

Maternal occupational exposure to noise: prevalence, maternal effects and infant outcomes in the National Birth Defects Prevention Study, 1997–2011

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ABSTRACT

Objectives We investigated associations between occupational noise and gestational diabetes mellitus, pregnancy-related hypertension (including pre-eclampsia/eclampsia), preterm birth and small for gestational age (SGA) infants.

Methods Data were analysed for 7889 singleton, live-born infants without major birth defects or chromosomal disorders and their mothers from the National Birth Defects Prevention Study from 1997 to 2011. Typical maternal occupational noise exposure in all jobs held from 1 month prior to conception through the end of pregnancy was estimated by expert rater and categorised as quiet (<60A-weighted decibels (dBA)), low (60–75 dBA), moderate (76–84 dBA) or loud (>85 dBA). Multiple logistic regression was used to estimate associations (adjusted ORs and 95% CIs) between noise exposure levels and outcomes.

Results Approximately 77.4% of pregnant workers had quiet levels of occupational noise exposure, 11.0%, 10.1% and 1.5% had low, moderate and loud exposure levels, respectively. Compared with quiet levels of noise, pregnant workers exposed to low levels of noise had decreased odds of delivering an SGA infant (adjusted OR (aOR)=0.72; 95% CI 0.53 to 0.99) and those exposed to moderate levels had increased odds of delivering an SGA infant (aOR=1.37; 95% CI 1.05 to 1.77). No other significant associations were observed.

Conclusion Maternal occupational noise exposure below the 85 dBA threshold recognised as hazardous may be associated with SGA among infants. Elevated point estimates (>1) were observed for the highest noise exposure category and all outcomes, though CIs were wide and statistical significance was not attained. Further research is warranted to address existing knowledge gaps.

BACKGROUND

Occupational noise exposure remains a significant public health concern.¹ In addition to hearing loss, occupational noise has been reported to be associated with stress, sleep disturbance, impaired cognitive performance, hypertension, elevated cholesterol and coronary heart disease.^{2–4} While the mechanisms linking noise to these outcomes remain unclear, evidence suggests that noise affects

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Exposure to loud noise is associated with hearing loss. Both duration and intensity (as measured in A-weighted decibels (dBA)) increase risks of hearing loss. The Occupational Safety and Health Administration requires employers to implement a hearing conservation programme when noise exposure is >85 dBA, which is also the National Institute for Occupational Safety and Health recommended exposure limit, averaged over an 8-hour workday.
- ⇒ Epidemiologic studies have shown associations between chronic noise exposure and hypertension, elevated cholesterol and coronary heart disease even at levels below 85 dBA.
- ⇒ Occupational noise can be transmitted to a developing fetus, and maternal use of hearing protection does not reduce fetal noise exposure.
- ⇒ Studies of the association between occupational noise exposure and health among pregnant workers in the USA are limited.

the sympathetic nervous and endocrine systems, resulting in acute physiological responses (eg, heart rate, blood pressure and stress hormones) that may be prolonged with chronic noise exposure.^{4–6} The Occupational Safety and Health Administration requires employers to implement a hearing conservation programme when noise exposure is >85 A-weighted decibels (dBA), which is also the National Institute for Occupational Safety and Health recommended exposure limit (REL), averaged over an 8-hour workday.

Studies of noise exposure at levels below permissible and RELs in the USA (<90 dBA and <85 dBA, respectively) have demonstrated such physiological responses, including increased release of adrenal hormones, below these occupational exposure limits.^{7,8} This could explain maternally mediated mechanisms of health effects on a pregnancy. Alternatively, because noise can be transmitted to the developing fetus irrespective of maternal hearing protection, loud noise could also have direct effects on the fetus once fetal hearing is developed. Epidemiologic investigations of noise exposure



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WHAT THIS STUDY ADDS

- ⇒ We assessed probable occupational noise exposure during pregnancy among a large population-based sample of women giving birth to singleton, live-born infants in the USA who did not have chromosomal disorders, structural birth defects or congenital syndromes apparent at birth.
- ⇒ Approximately 11.6% of participating pregnant workers had probable exposure to noise levels ≥ 76 dBA during the majority of work hours, including 1.5% exposed to levels ≥ 85 dBA.
- ⇒ Pregnant workers estimated to have probable exposure to occupational noise between 76 and 84 dBA were at increased odds of delivering an infant characterised as small for gestational age, compared with pregnant workers with probable exposure to the lowest levels of occupational noise (< 60 dBA).
- ⇒ US-born Hispanic mothers aged 26–34 years were significantly more likely to work jobs with probable exposure to the loudest (≥ 85 dBA) occupational noise.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Occupational noise exposure between 76 and 84 dBA is below the threshold recognised as hazardous (85 dBA) but might adversely impact fetal growth.
- ⇒ Observed differences in probable occupational exposure to elevated noise levels by maternal race/ethnicity highlight the need to consider disparities in occupational environmental conditions as a potential contributor to disparities in maternal and fetal morbidity.

in pregnancy have shown associations with adverse maternal conditions during pregnancy such as gestational diabetes mellitus (GDM) and pregnancy-related hypertensive disorders (eg, pre-eclampsia, a pregnancy-specific disorder on the hypertensive spectrum characterised by hypertension, high levels of protein in the urine or other signs of organ damage).^{9–13} However, previous studies are limited and have been conducted predominantly in non-US working populations.¹³ Some studies have also demonstrated potential associations with adverse birth outcomes, such as increased risk of low birth weight, preterm birth (PTB) and small for gestational age (SGA), though results are inconsistent.¹⁴

Additional studies examining the health impacts of occupational noise exposure occurring during pregnancy, as well as the distribution of workplace noise exposure among pregnant workers, are indicated by existing knowledge gaps. The objectives of this study were to (1) characterise the prevalence of probable occupational noise exposure among pregnant workers and (2) investigate associations between occupational noise exposure and adverse pregnancy (GDM, pregnancy-related hypertension (with or without pre-eclampsia/eclampsia) and birth outcomes (SGA and PTB)) in a large sample of pregnant workers in the USA from 1997 to 2011.

METHODS**Study population**

We examined data from the National Birth Defects Prevention Study (NBDPS), a large population-based case-control study designed primarily to investigate risk factors for major structural birth defects.¹⁵ The NBDPS included study sites in Arkansas,

California, Georgia, Iowa, Massachusetts, New Jersey, New York, North Carolina, Texas and Utah. Some sites only included select counties, and not all study sites contributed data for all the study years (1997–2011). Randomly selected live-born infants without a chromosomal or major structural birth defect were identified from birth records or hospital delivery records and recruited as population-based controls from the same catchment areas as cases. Control infants have been found to be generally representative of infants from the NBDPS catchment area,¹⁶ and they have been used as a birth cohort by others to study pregnancy-related risk factors (eg, nutrient intake) and outcomes (eg, SGA and PTB).^{17,18} The current study used a ‘control-only’ mother–infant cohort, and the study population consisted of mothers and their live-born infants without a birth defect(s) with delivery on/after 1 October 1997 and estimated date of delivery on/before 31 December 2011. The NBDPS is one of the very few large, population-based studies in the USA that contains both necessary components—including health data and job history data—for associations between occupational noise exposure and select maternal and birth outcomes to be investigated.

Detailed methodology has been previously reported,¹⁵ but in brief, data were available on all mothers of infants without birth defects who participated in the NBDPS computer-assisted telephone interview (CATI) in either English or Spanish (65% response among eligible mothers of infants without birth defects). Our study sample included mothers who reported employment for at least one calendar month during the time period of 1 month prior to conception to date of birth (B_1 – P_{end}) and provided sufficient job history information to enable the assessment of maternal exposure to occupational noise ($n=8216$). The exposure window included the month prior to conception to account for influences during this vulnerable period, which includes egg maturation and release, as well as other critical processes of fetal development. Because pregestational diabetes and multiple pregnancies are strong risk factors for all study outcomes of interest, NBDPS participant mothers with type 1 or 2 diabetes diagnosed before the index pregnancy (ie, pregestational diabetes) ($n=54$) as well as non-singleton pregnancies ($n=266$) were excluded from all analyses. In addition, infants with ambiguous or unreported sex at birth ($n=4$) and those born outside the range of 22–44 weeks’ gestation ($n=3$) were also excluded as outcome measures could not be assessed.

Exposure assessment

Participating mothers self-reported the following occupational information during the NBDPS interview: company name, job title, goods or services produced and main job activities and duties. All responses were recorded as open free text for each job held for at least one calendar month during the exposure period (B_1 – P_{end}). Mothers could report multiple jobs. A team of occupational epidemiologists and industrial hygienists (IHs) used job history data to assign standard codes from the 2000 Standard Occupational Classification (SOC) System for occupation and the 1997 North American Industry Classification System (NAICS) for industry.

Assigned industry and occupation codes (ie, NAICS and SOC codes), as well as keywords derived from narrative job variables, were used to screen all reported jobs ($n=9407$) held during the exposure period (B_1 – P_{end}) to identify and flag an initial set of jobs with potential for occupational noise exposure of > 60 dBA. Flagging criteria (including NAICS/SOC codes and keywords) were identified from population surveys of workers, including the Occupational Requirements Survey,¹⁹ studies of occupational

hearing loss,²⁰ studies that used job-exposure matrix data from multiple sources²¹ and expert opinion of coauthors. A total of 2823 flagged jobs (ie, those jobs initially identified with potential noise exposure >60 dBA) held by 2577 participant mothers were then sent to an expert IH rater, who assigned a final occupational noise category rating of either 'very loud,' 'loud,' 'moderate,' 'low' or 'quiet' noise levels of exposure for the majority of work hours. Categorical noise levels were estimated to correspond to quantitative decibel ranges (>95 dBA = 'very loud' noise; 85–95 dBA = 'loud' noise; 76–84 dBA = 'moderate' noise; 60–75 dBA = 'low' noise; <60 dBA = 'quiet' noise levels). Occupational noise categories were determined via subject matter expertise and align with similar studies of occupational noise exposure and reproductive outcomes.¹⁴

Jobs that were not initially flagged for further exposure assessment during the initial screening process (n=6584) were assumed to have occupational noise exposure levels below 60 dBA. Few (4.9%; 386/7889) mothers started, stopped or changed jobs in the study period (B_1 – P_{end}). Mothers were assigned to the noise exposure category that represented the highest number of working days during the exposure window (B_1 – P_{end}) when they were not continuously employed in a single job throughout the entire exposure window (eg, reported working multiple jobs or quit working at some point during the pregnancy) in order to prioritise exposure duration throughout pregnancy in absence of a known period of susceptibility.

Outcome classification

Maternal health outcomes of GDM and pregnancy-related hypertension (with or without pre-eclampsia/eclampsia) were assessed from self-report via the NBDPS CATI Questionnaire. GDM was defined as gestational diabetes diagnosed during the index pregnancy among those with no prior diabetes diagnosis. Pregnancy-related hypertension (with or without pre-eclampsia/eclampsia) was defined as the occurrence of high blood pressure during the index pregnancy among those with no prior history of high blood pressure and no use of medications to treat high blood pressure. Data used to calculate pregnancy-related hypertension were limited to mothers with deliveries beginning in 2006, due to a change in the interview instrument making it possible to distinguish between chronic hypertension occurring within the index pregnancy and pregnancy-related hypertension.

Fetal growth restriction was measured by SGA, defined by sex-specific birth weight below the 10th percentile for a given gestational age among deliveries occurring between 22 and 44 gestational weeks using a US-based birth weight for gestational age reference reported by Talge *et al.*²² PTB was classified as birth occurring prior to 37 gestational weeks.²³

Covariates

Descriptive statistics were generated for mothers using the assigned category of occupational noise exposure. P values were calculated using χ^2 tests to assess differences and considered significant if $p < 0.05$. Potential confounders were identified through directed acyclic graphs²⁴ and supported by a priori knowledge of previous literature of factors known to influence maternal occupational exposure to noise and all study outcomes of interest.^{25–29} Covariates considered were based on status at the time of infant delivery unless otherwise stated, and included: maternal residence (NBDPS study location), age (≤ 20 years, 21–25 years, 26–34 years, ≥ 35 years), race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, non-Hispanic another race), nativity (US born, foreign born), education (≤ 12

years, >12 years), maternal pre-pregnancy body mass index (BMI) (underweight, <18.5 kg/m²; normal weight, 18.5 to ≤ 24 kg/m²; overweight, 25 to <30 kg/m²; obese, >30 kg/m²), smoking (any use during early pregnancy, none), alcohol use (any use during early pregnancy, none) and secondhand smoke exposure (any exposure from home and/or work during early pregnancy, none). 'Early pregnancy' was defined as exposure occurring anytime from 1 month prior to conception through the first trimester.

Multiple logistic regression was used to estimate the association between maternal categorical occupational noise exposure ('low' noise (60–75 dBA); 'moderate' noise (76–84 dBA); 'loud' noise (≥ 85 dBA)) compared with quiet (<60 dBA) during the exposure period (B_1 – P_{end}) and all maternal and infant health outcomes under study. Due to small cell size in the highest exposure group ($n \leq 5$), the two highest exposure groups ('loud'=85–95 dBA; 'very loud' ≥ 95 dBA) were combined throughout. A Cochran-Armitage trend analysis was also conducted to examine if there was a significant dose-response effect by increasing categorical noise exposure with each binary outcome of interest. All statistical analyses were performed using SAS V.9.4 and independently replicated by a second analyst.

RESULTS

The final study population consisted of 7889 eligible pregnant workers and their infants. Approximately 1.5% ($n = 117/7889$) of pregnant workers were deemed to have 'loud' noise exposure (≥ 85 dBA); 10.1% ($n = 795/7889$) to have 'moderate' noise exposure (76–84 dBA); 11.0% ($n = 867/7889$) to have 'low' noise exposure (60–75 dBA); and 77.4% ($n = 6110/7889$) were deemed to have exposure to 'quiet' (<60 dBA) levels of occupational noise during the majority of working hours at their jobs.

Table 1 reports the distribution of occupational noise categories among the major occupational groups (SOC) held by pregnant workers in this population-based study; although there are 23 major SOC groups, we collapsed those with very few or no representatives in any noise exposure category ('low', 'moderate' and 'loud') among our population into a single 'other occupations' category (collectively representing <4% of exposed study participants). The majority of participant mothers with probable exposure to the loudest categorical noise level (≥ 85 dBA) were employed in Transportation and Material Moving occupations (15.3%) and Farming, Fishing, and Forestry occupations (23.9%).

Table 2 reports the distribution of occupational noise category for mothers by select maternal socioeconomic and demographic variables. Maternal occupational exposure to categorical levels of noise varied significantly by state of residence, age, race/ethnicity, education, nativity, secondhand smoke exposure, as well as self-reported smoking and alcohol use in early pregnancy. Compared with participant mothers with 'quiet' occupational noise exposure (<60 dBA), mothers exposed to 'loud' levels of occupational noise (≥ 85 dBA) were generally younger (14.5% <20 years vs 8.4% <20 years, respectively), had lower reported levels of educational attainment (62.1% with <12 years vs 29.9% with <12 years, respectively), were less likely to be born in the USA (57.8% US born vs 83.9% US born, respectively) and reported a higher prevalence of secondhand smoke exposure during early pregnancy (34.2% vs 22.5%, respectively). Significant differences in maternal race/ethnicity were also observed, with Hispanic mothers most likely to be exposed to the loudest (≥ 85 dBA) levels of occupational noise. There

Table 1 Distribution of probable noise exposure category by major SOC groups among jobs held during periconception and pregnancy (B₁–P_{end}) by mother of live-born infants without malformations, * National Birth Defects Prevention Study, USA, 1997–2011 (n=7889)

	Quiet <60 dBA n=6110 (77.4)	Low noise 60–75 dBA n=867 (11.0)	Moderate noise 76–84 dBA n=795 (10.1)	Loud noise† ≥85 dBA n=117 (1.5)
SOC Major Group	n (%)‡	n (%)‡	n (%)‡	n (%)‡
Food Preparation and Serving Related Occupations (n=744)	121 (16.3)	33 (4.4)	582 (78.2)	8 (1.1)
Educational Instruction and Library Occupations (n=739)	342 (46.3)	389 (52.6)	7 (0.9)	1 (0.1)
Personal Care and Service Occupations (n=489)	259 (53.0)	214 (43.8)	14 (2.9)	2 (0.4)
Sales and Related Occupations (n=928)	735 (79.2)	80 (8.6)	113 (12.2)	0 (0.0)
Office and Administrative Support Occupations (n=1480)	1405 (94.9)	53 (3.6)	20 (1.4)	2 (0.1)
Healthcare Practitioners and Technical Occupations (n=665)	613 (92.2)	39 (5.9)	0 (0.0)	13 (2.0)
Building and Grounds Cleaning and Maintenance Occupations (n=229)	190 (83.0)	16 (7.0)	20 (8.7)	3 (1.3)
Transportation and Material Moving Occupations (n=157)	120 (76.4)	5 (3.2)	8 (5.1)	24 (15.3)
Production Occupations (n=323)	289 (89.5)	7 (2.2)	7 (2.2)	20 (6.2)
Farming, Fishing, and Forestry Occupations (n=117)	84 (71.8)	4 (3.4)	1 (0.9)	28 (23.9)
Other Occupations§ (n=2018)	1952 (96.7)	27 (1.3)	23 (1.1)	16 (0.8)

*Infants who did not have chromosomal disorders, structural birth defects or congenital syndromes apparent at birth.

†Noise exposure levels of 'loud' and 'very loud' were combined due to small cell size.

‡Percentages are calculated across rows to illustrate variation in estimated noise exposure within occupational groups, as indicated by the individual job description narratives.

§Other occupations include those in Management occupations, Business and Financial Operations occupations, Computer and Mathematical occupations, Architecture and Engineering occupations, Life, Physical, and Social Science occupations, Community and Social Service occupations, Legal occupations, Arts, Design, Entertainment, Sports, and Media occupations, Healthcare Support occupations, Protective Service occupations, Construction and Extraction occupations, Installation, Maintenance, and Repair occupations, and Military Specific occupations.

B1, one month prior to conception; dBA, A-weighted decibel; P_{end}, estimated date of delivery; SOC, Standard Occupational Classification.

were no significant differences in categorical BMI between noise exposure groups (data not shown).

Associations between maternal occupational noise exposure and maternal and infant health outcomes are reported in [table 3](#). No significant associations were observed between categorical occupational noise exposure and GDM or pregnancy-related hypertension, even after adjustment for potential confounders (maternal education, maternal residence, maternal age and maternal race/ethnicity). Pregnant workers exposed to 'moderate' levels (76–84 dBA) of occupational noise had a significantly increased likelihood of delivering an infant characterised as SGA (adjusted OR (aOR)=1.37; 95% CI 1.05 to 1.77), compared with pregnant workers exposed to quiet levels of workplace noise (<60 dBA). Protective effects were observed between mothers with 'low' noise exposure (60–75 dBA) and SGA (aOR=0.72; 95% CI 0.53 to 0.99), compared with mothers exposed to quiet levels of workplace noise (<60 dBA). In a trend analysis across all three categorical noise levels, categorical increases in noise were associated with increasing odds of delivering an SGA infant (p value<0.05). No significant associations were observed between categorical occupational noise exposure and PTB, even after adjustment for potential confounders.

DISCUSSION

We investigated associations between expert-rated occupational exposure to noise in periconception and pregnancy and both maternal complications (GDM and pregnancy-related hypertension (with or without pre-eclampsia/eclampsia)) and adverse birth outcomes (SGA and PTB) in a population-based sample of US women giving birth from 1997 to 2011 to live, singleton infants without major structural or chromosomal disorders. Because studies of occupational noise exposure among pregnant workers are limited, we also characterised occupational noise exposure in pregnancy/periconception by select maternal socioeconomic and demographic variables.

Sample sizes were limited for women exposed to loud noise at work and may have impacted the power to detect associations, so caution should be used in interpreting findings for this group. However, we noted that point estimates for all adverse outcomes (GDM, pregnancy-related hypertension, SGA and PTB) were >1 for the highest category of noise exposure, though these associations were subject to several limitations: (1) findings for GDM were heavily attenuated by adjustment for covariates; (2) sample sizes were small and CIs were wide and (3) other workplace covariates (such as changes in tasks during pregnancy or co-occurring workplace exposures) were not able to be controlled for. In addition, we observed a statistically significant trend across categorical increases in noise exposure levels associated with increasing odds of delivering an SGA infant (crude p trend<0.001; adjusted p trend=0.003), suggesting that additional research among pregnant women exposed to loud noise (≥85 dBA) is likely warranted.

Our results suggest pregnant workers with probable occupational noise exposure at a 'moderate' (76–84 dBA) level were at increased odds of delivering an infant characterised as SGA (aOR=1.37; 95% CI 1.05 to 1.77) compared with women with probable exposure to 'quiet' levels of occupational noise (<60 dBA) at their jobs. This is particularly noteworthy because this effect was observed at levels below occupational exposure limits. This result is also consistent with findings from Selander *et al* among full-time employed pregnant workers with low absence from work exposed to occupational noise levels between 75 and 84 dBA and SGA infants (aOR=1.09; 95% CI 1.02 to 1.17),³⁰ though we were unable to differentiate among full-time workers or workplace absenteeism in our study. Interestingly, our study also found a protective effect between maternal occupational exposure to 'low' (60–75 dBA) levels of noise and SGA among infants, compared with women estimated to work in quiet jobs (<60 dBA; aOR=0.72; 95% CI 0.53 to 0.99). This finding might reflect other factors varying between very quiet (eg, similar noise

Table 2 Distribution of select maternal characteristics by categorical occupational noise exposure, National Birth Defects Prevention Study, USA, 1997–2011 (n=7889)

	Quiet <60 dBA (n=6110)	Low noise 60–75 dBA (n=867)	Moderate noise 76–84 dBA (n=795)	Loud noise* ≥85 dBA (n=117)
	nt (%)	nt (%)	nt (%)	nt (%)
Maternal residence at delivery†				
Arkansas	749 (12.3)	115 (13.3)	139 (17.5)	14 (12.0)
California	550 (9.0)	77 (8.9)	78 (9.8)	31 (26.5)
Georgia	691 (11.3)	95 (11.0)	83 (10.4)	8 (6.8)
Iowa	794 (13.0)	123 (14.2)	93 (11.7)	15 (12.8)
Massachusetts	859 (14.1)	92 (10.6)	71 (8.9)	6 (5.1)
New Jersey	335 (5.5)	36 (4.2)	21 (2.6)	NR
New York	566 (9.3)	81 (9.3)	64 (8.1)	4 (3.4)
North Carolina	509 (8.3)	71 (8.2)	81 (10.2)	13 (11.1)
Texas	508 (8.3)	91 (10.5)	106 (13.3)	11 (9.4)
Utah	549 (9.0)	86 (9.9)	59 (7.4)	13 (11.1)
Maternal age at delivery†				
≤20 years	514 (8.4)	83 (9.6)	266 (33.5)	17 (14.5)
21–25 years	1391 (22.8)	215 (24.8)	291 (36.6)	38 (32.5)
26–34 years	3229 (52.8)	466 (53.7)	198 (24.9)	48 (41.0)
≥35 years	976 (16.0)	103 (11.9)	40 (5.0)	14 (12.0)
Maternal race/ethnicity†				
Non-Hispanic white	3917 (64.1)	588 (67.8)	424 (53.3)	44 (37.6)
Non-Hispanic black	698 (11.4)	109 (12.6)	103 (13.0)	10 (8.5)
Hispanic	1099 (18.0)	125 (14.4)	217 (27.3)	54 (46.2)
Non-Hispanic another race	396 (6.5)	45 (5.2)	51 (6.4)	9 (7.7)
Maternal nativity†				
US born	5119 (83.9)	792 (91.3)	624 (78.6)	67 (57.8)
Foreign born	984 (16.1)	75 (8.7)	170 (21.4)	49 (42.2)
Maternal education†				
≤12 years	1823 (29.9)	210 (24.3)	528 (66.5)	72 (62.1)
>12 years	4277 (70.1)	655 (75.7)	266 (33.5)	44 (37.9)
Maternal smoking during early pregnancy†				
Yes	1055 (17.3)	123 (14.2)	289 (36.4)	28 (23.9)
No	5054 (82.7)	744 (85.8)	505 (63.6)	89 (76.1)
Maternal alcohol use during early pregnancy†				
Yes	2641 (43.3)	332 (38.4)	316 (40.0)	48 (41.0)
No	3453 (56.7)	532 (61.6)	474 (60.0)	69 (59.0)
Maternal secondhand smoke exposure during early pregnancy†				
Yes	1373 (22.5)	162 (18.7)	396 (50.0)	40 (34.2)
No	4721 (77.5)	704 (81.3)	396 (50.0)	77 (65.8)

Early pregnancy: exposure anytime during the period of time from 1 month prior to conception through the first trimester, NR: not reportable based on n<3.
 *Noise exposure levels of 'loud' and 'very loud' were combined due to small cell size.
 †Frequencies may not combine to sample totals where there are missing values (<5% for all categories).
 ‡p value for $\chi^2 < 0.05$.
 dBA, A-weighted decibel.

level to a quiet office or library) versus low-noise jobs (eg, similar to normal conversation or household appliances running) or that low background noise may have some benefits to health³¹—though further research is needed.

The prevalence of working during pregnancy in our sample was similar to US national estimates during the study period (69% vs 66%, respectively).³² Despite study limitations, our findings related to SGA and moderate levels of occupational noise were consistent with Selander *et al*,³⁰ suggesting that maternal noise exposure in the workplace might influence fetal development in utero. Given the potential lifelong impact of babies being born too small,³³ as well as inconsistent evidence in the

literature regarding occupational noise exposure and maternal/birth outcomes, this topic merits further study.

In describing maternal occupational noise exposure among a sample of pregnant US workers, we identified notable differences in maternal race/ethnicity, as well as maternal education. Hispanic mothers, as well as those with lower educational attainment (≤12 years), were determined to be disproportionately exposed to jobs with occupational noise exposure >85 dBA. Though differences were modest, NBDPS control participants were more likely than their base populations to be non-Hispanic white mothers with more than a high school education¹⁶; therefore, it's possible that we observed a lower-than-expected

Table 3 Association between maternal categorical occupational exposure to noise during pregnancy (B_1 – P_{end}) and gestational diabetes mellitus (GDM) and pregnancy-related hypertension (with or without pre-eclampsia/eclampsia) among mothers and small for gestational age (SGA) and preterm birth (PTB) among infants without birth defects, * National Birth Defects Prevention Study, USA, 1997–2011

GDM	n†	Crude OR	(95% CI)	Crude p trend	Adjusted OR‡	(95% CI)	Adjusted p trend‡
Quiet (<60 dBA)	279	1.0	(Ref)	0.099	1.0	(Ref)	0.447
Low noise (60–75 dBA)	29	0.73	(0.50 to 1.08)		0.77	(0.52 to 1.14)	
Moderate noise (76–84 dBA)	31	0.88	(0.61 to 1.29)		0.99	(0.67 to 1.48)	
Loud noise (≥85 dBA)§	7	1.39	(0.64 to 3.00)		1.04	(0.47 to 2.29)	
Pregnancy-related hypertension (with or without pre-eclampsia/eclampsia)¶	n†	Crude OR	(95% CI)	Crude p trend	Adjusted OR**	(95% CI)	Adjusted p trend**
Quiet (<60 dBA)	196	1.0	(Ref)	0.256	1.0	(Ref)	0.400
Low noise (60–75 dBA)	34	1.12	(0.77 to 1.64)		1.13	(0.77 to 1.65)	
Moderate noise (76–84 dBA)	31	1.08	(0.73 to 1.60)		1.02	(0.68 to 1.54)	
Loud noise (≥85 dBA)§	6	1.60	(0.67 to 3.80)		1.61	(0.67 to 3.89)	
SGA	n†	Crude OR	(95% CI)	Crude p trend	Adjusted OR‡	(95% CI)	Adjusted p trend‡
Quiet (<60 dBA)	412	1.0	(Ref)	<0.001	1.0	(Ref)	0.003
Low noise (60–75 dBA)	45	0.70	(0.51 to 0.95)		0.72	(0.53 to 0.99)	
Moderate noise (76–84 dBA)	86	1.71	(1.34 to 2.18)		1.37	(1.05 to 1.77)	
Loud noise (≥85 dBA)§	13	1.66	(0.93 to 2.98)		1.64	(0.91 to 2.97)	
PTB	n†	Crude OR	(95% CI)	Crude p trend	Adjusted OR**	(95% CI)	Adjusted p trend**
Quiet (<60 dBA)	453	1.0	(Ref)	0.171	1.0	(Ref)	0.240
Low noise (60–75 dBA)	68	1.03	(0.80 to 1.35)		1.04	(0.80 to 1.35)	
Moderate noise (76–84 dBA)	69	1.17	(0.90 to 1.52)		1.03	(0.78 to 1.35)	
Loud noise (≥85 dBA)§	12	1.39	(0.76 to 2.55)		1.36	(0.74 to 2.51)	

Bold values indicate statistically significant finding.

*Analytic sample excludes participant mothers who were missing a response for maternal education.

†Exposed pregnant workers, among those that experienced the pregnancy/birth outcome noted.

‡Model adjusted for maternal residence at delivery (National Birth Defects Prevention Study site), maternal education (referent≥12 years), maternal age (referent=26–34 years) and maternal race (referent=non-Hispanic white).

§Noise exposure levels of 'loud' and 'very loud' were combined due to small cell size.

¶Analysis limited to mothers with estimated dates of delivery between 2006 and 2011.

**Model adjusted for maternal residence at delivery, maternal education (referent≥12 years) and maternal race (referent=non-Hispanic white).

B_1 , 1 month prior to conception; dBA, A-weighted decibel; P_{end} , estimated date of delivery.

prevalence of maternal occupational noise exposure among our study sample, particularly among the loudest noise levels. Observed differences across maternal demographics and socio-economic characteristics may help identify those working populations at increased risk of exposure to louder levels of occupational noise.

This study has several strengths and limitations. A major limitation of this study is that we relied on retrospective exposure assessment, as opposed to real-time quantitative measurements. This may have contributed to non-differential misclassification bias and residual confounding effects on the overall association between occupational noise exposure and SGA among infants, even after model adjustments. Capturing real-time measurements from pregnant workers, however, would require a prospective study of pregnancy. Because we could not measure or adjust for all potentially relevant variables, we cannot rule out the possibility of residual confounding that may have influenced study findings. Additionally, the response rate among NBDPS controls was approximately 65%,¹⁵ which could be influenced by differential participation that has the potential to bias effect estimates; however, it is unlikely that non-response among controls differed by job type based on retrospective exposure assessment of occupational noise.

The birth outcomes in our study were infrequent (<10% of live births) and most recent US prevalence estimates for both PTB and SGA are approximately 10% and 11%, respectively.^{34 35} Therefore, we believe prospective studies would be challenging to conduct

with an adequate sample size, and why we feel studies like ours are important to do even with imperfect measures. Even in as large a sample size as our study, maternal occupational exposure to 'loud' and 'very loud' (≥85 dBA) noise among pregnant workers was still rare among the general population, leading to suboptimal statistical power. Future studies focusing on occupations with loud (≥85 dBA) noise exposure and pregnancy would likely be beneficial to exploring this association and determining whether a dose–response relationship truly exists. In addition, we did not have sufficient data to predict the effects of impulsive noise (very brief and typically at a very high noise level).

Both GDM and pregnancy-related hypertension are commonly diagnosed after 20 weeks' gestation. We had originally planned sensitivity analyses to evaluate time-to-event data or test different exposure windows. However, we found that it was uncommon for participants to start, stop or change jobs during the study period. When women changed jobs, they also tended to move into similar jobs—thus only 4.9% of participant mothers changed exposure category at any time over the pregnancy and periconceptional period (data not shown), suggesting that the impact of modelling exposure up to the time of diagnosis (vs at any time during pregnancy) would likely be negligible.

The NBDPS provides a unique opportunity to examine the influence of a wide range of occupational exposures on reproductive outcomes. Study participants were drawn from a large population-based case–control study (NBDPS) with enrolment

from 10 states across the USA. As such, control mother–infant pairs were typically representative of the study region in which they were collected, leading to increased overall generalisability of findings.^{15 16} Similarly, because the study sample was limited to only participants reporting at least one job for at least 1 month during pregnancy, factors observed to be different between working and non-working pregnant women likely had a minimal influence in this study's overall findings. Though occupational noise exposure levels were not quantitatively measured in real time, they were determined by an expert IH rater and not collected via self-report, reducing the potential for recall bias. Compared with using a job-exposure matrix, our approach was also able to incorporate individual experiences in their job narrative to account for differences in work experience.

To the best of our knowledge, this study is the first to characterise and describe occupational noise exposure among a large sample of pregnant workers in the USA. The significant study finding of an association between maternal occupational exposure to 'moderate' (76–84 dBA) noise levels and increased SGA among infants, as well as the protective finding associated with maternal exposure to 'low' (60–75 dBA) noise levels, suggests that occupational noise exposure below 85 dBA warrants further study. In addition, observed differences between noise exposure levels, particularly by maternal race/ethnicity and education, highlight the need to address disparities in occupational exposure as a potential contributing factor towards disparities in maternal and infant health outcomes.

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Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants. All interviewed study participants provided informed consent. The study protocols were approved by the human subjects review board for the Centers for Disease Control and Prevention (protocol #2087) and the institutional review board for each participating site, and all participating mothers provided informed consent (see 45 C.F.R. part 46; 21 C.F.R. part 56). Participants gave informed consent to participate in the study before taking part.

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Data availability statement Data are available on reasonable request. The study questionnaires and process for accessing the data used in this study are described at <https://www.cdc.gov/ncbddd/birthdefects/nbdps-public-access-procedures.html>. The code book and analytic code may be made available on request.

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