ORIGINAL ARTICLE

Maternal occupational exposure to polycyclic aromatic hydrocarbons and small for gestational age offspring

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ABSTRACT

Objectives While some of the highest maternal exposures to polycyclic aromatic hydrocarbons (PAHs) occur in the workplace, there is only one previous study of occupational PAH exposure and adverse pregnancy outcomes. We sought to extend this literature using interview data combined with detailed exposure assessment.

Methods Data for 1997–2002 were analysed from mothers of infants without major birth defects in the National Birth Defects Prevention Study, a large population-based case-control study in the USA. Maternal telephone interviews yielded information on jobs held in the month before conception through delivery. From 6252 eligible control mothers, 2803 completed the interview, had a job, met other selection criteria, and were included in the analysis. Two industrial hygienists independently assessed occupational exposure to PAHs from the interview and reviewed results with a third to reach consensus. Small for gestational age (SGA) was the only adverse pregnancy outcome with enough exposed cases to yield meaningful results. Logistic regression estimated crude and adjusted ORs.

Results Of the 2803 mothers, 221 (7.9%) had infants who were SGA. Occupational PAH exposure was found for 17 (7.7%) of the mothers with SGA offspring and 102 (4.0%) of the remaining mothers. Almost half the jobs with exposure were related to food preparation and serving. After adjustment for maternal age, there was a significant association of occupational exposure with SGA (OR=2.2, 95% CI 1.3 to 3.8).

Conclusions Maternal occupational exposure to PAHs was found to be associated with increased risk of SGA offspring.

What this study adds

- While some of the highest maternal exposures to polycyclic aromatic hydrocarbons (PAHs) occur in the workplace, there is only one previous study of occupational PAH exposure and adverse pregnancy outcomes. We sought to extend this literature using control data from a large multisite birth defects study.
- Almost half the jobs with occupational PAH exposure were related to food preparation and serving. There was a significant association of occupational exposure with small for gestational age offspring (OR=2.2, 95% CI 1.3 to 3.8). However, there was no clear exposure-response relationship.

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are lipophilic compounds formed during the incomplete burning of coal, tobacco or other organic substances. Humans are exposed by smoke from tobacco and other sources; by ambient air pollution; or by consuming PAHs in food, especially in charbroiled foods.[1] While environmental sources contribute to the total exposure burden, some of the highest exposure levels are found in the workplace.[2 3] Occupational exposures can occur in common workplace settings such as restaurants.[4]

PAHs and PAH-DNA adducts have been found in placental tissues of women, amniotic fluid samples and umbilical cord blood samples from newborns.[5–10] PAH adducts can disrupt the cell's microenvironment,[1 11] to which the rapidly growing and differentiating cells of the fetus may be particularly vulnerable.[12] PAHs may also lead to periods of fetal hypoxia through reduced placental blood flow.[13] PAHs have been shown in lab animals to be reproductive toxicants, causing fetal death and low fetal weight.[14] Information on reproductive toxicity in humans is somewhat sparse. Maternal exposure to PAHs has been associated with low birth weight, preterm birth or intrauterine growth restriction, whether based on a job exposure matrix using job title and workplace,[15] stationary air monitoring[16] or personal air monitoring.[11 17 18] Newborns with high levels of PAH-DNA adducts in umbilical cord blood or placental samples had significantly decreased birth length, weight or head circumference,[19 20] however the evidence for this association is equivocal.[7 21]

Occupational exposures are often higher than environmental exposures and may be more amenable to intervention. However, only one study was found that specifically examined the association of occupational exposure to PAHs and adverse birth outcomes; it reported a significant decrease in fetal

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weight but not fetal head circumference or fetal length.[15] The objective of the current study was to extend current knowledge by examining small for gestational age (SGA) among controls from a large ongoing case-control study of birth defects. Although preterm birth and term low birth weight were also initially considered as outcomes, their exposed sample size was too small.

METHODS

Study population

This analysis used data from the National Birth Defects Prevention Study (NBDPS), an ongoing, population-based, case-control study of structural birth defects. Detailed study methods have been published elsewhere.[22] Only control infants (without major structural birth defects) were used for this analysis. They were live-born, and were selected at random from birth certificates or birth hospital records in eight sites (Arkansas, California, Georgia, Iowa, Massachusetts, New Jersey, New York and Texas). More precisely, they were selected using software to randomly identify a subset of births from lists (of birth certificate numbers or projected deliveries in hospitals) that would reflect the number of births in that facility and month. If a selected birth turned out to have any birth defect, the next immediately following birth was selected instead. All mothers participated in a computer-assisted telephone interview (CATI) in English or Spanish, administered from 6 weeks through 24 months after their estimated due dates. Mothers were asked questions on a variety of topics including demographics, maternal illnesses and medication use, vitamin intake, tobacco use and information about jobs held for 1 month or more during preconception and pregnancy. The NBDPS and its informed consent procedures were approved by the Office of Management and Budget, and the appropriate institutional review boards at the Centers for Disease Control and Prevention (CDC) and at each participating site.

The study population for the current analysis started with 6252 mothers of NBDPS control infants with estimated dates of delivery from October 1, 1997 through December 31, 2002, since that was the period for which the exposure assessment described below had been conducted. Of those, 4139 completed all or part of the interview, leading to a participation rate of 66.2%. Selecting mothers who held a job for at least 1 month during the pregnancy and the month before conception narrowed the number to 2937. From that total, 134 subjects were excluded using the following criteria: (A) infants from plural births; with gestational ages greater than 42 weeks; or with missing gestational age, birth weight or sex; (B) mothers with missing parity information or with prepregnancy diabetes. That resulted in 2803 mothers.

We applied published sex-specific, race/ethnic-specific and parity-specific growth curves (Zhang and Bowes,[23] and Overpeck *et al*[24]) to our study population and defined infants as SGA if their birth weight for gestational age was less than the 10th centile for the corresponding stratified growth curve. SGA infants were compared with all other infants.

Exposure assessment

For each job reported during the CATI, the mother was asked to provide the employer's name, job title, descriptions of the company's product/service, main job activities/duties, chemicals/substances handled and machines used on the job. Mothers also provided job start and end dates and the usual number of days worked per week and hours worked per day. Each self-reported job was then assigned standard codes corresponding to its occupation using the 2000 Standard Occupational Classification System,[25] and its industry using the 1997 North American Industry Classification System.[26]

Using the CATI data, exposure classification was conducted by two industrial hygienists (raters), blinded to NBDPS case/control status. For jobs considered possibly exposed to PAHs, the industrial hygienists independently assigned the following characteristics: (1) whether inhalation exposure was direct, indirect or both; (2) whether the inhalation exposure was continuous, intermittent or both; (3) the fraction of total hours worked when exposure was direct; (4) the fraction of total hours worked when exposure was indirect; (5) the intensity of any direct inhalation exposure during the period of direct exposure; (6) the intensity of any indirect inhalation exposure during the period of indirect exposure. Discrepancies between the two hygienists were resolved at a consensus conference with a third industrial hygienist. This expert review strategy was based on an approach that had been previously developed and used for other occupational exposures (eg, solvents, lead, radiation) in the Baltimore-Washington Infant Study.[27 28]

As part of the exposure assessment, a dichotomous yes/no rating of occupational exposure to PAHs was determined for each 'relevant' job (jobs considered relevant for SGA were those held in the period of 1 month before conception through delivery). A woman was classified as exposed if one or more of her relevant jobs was rated as exposed, whether part-time or full-time jobs. She was considered unexposed if all her relevant jobs were rated as unexposed.

In addition to the dichotomous rating, cumulative PAH exposure (unit-hours) was estimated by the industrial hygienists using: (weighted intensity in $\mu g/m^3$)×((hours worked per week)/(7 days per week))×(number of days worked in the relevant period). The resulting cumulative exposure value was job-specific rather than woman-specific; a woman's total occupational PAH exposure was calculated as the sum of the job-specific cumulative exposures in the relevant period.

'Occupational exposure' refers here to inhalation exposures inherent in the job or workplace aside from secondhand smoke, and did not consider exposure through skin or ingestion. Other sources of potential PAH exposure obtained from the interview included maternal smoking, secondhand smoke at home and secondhand smoke at work. Six women were excluded whose occupational PAH exposure could not be assigned for one or more of the jobs due to insufficient information.

Covariates

Several covariates were considered as potential confounders or effect measure modifiers based on associations either with SGA or with PAH exposure as reported in the literature. The CATI yielded data on the following maternal characteristics of interest as potential confounders (categories shown in table 1): age at delivery; race/ethnicity (based on standard CDC classification); education; number of previous live births; prepregnancy body mass index (categorised according to the National Heart, Lung, and Blood Institute cut-offs as underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2) and obese (≥30.0 kg/m^2)). Data on the following additional maternal characteristics also obtained during the CATI pertained to exposure in the period from 1 month before conception through delivery: use of folate antagonist medications (trimetrexate, methotrexate, carbamazepine, valproic acid, dilantin); consumption of folic acid supplements; cigarette smoking; secondhand smoke exposure at home; secondhand smoke exposure at work; and consumption of alcohol. The following were

Table 1 Association of maternal and infant factors with small for gestational age (SGA) offspring among National Birth Defects Prevention Study controls, 1997–2002

	SGA		Not SGA		
Characteristic	**n**	**%**	**n**	**%**	**p Value***
Total	221		2582		
Age (years)					
<20	10	4.5	216	8.4	0.35
20–24	64	29.0	551	21.3	
25–29	58	26.2	706	27.3	
30–34	45	20.4	747	28.9	
35+	44	19.9	362	14.0	
Missing	0		0		
Race/ethnicity					
White non-Hispanic	141	63.8	1690	65.5	0.14
Black non-Hispanic	19	8.6	334	12.9	
Hispanic	47	21.3	442	17.1	
Other	14	6.3	116	4.5	
Missing	0		0		
Education (years)					
<12	31	14.0	246	9.5	0.04
12	56	25.3	637	24.7	
>12	134	60.6	1697	65.8	
Missing	0		2		
Parity / number of previous live births					
0	82	37.1	1148	44.5	0.15
1	92	41.6	895	34.6	
2+	47	21.3	539	20.9	
Missing	0		0		
Infant sex					
Male	121	54.8	1280	49.6	0.14
Female	100	45.2	1302	50.4	
Missing	0		0		
Cigarette smoking†					
Yes	58	26.2	522	20.2	0.03
No	163	73.8	2060	79.8	
Missing	0		0		
Secondhand smoking at home†					
Yes	53	24.0	463	17.9	0.03
No	168	76.0	2119	82.1	
Missing	0		0		
Secondhand smoking at work†					
Yes	52	23.5	512	19.8	0.19
No	169	76.5	2070	80.2	
Missing	0		0		
Alcohol drinking†					
Yes	108	48.9	1209	46.8	0.56
No	113	51.1	1373	53.2	
Missing	0		0		
Prepregnancy body mass index					
Underweight	17	8.0	126	5.0	0.01
Normal weight	136	63.8	1437	56.7	
Overweight	36	16.9	588	23.2	
Obese	24	11.3	385	15.2	
Missing	8		46		
Folate antagonist medication†					
Yes	1	0.5	2	0.1	0.22‡
No	220	99.5	2580	99.9	
Missing	0		0		

Continued

Table 1 Continued

Characteristic	SGA n	SGA %	Not SGA n	Not SGA %	p Value*
Folic acid supplements†					
Yes	204	92.3	2437	94.4	0.20
No	17	7.7	145	5.6	
Missing	0		0		
Household income ($)					
<20 000	62	32.3	573	25.3	0.47
20 000–49 999	66	34.4	805	35.6	
50 000+	64	33.3	886	39.1	
Missing	29		318		
Study centre					
Arkansas	32	14.5	323	12.5	0.79
California	23	10.4	306	11.9	
Iowa	28	12.7	364	14.1	
Massachusetts	32	14.5	370	14.3	
New Jersey	34	15.4	347	13.4	
New York	22	9.9	297	11.5	
Texas	29	13.1	267	10.3	
CDC/Atlanta	21	9.5	308	11.9	
Missing	0		0		

*Probability calculated with χ^2 test based on non-missing values.
†Maternal exposure during the period from 1 month before conception through delivery.
‡Fisher's exact test.

also considered as potential confounders: infant sex; annual household income; and study site. In the analysis, all covariates were treated as categorical variables.

Statistical analysis

The association between each potential covariate and (A) SGA and (B) PAH exposure (yes/no) was examined using a χ^2 test or Fisher's exact test when cell sizes were less than 5 subjects. The number of jobs falling into the 23 Standard Occupational Classification-based major job groups were tabulated over all subjects, stratified by occupational PAH exposure status (yes/no).

Mothers of SGA infants were compared with mothers of all remaining infants. Using unconditional logistic regression, crude and adjusted ORs and 95% CIs were estimated to evaluate the association of maternal occupational exposure to PAHs with SGA in offspring. All the potential covariates described in the above section were included in a full model and submitted to manual backward stepwise logistic regression, removing the covariate with the highest p value. That covariate was retained if its removal resulted in a change of ≥10% in the effect measure estimate for PAHs and SGA. Then the next covariate with the highest p value was removed and checked for the 10% change, and so on. This continued until all remaining covariates exhibited the 10% change.

Several subanalyses were conducted: (1) To evaluate the independent effect of occupational PAHs, the first subanalysis adjusted for the most common source of human PAH exposure: active smoking and secondhand smoke exposure at home or at work. (2) Subjects were stratified by prepregnancy body mass index (obese/overweight mothers vs all others), as body fat may influence the storage and transformation of PAHs,[1] and stratified ORs were calculated to check for effect measure modification. Mothers were also stratified by age, race/ethnic group, education, folic acid supplement use, active smoking and any smoking exposure to examine possible effect measure modification. (3) To evaluate a possible exposure-response relationship, cumulative exposure level was categorised into none, low and high based on the frequency distribution among exposed mothers of non-SGA infants. The two-sided Cochran-Armitage trend test was used to test for trend. Estimated cumulative exposure was also analysed as a continuous variable, crude and adjusted for maternal age. (4) The largest number of PAH-exposed mothers was in food services. In order to control for possible residual confounding by socioeconomic status and to consider the possibility that exposures might be industry-specific, the PAH exposure (yes/no) analysis was stratified into women who worked in food services versus all other women. (5) To more closely replicate the previous study on occupational PAH exposure and birth outcomes,[15] birth weight was analysed in a linear model adjusted for gestational age. As in that study, customised growth curves for the entire study population were constructed, SD scores for each birth were calculated as a deviation from the 'overall' average at that gestational week, and represented as the equivalent Z-scores.[29] The linear model initially contained terms for exposure, gestational week, and exposure × gestational week, and non-significant terms were dropped.

RESULTS

Of the 2803 mothers included in the current study, 199 (7.1%) had preterm births, 35 (1.2%) had term deliveries that were low birth weight and 221 (7.9%) had offspring who were SGA. Mothers of SGA infants had a lower education, were more likely to smoke or report secondhand smoking at home, and to be underweight or normal weight compared with mothers of non-SGA infants (table 1). Other maternal characteristics that were checked but not significantly different were maternal age, race/ethnicity, parity, household income or NBDPS Center of residence, as well as the following exposures from 1 month before conception through delivery: secondhand smoke exposure at work, alcohol drinking, taking folic acid supplements.

Table 2 Association between maternal occupational exposure to polycyclic aromatic hydrocarbons (PAHs) and small for gestational age (SGA) offspring, National Birth Defects Prevention Study, 1997–2002

	Crude analysis				Adjusted analysis*			
	SGA n	Not SGA n	OR	95% CI	SGA n	Not SGA n	OR	95% CI
Dichotomous Exposure								
No PAH exposure	204	2480	1.0	(Reference)	204	2480	1.0	(Reference)
Any PAH exposure	17	102	2.0	(1.2 to 3.5)	17	102	2.2	(1.3 to 3.8)
Cumulative Exposure†‡								
No PAH exposure (0)	204	2480	1.0	(Reference)	204	2480	1.0	(Reference)
Low PAH exposure (8–205)	9	50	2.2	(1.1 to 4.5)	9	50	2.5	(1.2 to 5.3)
High PAH exposure (206–3086)	8	52	1.9	(0.9 to 4.0)	8	52	1.9	(0.9 to 4.2)

*Adjusted for maternal age.
†Cumulative exposure (unit-hours) was estimated by: (weighted intensity in $\mu g/m^3/h$)×((exposure frequency in hrs/week)/(40 h/week))×((hours worked per week)/(7 days per week))×(number of days worked from 1 month before conception to delivery).
‡Cumulative exposure measured in $\mu g/m^3$-h.

Sex was not significantly different between SGA and non-SGA infants.

Of the 2803 mothers, 119 (4.2%) had occupational PAH exposure in one or more jobs. Occupational PAH exposure was significantly more common among mothers who were younger, non-Hispanic Black or Hispanic, overweight or obese, had lower education, avoided drinking alcohol, smoked cigarettes, were exposed to secondhand smoking at home or work, or had lower income (data not shown). Among all jobs reported by mothers, almost half (45%) of those with occupational exposure to PAHs were in food preparation or serving related jobs (see online supplementary table). About 35% were in sales and related positions. Upon closer examination, it was found that most of the latter were (1) cashiers at fast food restaurants who worked the drive-through window, which is usually back in the kitchen area and very close to the grills/fryers, or (2) assistant managers, managers or floating staff in restaurants who work multiple positions, including sometimes cooking.

Occupational PAH exposure was found for 17 (7.7%) of the 221 mothers with SGA offspring and 102 (4.0%) of the remaining 2582 mothers. Maternal occupational PAH exposure was associated with having SGA offspring, whether crude or adjusted for maternal age (table 2); the adjusted OR was 2.2, with a 95% CI 1.3 to 3.8.

Statistically elevated ORs for SGA persisted whether adjusting for all smoking exposure (mother smoking or exposed to secondhand smoke at home or work) (OR 1.9, 95% CI 1.1 to 3.2), or for maternal age and all smoking exposure (OR 2.1, 95% CI 1.2 to 3.7). In our data, there was no evidence of statistically significant effect measure modification by any covariate of interest (data not shown). Even though the crude and adjusted models for SGA yielded a statistically significant trend with levels of estimated cumulative exposure ($p<0.02$), the increases were not monotonic so evidence of a trend was lacking (table 2). Increases were similar after adjusting for maternal age, smoking (active and secondhand) and education; the ORs and 95% CIs were no PAH exposure=1.0 (ref), low PAH=2.2 (1.0, 4.6), high PAH=1.8 (0.8, 3.9). Analysing cumulative exposure as a continuous variable also did not provide conclusive evidence of a trend; crude and adjusted for maternal age, OR=1.1 per 100 $\mu g/m^3$-h, 95% CI 1.0 to 1.1. When stratified by type of occupation, 10 women in food services with SGA offspring were occupationally exposed to PAHs, yielding a crude OR of 2.2 (95% CI 0.9 to 5.2). Seven women in other job types with SGA offspring were exposed to PAHs, with a crude OR of 1.5 (95% CI 0.7 to 3.3). Treating birth weight as a continuous variable adjusted for gestational age (ie, using Z-scores as the outcome) yielded non-significant terms for gestational age and the interaction which were dropped. The term for occupational PAH exposure was statistically significant, crude ($p=0.01$) and adjusted for maternal age ($p=0.01$).

DISCUSSION

This large population-based case-control study found that occupational exposure to PAHs was associated with an increased occurrence of SGA offspring. We did not observe a clear dose-dependent relationship in these analyses; the highest cumulative PAH exposure level showed a lower effect estimate with a CI that included 1.0. Occupational PAH exposure in food preparation and serving related jobs seemed to have a somewhat stronger association with SGA offspring than other occupations grouped together; however, the numbers were small.

Our observations were generally consistent with the only study we found that examined occupational exposure to PAHs and adverse birth outcomes, though the primary effect measures were different.[15] Their exposure information was gathered by a questionnaire completed in mid-pregnancy by Dutch women; exposure assessment involved using a job exposure matrix followed by consensus of three experts, yielding 1.3% of the women exposed to PAHs compared with 4.2% in the current analysis. Their outcomes (including fetal weight) were measured during pregnancy, while ours were at the end. Their study reported that fetal weight adjusted for gestational age (crude and adjusted for confounders) significantly decreased with maternal occupational PAH exposure. Our supplemental analysis of fetal weight adjusted for gestational age also found a statistically significant association with occupational PAH exposure. However, the estimated levels of exposure were not reported in the Dutch study, so those could not be compared with the current paper. The previous study found no statistically significant association of PAH exposure with fetal head circumference or length.

The current study was also consistent with a report of intrauterine growth restriction and environmental PAH exposure using stationary air monitoring.[16] It was partly consistent with two studies[17 18] that reported a statistically significant decrease in birth weight (adjusted for gestational age) with PAH exposure measured through personal monitoring in Krakow Caucasians

and New York City (NYC) African Americans. A later analysis from the same study[11] specifically examined SGA, and also found a significant association with PAH exposure among NYC African Americans.

A causal relationship between occupational PAH exposure and SGA is biologically plausible. PAHs cross the placenta[7] and have been found in cord blood.[5] They have been shown to form bulky DNA adducts in mothers and offspring.[30] Workers exposed to PAHs have higher levels of PAH-DNA adducts compared with the general population.[2 31] If not repaired, these adducts can disrupt the cell's microenvironment, leading to inhibition of important enzymes, cell death and alteration of other cells.[1 11] The fetus is particularly vulnerable due to its rapid growth, the process of cellular differentiation, the immaturity of its metabolic pathways and the stage of development of vital organs.[12] Another possible mechanism is if PAH exposure leads to periods of fetal hypoxia through reduced placental blood flow.[13] Future research with a greater sample size might start to clarify possible mechanisms by examining different exposure periods during pregnancy.

PAHs occur in complex and dynamic mixtures that may contain hundreds of separate chemicals; the individual components of these mixtures vary widely. For example, the PAH composition of a cigarette depends on the brand and its proprietary additives, and on the batch of tobacco (and where that batch was grown) and the batch of paper (and the species of trees, and where the trees grew, that were used in making the paper). This makes evaluating specific types of PAHs, rather than PAHs as a group, very difficult. Assessing PAHs as a group is sensible, in that it allows us to move beyond assessment by job title to begin zeroing in on a group of related chemicals that co-occur.

A limitation of this study was the potential for exposure misclassification. The three industrial hygienists had several years of experience and working knowledge, but the maternal occupational information reported during the CATI was limited and did not include potentially important exposure-modifying factors such as use of personal protective equipment and ventilation practices. However, the expert rater approach used was superior to relying exclusively on maternal self-report of PAH exposure, which is likely to be limited and could introduce recall bias.[32] In our dichotomised exposure analyses (any/none), non-differential errors in exposure misclassification would be expected to bias the ORs towards the null.

Another limitation was that in using these data, there was little information about other sources of PAH exposure. For example, subjects came from different areas of the country (highly industrialised to rural), and PAH levels are associated with certain industries and exposure to traffic.[1 10] The main source of ingested PAHs are some foods such as grilled, fried or smoked meat products which might partly explain the apparent higher OR for mothers in food service industries (although that might also have been due to residual confounding by socioeconomic status). Also, exposure assessment had not been conducted for paternal occupational exposure to PAHs. However, smoking is an important source of PAHs,[1] and we were able to account for personal tobacco use and exposure to secondhand tobacco smoke at home or at work. Further, since levels of occupational PAH exposure are generally higher than environmental sources,[2 3] it is important to consider exposure in the workplace separately from other sources.

A third limitation of this study was the small number of occupational exposed case mothers. This made it particularly challenging to fully assess a possible dose-response pattern and to adequately control for a full range of potential confounders. It also increased the likelihood that our results, though statistically significant, may have arisen by chance and thus require replication in future studies.

With an interview participation rate of 66.2%, this study was susceptible to selection bias. In general, NBDPS control participants differed from their base populations in maternal education, entry into prenatal care, birth weight, gestational age and plurality,[33] all of which may be related to SGA births and may impact generalisability to other populations. On the other hand, the participants did not differ in maternal age, smoking or diabetes. Selection bias may also be partly responsible for the difference in prevalence of occupational PAH exposure in this study (4.2%) versus the Dutch study (1.3% with an interview response rate of 76.9%), even though both sought a representative sample of births in their respective populations.

While defining SGA using a birth weight below the 10th centile for a given gestational age is currently the most common threshold used in research and clinical practice for which adverse perinatal outcomes are substantially increased,[34 35] there is no general agreement as to the most appropriate threshold value to use.[36–38] Two analyses, for example, have examined SGA using birth weights < 5th[39] and <3rd[40] centiles for gestational age, and further note that using an alternative cut point may be more appropriate for understanding pathological growth restriction. Given the small number of subjects meeting the criteria for a <5th centile cut point in our sample, however, we were unable to assess the impact of a more restrictive threshold as an outcome in our analysis.

One of the major strengths of this study was its use of thorough interview data available from the NBDPS. It yielded data on potentially important confounding factors. The occupational exposure assessment improved accuracy compared with the use of only job title or self-reported exposure, and the cumulative exposure estimation allowed examination of exposure-response relationships.

In summary, this study indicated a positive association between maternal occupational exposure to PAHs and SGA offspring. This was generally consistent with previous findings, and the study adds data to the sparse literature on maternal occupational PAH exposure studies. The large number of women in food service occupations and their slightly higher ORs may have implications for prevention strategies.

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