386.6 ± 496.3	3408.0 ± 545.8	3411.4 ± 549.5	0.88
Apgar 1 min	8.0 ± 1.3	7.9 ± 1.5	7.9 ± 1.4	0.67
Apgar 5 min	8.9 ± 0.5	8.9 ± 0.5	8.8 ± 0.5	0.22
NICU observation	38 (9.5)	17 (12.4)	12 (16.7)	0.16

Abbreviation: NICU, neonatal intensive care unit.

^aBMI calculated as weight in kilograms divided by height in meters squared.^bData are presented as mean ± SD or number (percentage).