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Impact of barbecued meat consumed in pregnancy on birth outcomes accounting for personal prenatal exposure to airborne polycyclic aromatic hydrocarbons: Birth cohort study in Poland

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ABSTRACT

Objective: We previously reported an association between prenatal exposure to airborne polycyclic aromatic hydrocarbons (PAH) and lower birth weight, birth length, and head circumference. The main goal of the present analysis was to assess the possible impact of coexposure to PAH-containing barbecued meat consumed during pregnancy on birth outcomes.

Materials and Methods: The birth cohort consisted of 432 pregnant women who gave birth at term (>36 wk of gestation). Only non-smoking women with singleton pregnancies, 18–35 y of age, and who were free from chronic diseases such as diabetes and hypertension, were included in the study. Detailed information on diet over pregnancy was collected through interviews and the measurement of exposure to airborne PAHs was carried out by personal air monitoring during the second trimester of pregnancy. The effect of barbecued meat consumption on birth outcomes (birth weight, length, and head circumference at birth) was adjusted in multiple linear regression models for potential confounding factors such as prenatal exposure to airborne PAHs, child's sex, gestational age, parity, size of mother (maternal prepregnancy weight, weight gain in pregnancy), and prenatal environmental tobacco smoke.

Results: The multivariable regression model showed a significant deficit in birth weight associated with barbecued meat consumption in pregnancy (coeff = −106.0 g; 95%CI: −293.3, −35.8). The effect of exposure to airborne PAHs was about the same magnitude order (coeff. = −164.6 g; 95% CI: −172.3, −34.7). Combined effect of both sources of exposure amounted to birth weight deficit of 214.3 g (95%CI: −419.0, −9.6). Regression models performed for birth length and head circumference showed similar trends but the estimated effects were of borderline significance level. As the intake of barbecued meat did not affect the duration of pregnancy, the reduced birth weight could not have been mediated by a shortened gestation period.

Conclusion: In conclusion, the study results provided epidemiologic evidence that prenatal PAH exposure from diet including grilled meat might be hazardous for fetal development.

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Introduction

Polycyclic aromatic hydrocarbons (PAHs), being widely present in the human environment, pose health risks because some of them, such as benzo[a]pyrene (BaP) or benzo[a]

anthracene, are classified as “probably carcinogenic” to humans. PAH compounds are generated in the course of processing of coal, crude oil, and natural gas [1]. They are inhaled with outdoor and indoor pollutants generated by incomplete burning of oil and gas used for heating, vehicle transportation, and tobacco smoking. However, diet is a major source of exposure to PAH for an adult person [2–6], which is estimated to contribute to about 70–90% of total exposure [2,7].

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A high contribution of PAH exposure from the alimentary tract results to a great extent from the fact that cooking foods at high temperatures (grilling, roasting, frying) generates large amounts of PAHs [8–10]. While grilling, meat or its products are cooked over dry heat from open fire, and the term "barbecue" is traditionally applied to a cooking method, where grilling fire comes from smoking wood. When food is in direct contact with flame, pyrolysis of fats in the meat and smoke of hot charcoal generate great amounts of PAHs that mostly accumulate in the outer surface of the barbecued meat [9,10]. Other minor sources of exposure to PAHs are ingestion of house dust, or dermal absorption from contaminated soil and water [11,12].

After being absorbed into the human body, PAH compounds are distributed to almost all internal organs and are transferred through the placenta to the fetus [13,14], where they are metabolized to form a number of metabolites that may bind to DNA to form a moiety called PAH-DNA adducts. The formation of PAH-DNA adducts can alter DNA replication and may be associated with an increased risk of several forms of cancer [15,16].

Reproductive epidemiology provides solid evidence that the developing fetus and infants are significantly more sensitive to environmental toxicants than adults. This is due to differential exposure, physiologic immaturity, and a longer lifetime over which disease initiated in the early life can develop. It was shown that newborns and young children are especially vulnerable to the toxic effects of airborne PAH in terms of birth outcomes [17–21]. Most importantly, it was recently documented that prenatal exposure to airborne PAH may also have an effect on the future cognitive development of children [22].

The main goal of the present analysis is to assess the possible relationship between meat prepared by different cooking methods consumed during pregnancy and birth outcomes, after accounting for individual prenatal exposure to airborne PAH measured in the second trimester of pregnancy together with other potential confounders such as child's sex, gestational age, parity, size of mother (maternal prepregnancy weight, weight gain in pregnancy), and prenatal environmental tobacco smoke (ETS). The secondary goal of the study was to assess the effect of barbecued meat consumption in pregnancy on the level of cord blood B[a]P-DNA adducts, which is believed to be a molecular dosimeter of PAH compounds and can serve as a proxy for PAH-DNA adducts.

Material and methods

This study is part of an ongoing, longitudinal investigation of the health effects of prenatal exposure to outdoor and indoor air pollution on infants and children in Krakow, Poland. As described previously [23], between January 2001 and February 2004, we recruited 505 women registered at prenatal health care clinics in the central area of Krakow, where they had also lived for at least a year preceding screening. Each woman visiting the prenatal clinic because of her pregnancy received a letter of introduction and a short screening questionnaire to find whether the eligibility criteria are met. Eligibility criteria included: age ≥ 18 y, non-smoking, singleton pregnancies, no current occupational exposure to known developmental toxicants, no history of illicit drug use, pregnancy-related diabetes, or hypertension.

A total of 432 pregnant women who gave birth between January 2001 and February 2004 met these criteria and gave birth to term babies (>36 wk of gestation). On enrollment, a detailed questionnaire was administered to each subject at the entry to the study and in the third trimester to gather demographic data and information on house characteristics, date of the last menstrual period, medical and reproductive history, occupational hazards, alcohol consumption, and smoking practices of others present in the home. The questionnaires also elicited detailed information on dietary PAHs (specifically, the frequency of grilled, broiled, fried, or smoked meat consumption during each trimester of pregnancy). Prenatal ETS exposure was assessed by questionnaire data and cord blood cotinine measurements.

After giving birth, maternal and hospital records were reviewed to obtain data on delivery complications. Weight, length, and head circumference at birth were recorded for all infants. Gestational age at birth was defined as the interval between the last day of the mother's last menstrual period and the date of birth. The study was approved by the ethics committee of the Jagiellonian University.

Air monitoring

Study participants were monitored for personal exposure to airborne PAHs during the second trimester of pregnancy using a personal environmental monitoring sampler measuring particles and gases developed at Harvard School of Public Health. Details of the personal air monitoring conducted in Krakow have been published earlier [23]. Briefly, on completion of the interview, the women were given a backpack containing a portable air monitor to be worn during the day and kept near the bed at night during a consecutive 48-h period. The sampling pump draws air through a polyurethane sampler to measure PAH. After sampling, the field samples were frozen and shipped on dry ice to Southwest Research Institute in Texas. Personal air monitoring data were given a quality assurance score (0–3) for flow rate, flow time, and completeness of documentation [24]. Final quality assurance score of 0 (highest quality) or 1 (high quality) was required for inclusion. Air samples were analyzed at Southwest Research Institute for levels of pyrene and eight carcinogenic PAHs. Determination of total PAH concentration (benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene/iso-chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene, and pyrene) in extracts was performed. Chemical procedures in the analysis of the collected samples were described elsewhere [25]. In the present analysis, only total PAH measurements were considered.

Dosimetry of PAH-DNA adducts

Maternal blood (30–35 mL) was collected within 1 d postpartum, and umbilical cord blood (30–60 mL) was collected at delivery. Samples were transported to the laboratory immediately after collection. The buffy coat, packed red blood cells, and plasma were separated and stored at -70°C . BaP-DNA adducts in extracted WBC DNA white blood cells were analyzed using the high-performance liquid chromatography-fluorescence method of Alexandrov et al. [26], which detects BaP tetraols. The assay gives zero values when unexposed calf thymus DNA is tested (D. Tang, personal communication). The method has a variation coefficient of 12% and a lower limit of detection of 0.125 adducts per 10^8 nucleotides. High-performance liquid chromatography analysis of DNA samples for BaP-DNA adducts was performed in batches, with 18 paired maternal and newborn samples in the same batch.

Dosimetry of cord blood cotinine

In cord blood samples the serum cotinine concentration was measured at the Centers for Disease Control and Prevention using a sensitive isotope-dilution high-performance liquid chromatographic/atmospheric pressure ionization tandem spectrometric procedure. Limits of detection (LOD) were set below 0.05 ng/mL. About 20% of specimens had cotinine levels below the LOD. For statistical analysis, samples with non-detectable levels were assigned a value of 0.025, midway between the LOD of 0.05 and 0. Maternal blood cotinine level below 15.0 ng/mL was considered the borderline, separating smokers from non-smokers [27,28].

Statistical methods

The main birth outcomes were birth weight, length, and head circumference at birth; their association with exposure was examined by univariate and multivariate models. Several models were constructed. Following estimation of crude effects in multivariable linear regression models, the impact of the consumption of barbecued meat on the birth outcomes was adjusted for potential confounders (airborne PAHs, child's sex, gestational age, parity, maternal prepregnancy weight, weight gain in pregnancy, and prenatal environmental tobacco smoke). In all statistical analyses, the significance level was set at $P < 0.05$. All statistical analyses have been performed with STATA version 11.1 software.

Results

Analysis of personal air samples from 432 pregnant women included in the study showed that PAHs exposure (expressed in geometric means) in the second trimester of pregnancy averaged 26.05 ng/m³ (95%CI: 23.3–29.1 ng/m³ and for B[a]P, 2.7 ng/m³ (95%CI: 2.4–3.1). There was perfect correlation between total PAH and B[a]P concentrations ($\rho = 0.99$).

The mean weight, length, head circumference at birth, and gestational age for term babies under study were 3456.7 g, 54.8 cm, 33.9 cm, and 39.6 wk, respectively. The newborns of mothers who consumed any barbecued meat in the third pregnancy trimester showed significantly lower birth weight by 189.4 g, and shorter length at birth (of border significance level) by 0.82 cm. The corresponding reduction in head circumference was 0.4 cm, also of border significance (Table 1). Those subjects who consumed barbecued meat were exposed to much lower airborne PAHs concentrations and it resulted from the fact that the majority of them (73%) lived in the city areas with lower pollution level. The correlation between barbecued meat and airborne PAH was insignificant.

In the univariate analysis of the relationship between birth outcomes and the consumption of barbecued, roasted, fried,

and smoked meats in the various trimesters of pregnancy, non-parametric trend tests were used (Table 2). It has been shown that out of all individual food items considered, only barbecued meat consumed in the third trimester of pregnancy was significantly and inversely correlated with birth weight ($z = -1.99, P = 0.047$). Similar but insignificant trends were observed between other birth outcomes (length at birth and head circumference) and the consumption of barbecued meat in the last trimester of pregnancy.

There was a significant positive association between the level of the airborne PAHs (in tertiles) and cord blood adducts (non-parametric trend $z = 3.43, P = 0.001$) and a border significant association for maternal blood adducts and the airborne PAHs ($z = 1.68, P = 0.092$). However, the association between cord or maternal blood adducts and the consumption of barbecued,

Table 1

Characteristics of the study sample (term babies) by the consumption of barbecued meat in the third trimester of pregnancy

Variables	Total sample $N = 432$	Consumption of barbecued meat in the 3rd trimester of pregnancy		P level for difference
		No ($n = 384$)	Yes ($n = 48$)	
Mother's age				
Mean	27.57	27.52	27.96	0.4186
SD	3.55	3.60	3.10	
Maternal education (y of schooling)				
Mean	15.57	15.58	15.42	0.6980
SD	2.80	2.79	2.89	
Mother's height (cm)				
Mean	165.0	165.1	164.3	0.3389
SD	5.560	5.620	5.052	
Mother's prepregnancy weight (kg)				
Mean	58.15	58.17	57.98	0.8832
SD	8.669	8.632	9.057	
Maternal weight gain in prepregnancy (kg)				
Mean	15.41	15.48	14.90	0.4552
SD	5.095	5.054	5.443	
Gestational age				
Mean (wk) >36	39.57	39.58	39.50	0.6297
SD	1.127	1.128	1.130	
Birth order				
1, n (%)	272 (63.0)	244 (63.5)	28 (58.3)	0.5851
≥ 2 , n (%)	160 (37.0)	140 (36.5)	20 (41.7)	
Gender of baby				
Boys, n (%)	221 (51.2)	200 (52.1)	21 (43.7)	0.3494
Girls, n (%)	211 (48.8)	184 (47.9)	27 (56.2)	
Birth weight (g)				
Mean	3456.7	3477.7	3288.3	0.0048
SD	439.57	436.14	435.08	
Length at birth (cm)				
Mean	54.75	54.84	54.02	0.0437
SD	2.659	2.648	2.662	
Head circumference at birth (cm)				
Mean	33.94	33.99	33.58	0.0559
SD	1.388	1.375	1.456	
PAH total (ng/m ³)				
Mean	52.22	55.25	28.02	0.0073
SD	66.45	68.23	43.44	
Benzo(a)pyrene (ng/m ³)				
Mean	6.574	6.983	3.299	0.0056
SD	8.707	8.905	6.062	
Maternal blood adducts				
Mean	0.262	0.266	0.230	0.0596
SD	0.120	0.121	0.109	
Missing date	15	13	2	
Cord blood adducts				
Mean	0.271	0.270	0.284	0.5457
SD	0.153	0.135	0.254	
Missing date	42	39	3	
Prenatal ETS (+), n (%)	114 (26.4)	100 (26.0)	14 (29.2)	0.7722
Consumption of barbecued meat in the 2nd trimester of pregnancy (y/n), n (%)	112 (25.9)	96 (25.0)	16 (33.3)	0.2858

Table 2
Relation between birth weight and the frequency of barbecued, roasted, fried, and smoked foods consumed over pregnancy trimesters (nonparametric trend)

Type of foods	First two trimesters of pregnancy	Third trimester of pregnancy	Whole pregnancy
Barbecued meat	Z = -1.20, P = 0.230	Z = -1.99, P = 0.047	Z = -1.78, P = 0.075
Barbecued fish	Z = 0.36, P = 0.719	Z = -1.61, P = 0.107	Z = -0.67, P = 0.506
Total barbecued food	Z = -1.02, P = 0.307	Z = -2.05, P = 0.040	Z = -1.76, P = 0.079
Roasted meat	Z = 0.11, P = 0.911	Z = 0.91, P = 0.363	Z = 0.51, P = 0.608
Roasted fish	Z = 0.96, P = 0.355	Z = 1.47, P = 0.141	Z = 1.52, P = 0.127
Total roasted food	Z = 0.30, P = 0.762	Z = 1.22, P = 0.222	Z = 0.82, P = 0.414
Fried meat	Z = 1.15, P = 0.248	Z = 0.08, P = 0.935	Z = 0.87, P = 0.363
Fried fish	Z = -0.33, P = 0.742	Z = -1.39, P = 0.165	Z = -0.91, P = 0.363
Total fried food	Z = 0.90, P = 0.366	Z = -0.090, P = 0.366	Z = 0.46, P = 0.645
Smoked meat	Z = -2.88, P = 0.004	Z = 1.44, P = 0.151	Z = -1.05, P = 0.296
Smoked fish	Z = 1.44, P = 0.150	Z = 0.27, P = 0.791	Z = 1.18, P = 0.236
Smoked cheese	Z = -0.04, P = 0.969	Z = -1.18, P = 0.238	Z = -0.60, P = 0.550
Total smoked food	Z = -1.39, P = 0.165	Z = 0.85, P = 0.393	Z = -0.053, P = 0.598

roasted, fried, and smoked meats in various trimesters of pregnancy appeared to be insignificant.

For each birth outcome a separate model was constructed in which independent variables besides airborne exposure to PAH (dichotomized by median) included gender of child, parity, anthropometry of mother, gestational age, and ETS measured by cord blood cotinine. Although the effect of prenatal airborne PAH exposure (above median value) adjusted for a wide set of confounders amounted to a birth weight deficit of 106 g, the separate effect of ingested dietary PAH component resulted in a birth weight deficit of 165 g. The estimated combined exposure to both sources of PAHs produced the birth weight deficit of 214 g (Table 3). Of the total variability in birth weight (39%) estimated from the multivariable regression model, 1.3% could be attributed to prenatal airborne PAHs exposure, 0.9% to ETS exposure, and 0.8% to the consumption of grilled meat. The major variability in birth weight was explained by gestational age (13.4%), weight gain in pregnancy (7.9%), prepregnancy weight (7.3%), baby's sex (3.9%), and parity (3.3%).

A similar statistical approach for other birth outcomes revealed that the association between length and head circumference at birth and the consumption of barbecued meat was of borderline significance level (Tables 4 and 5).

In a separate analysis, we tested the effect of the consumption of barbecued meat in the last trimester of pregnancy on gestational age. There was no evidence that the intake of barbecued meat shortens the duration of pregnancy (results not shown here); thus, the observed deficit in birth weight and other birth outcomes could not have been mediated by shortening the gestation period.

Discussion

The results of our study suggest that both sources of PAH prenatal exposure (airborne and dietary) have an additive effect on birth weight deficit in newborns. Although the higher prenatal airborne PAH exposure (above median value) adjusted for a wide set of confounders amounted to a birth weight deficit of 106 g, the effect of ingested barbecued meat consumed in the last pregnancy trimester resulted in birth weight deficit of 165 g. The estimated combined exposure to both sources of PAHs produced the birth weight deficit of 214 g. Of the total variability in birth weight (39%) explained by the multivariable linear regression model, 1.3% could be attributed to prenatal airborne PAH exposure and only 0.8% to the consumption of charcoal grilled meat. A higher proportion of variability explained by airborne PAH was brought about by a higher prevalence of airborne PAH exposure (50%) compared with that of the grilled meat (11%). Because there was no significant interaction term between the exposure to airborne PAH and barbecued meat, the combined health effects should reflect summarized exposure to both PAH sources and not their synergistic effect. The reduction in birth weight attributed to the airborne PAHs or to the consumption of barbecued meat could not be mediated by shortening of gestation age, because duration of pregnancy was not associated with the exposures in question.

In earlier publications the formation of PAH-DNA adducts in peripheral white cells after consumption of charcoal broiled beef was reported by Rothman et al. [29,30]. In the latter study, the adducts levels were positively associated with the recent consumption of charcoal broiled food (median duration since the

Table 3
Estimated effect of barbecued meat consumed over the third trimester of pregnancy on birth weight adjusted for airborne PAHs and other potential confounders (multivariable linear regression model, N = 432, R² = 0.39)

Predictors	Coeff.	t	P > t	[95% Conf. Interval]
Maternal age	-4.50	-0.78	0.435	-15.80, 6.81
Maternal education (y)	13.47	1.95	0.052	-0.11, 27.04
Gestational age (wk)	140.38	9.20	0.000	110.39, 170.38
Maternal weight gain in pregnancy (kg)	26.48	7.63	0.000	19.65, 33.30
Prepregnancy maternal weight (kg)	14.72	7.47	0.000	10.85, 18.59
Child's sex (girls)	-189.93	-5.52	0.000	-257.62, -122.24
Parity	103.63	3.40	0.001	43.64, 163.63
Cord cotinine (lg)	-56.52	-2.22	0.027	-106.52, -6.51
Airborne PAH* (-)	reference			
Airborne PAH* (+)	-164.57	-2.51	0.012	-293.31, -35.83
Airborne PAH† (-)	-106.00	-2.92	0.004	-177.27, -34.65
Airborne PAH† (+)	-214.30	-2.06	0.040	-419.01, -9.59

* - Airborne PAHs below or equal median value (= 24.9 ng/m³); + Airborne PAHs above median value.

† - = no consumption of the barbecued meat; + = reported consumption of the barbecued meat in the last trimester of pregnancy.

Table 4

Estimated effect of barbecued meat consumed over the third trimester of pregnancy on length at birth adjusted for airborne PAHs and other potential confounders ($N = 432$, $R^2 = 0.27$)

Predictors	Coef.	t	$P > t$	[95% Conf. Interval]
Maternal age	0.04	0.93	0.354	–0.04, 0.11
Maternal education (y)	0.03	0.60	0.547	–0.06, 0.12
Gestational age (wk)	0.78	7.73	0.000	0.58, 0.98
Maternal weight gain in pregnancy (kg)	0.12	5.06	0.000	0.07, 0.16
Prepregnancy maternal weight (kg)	0.06	4.86	0.000	0.04, 0.09
Child's sex (girls)	–1.19	–5.19	0.000	–1.64, –0.74
Parity	0.17	0.83	0.408	–0.23, 0.57
Cord cotinine (lg)	–0.31	–1.84	0.066	–0.64, 0.02
Airborne PAH exposure*	–0.67	–2.90	0.004	–1.12, –0.22
Consumption of barbecued meat [†]	–0.67	–1.82	0.070	–1.39, 0.05

* Dichotomized by median value of the distribution (24.9 ng/m³).

[†] 0 = no consumption of the barbecued meat, 1 = reported consumption of the barbecued meat in the last trimester of pregnancy.

Table 5

Estimated effect of barbecued meat consumed over the third trimester of pregnancy on head circumference adjusted for airborne PAHs and other potential confounders ($N = 432$, $R^2 = 0.25$)

Predictors	Coef.	t	$P > t$	[95% Conf. Interval]
Maternal age	0.01	0.24	0.814	–0.04, 0.04
Maternal education (y)	0.05	2.08	0.039	0.01, 0.10
Gestational age (wk)	0.25	4.79	0.000	0.15, 0.36
Maternal weight gain in pregnancy (kg)	0.06	4.71	0.000	0.03, 0.08
Prepregnancy maternal weight (kg)	0.04	5.35	0.000	0.02, 0.05
Child's sex (girls)	–0.73	–6.12	0.000	–0.97, –0.50
Parity	0.32	3.01	0.003	0.11, 0.53
Cord cotinine (lg)	–0.14	–1.59	0.113	–0.31, 0.03
Airborne PAH exposure*	–0.32	–2.63	0.009	–0.55, –0.08
Consumption of barbecued meat [†]	–0.36	–1.87	0.062	–0.74, 0.02

* Dichotomized by median value of the distribution (24.9 ng/m³).

[†] 0 = no consumption of the barbecued meat; 1 = reported consumption of the barbecued meat in the last trimester of pregnancy.

last consumption was 6 d with a range of 1–30 d). Individuals who consumed barbecued food more than twice in the previous 2 wk had a four-fold increased risk of having elevated adducts level (>0.2 fmol/ μ g DNA) compared to individuals consuming this kind of food two or fewer times. However, the results of the analysis were not adjusted for airborne PAH exposure.

Our study did not show the association between consumption of barbecued meat in pregnancy and cord blood PAH-DNA adducts. However, DNA adduct formation is subject to a greater variability than external exposure. As this variability results from differences in metabolic phenotypes related to genetic polymorphisms in a variety of enzymes involved in the activation or detoxification of PAH or repair of PAH-DNA adducts, individuals with a similar level of external exposure might have different PAH-DNA adduct levels [15]. Moreover, the recent study by Kelvin et al. [31] showed that plasma antioxidants may also modulate the effect of prenatal PAH exposure on PAH-DNA adducts in cord blood.

Our study showing the negative impact of airborne PAH exposure on birth outcomes corresponds with earlier findings of Perera et al. [32–34] and with the results of the analysis published by Choi et al. [21]. Choi et al. demonstrated in two separate but parallel prospective cohort studies that prenatal exposure to airborne PAHs was significantly associated with adverse effects of fetal growth in both Polish and US African Americans.

The potential strength of our study comes from the fact that personal monitoring of exposure to airborne PAHs in pregnancy was performed in each study subject. In our study, the most important confounders of the birth outcomes, such as the presence of chronic diseases or active tobacco smoking by

mothers in pregnancy, have been removed through entry criteria. Other risk factors that might potentially have an effect on giving birth to newborns with lower growth, such as maternal prepregnancy weight, weight gain in gestation, gestation age, parity, child's sex, and prenatal ETS, were also accounted for in the analysis.

Main weak points of our study result from the relatively small number of individuals who consumed barbecued food in pregnancy, the lack of more precise information on timing of the ingestion of barbecued meat in the last pregnancy trimester, and estimates of PAH dietary exposure based only on the food frequency questionnaires, which are prone to recall bias. Moreover, the consumption of barbecued meat might be a proxy indicator of some life style factors such as particular maternal nutrition patterns, inadequate provision of polyunsaturated fatty acids [35], folic acid or iron in maternal diet [36], and other potential coexposures that were not controlled in the present study. Nevertheless, supplementary analysis of the data regarding alcohol intake in pregnancy did not show correlation with any of the main PAH exposure variables.

In conclusion, this study highlights the fact that not only contaminated air might be linked to detrimental birth outcome effects but also possibly the exposure to PAH compounds resulting from the consumption of grilled food in pregnancy, unrecognized to date as a reproductive health hazard. The results of the study may have practical implications for public health policy and establishing guidelines for the protection of newborns' health; however, more studies exploring the impact of dietary sources of PAH exposure in pregnancy on newborns' health are needed.

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