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Long term effects of prenatal and postnatal airborne PAH exposures on ventilatory lung function of non-asthmatic preadolescent children. Prospective birth cohort study in Krakow

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HIGHLIGHTS

- This is the first study on PAH exposure on lung function in non-asthmatic children
- Observed deficits of lung function due to PAH exposure persist through childhood
- Prenatal and postnatal PAH exposure compromises the respiratory airways development

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ABSTRACT

The main goal of the study was to test the hypothesis that prenatal and postnatal exposures to polycyclic aromatic hydrocarbons (PAH) are associated with depressed lung function in non-asthmatic children. The study sample comprises 195 non-asthmatic children of non-smoking mothers, among whom the prenatal PAH exposure was assessed by personal air monitoring in pregnancy. At the age of 3, residential air monitoring was carried out to evaluate the residential PAH exposure indoors and outdoors. At the age of 5 to 8, children were given allergic skin tests for indoor allergens; and between 5 and 9 years lung function testing (FVC, FEV_{05}, FEV_1 and FEF_{25-75}) was performed. The effects of prenatal PAH exposure on lung function tests repeated over the follow-up were adjusted in the General Estimated Equation (GEE) model for the relevant covariates. No association between FVC with prenatal PAH exposure was found; however for the FEV_1 deficit associated with higher prenatal PAH exposure (above 37 ng/m^3) amounted to 53 mL ($p = 0.050$) and the deficit of FEF_{25-75} reached 164 mL ($p = 0.013$). The corresponding deficits related to postnatal residential indoor PAH level (above 42 ng/m^3) were 59 mL of FEV_1 ($p = 0.028$) and 140 mL of FEF_{25-75} ($p = 0.031$). At the higher residential outdoor PAH level (above 90 ng/m^3) slightly greater deficit of FEV_1 (71 mL, $p = 0.009$) was observed. The results of the study suggest that transplacental exposure to PAH compromises the normal developmental process of respiratory airways and that this effect is compounded by postnatal PAH exposure.

1. Introduction

Over the last several decades there have been a number of cross-sectional studies, mainly in Europe and North America, on the association between depressed lung function and lung growth in children with postnatal chronic exposure to airborne particulate matter (PM) or other airborne toxins, such as NO_2, SO_2 or ozone (Frye et al., 2003; He et al., 1993; Peters et al., 1999; Raizenne et al., 1996; Speizer et al., 1980). Although the results were not always consistent, they suggested that poor ambient air quality may be causally connected with impaired lung function. Longitudinal studies undertaken on chronic postnatal

Abbreviations: DEP, diesel exhaust particles; ETS, environmental tobacco smoke; FVC, forced volume capacity; FEV_{05}, forced expiratory volume in 0.5 s; FEV_1, forced expiratory volume in 1 s; FEF_{25-75}, forced expiratory flow 25–75%; GEE, Generalized Estimation Equations; PAH, polycyclic aromatic hydrocarbons.

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exposure provided further evidence on the role of air pollutants in the lung growth, but they did not show the type of lung damage attributable to individual components of ambient air pollution (Detels et al., 1991; Dockery and Brunekreft, 1996; Frischer et al., 1999; Gauderman et al., 2000, 2002, 2004; Jedrychowski et al., 1999). Moreover, the lack of epidemiologic studies on the prenatal effects of air pollutants made it impossible to assess the full impact of ambient air pollution on the natural history of lung function growth. Recently published papers have identified exposure to diesel exhaust particles (DEP) as an important cause of respiratory illness. DEP contain a broad spectrum of PAH and are common outdoor airborne pollutants. Animal and human exposure studies have shown that inhaled DEP induce an inflammatory response in human airways and exert adverse effects on pulmonary function (Brunekreef et al., 1997; Maeda et al., 1991; Sydbom et al., 2001).

Not only diesel exhausts, but also coal combustion is a major source of PAH (Junninen et al., 2009; Lvovsky et al., 2000). Ambient air pollutants penetrate readily into the indoor environment (Junninen et al., 2009; Jung et al., 2010), however, PAH compounds are also generated indoors by residential heating (e.g., coal or wood stoves, fireplaces, kerosene heaters, unvented gas appliances, environmental tobacco smoke (ETS), and fumes from cooking, grilling, and frying (Zedeck, 1980). Up to now, investigations on the specific effects of PAH exposure on children's respiratory health are scarce, though in the last decade the effect of PAH exposure on adverse birth outcomes, including low-birth weight, premature births, slower intrauterine growth and retardation in neurodevelopment was confirmed (Choi et al., 2006; Dejmek et al., 2000; Edwards et al., 2010; Perera et al., 1998, 2005, 2009).

It is realistic to assume that prenatal exposure to ambient air pollutants may have negative consequences for normal fetal development of various organs, such as the lungs, and the immunologic system. These, in turn, may lead to deficient function of the affected organs in postnatal life. For example, studies of ETS in pregnancy suggest that the environmental toxins can interfere with fetal development and their impact can be seen in postnatal life (Cunningham et al., 1994). Eventual changes in lung structure resulting from prenatal exposure can persist and lead to an increased burden of respiratory illness in adult life.

The main goal of this study was to test the hypothesis that lung function in non-asthmatic preadolescent children is associated with prenatal and early postnatal exposures to PAH. Assessment of individual prenatal exposure to airborne PAH compounds in each child under study was performed in the second trimester of pregnancy, a period when branching of the airway system is being completed. However further growth and cellular differentiation continue through postnatal life as well (Bucher and Reid, 1961), so each child's postnatal PAH exposure was also monitored. The study was restricted to non-asthmatic subjects as asthmatic children usually suffer from various chronic respiratory symptoms like wheezing, difficult breathing and attacks of shortness of breath, and for these reasons are under medical treatment with various drugs (bronchodilators and/or anti-allergic specimens) which make them less prone to the effects of air pollutants (Peters et al., 1997).

2. Materials and methods

This study is nested in a birth cohort study of children in Krakow, a collaborative research project of the Jagiellonian University in Krakow and Columbia University in New York. In the city of Krakow, coal combustion for domestic heating represents the major air pollution source, and automobile traffic emissions and coal-combustion for industrial activities are relatively minor contributors (Junninen et al., 2009). During heating seasons (October–April) ambient PAH concentrations within Krakow inner city area reach very high levels.

The present analysis was restricted to 195 non-asthmatic children who took part in the follow-up and performed reliable and acceptable spirometric tests. The design of the study and the detailed selection of the population sample have been described previously (Jedrychowski et al., 2003). In short, pregnant women were recruited from ambulatory prenatal clinics in their first or second trimester of pregnancy. Only women 18–35 years of age, who claimed to be non-smokers, with singleton pregnancies, with no history of illicit drug use and HIV infection, free from chronic diseases such as diabetes or hypertension, and who had resided in Krakow for at least one year prior to pregnancy were eligible for the study. Prior to participation, women read and signed an informed consent. The Ethical Committee of the Jagiellonian University in Krakow and Columbia Presbyterian Medical Center approved the research.

Upon enrollment, a detailed questionnaire was administered to each woman to solicit information on demographic data, home characteristics, medical and reproductive history, occupational hazards, and smoking practices of others present at home. After delivery, every

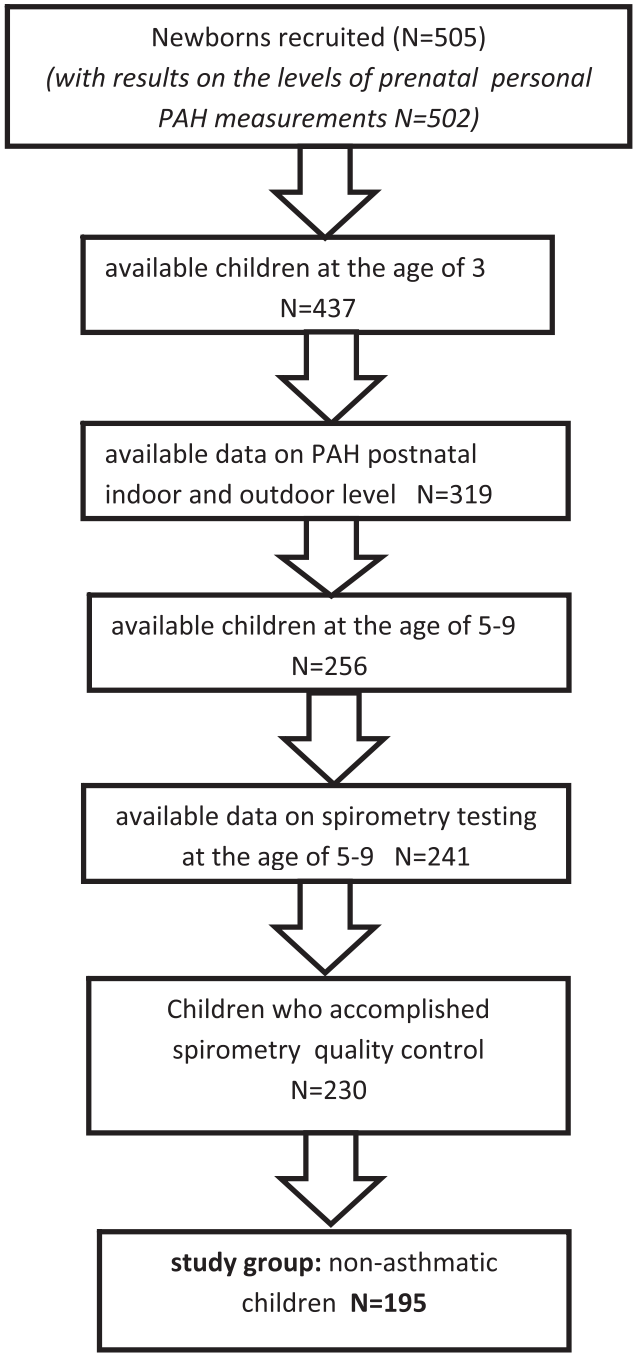

Fig. 1. Flow chart of participant recruitment and derivation of the study population used in the final analysis.