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Early life exposure to traffic-related air pollution and allergic rhinitis in preschool children

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ABSTRACT

Background: Evidence linking long-term exposure to outdoor air pollution with allergic rhinitis (AR) in children is scare, and the role of components of air pollution and timing of exposure remains unclear.
Objective: To assess the association of pre- and post-natal exposure to air pollution with life-time prevalence of AR in preschool children.
Methods: We conducted a cohort study of 2598 children aged 3–6 years in Changsha, China. The lifetime prevalence of AR was assessed by a questionnaire administered by parents. Children's exposures to dioxide nitrogen (NO_2), sulfur dioxide (SO_2) and particulate matter with an aerodynamic diameter $\leq$ 10 μm (PM_{10}) during different pre- and post-natal timing windows were estimated using the measured concentrations at monitoring stations. The odds ratio (OR) and 95% confidence interval (CI) of childhood AR for exposure to different air pollutants during different timing windows were assessed by logistic regression model in terms of an interquartile range (IQR) increase in exposure level.
Results: Life-time prevalence of AR in preschool children (7.3%) was associated with both pre- and post-natal exposure to traffic-related air pollution (TRAP), but only significant during the third trimester of pregnancy with adjusted OR = 1.40 (95% CI: 1.08–1.82) for a 15 μg/m^3 increase in NO_2 and during the first-year of life with adjusted OR = 1.36 (95% CI: 1.03–1.78) and 1.54 (95% CI: 1.07–2.21) respectively for 11 and 12 μg/m^3 increase in NO_2 and PM_{10}. The association of early life exposure to TRAP with childhood AR was robust by adjusting for other air pollutants and timing windows. Sensitivity analysis indicated that the association was higher in the children who are male, young, with genetic predisposition by parental atopy, and living in damp houses.
Conclusion: Early life exposure to traffic-related air pollutant during pregnancy and first-year of life may contribute to childhood AR.

1. Introduction

Allergic rhinitis (AR) is the most common chronic allergic disease and now recognized as a global health problem that affects as many as 40% children [1]. The prevalence of childhood AR has continuously increased in recent decades worldwide, including China [2]. Due to the fact that AR is not a serious condition, it is frequently ignored and under-diagnosed, especially in developing countries, leading to more serious complications in both upper and lower airways such as asthma, sinusitis, and otitis media [3]. Besides the significantly high morbidity and substantial health care costs, AR strongly affects children's mental health, quality of life, and learning at school [1]. The increasing trend and severity in AR deserve more attention. The major need lies in finding out the key causal factors and timing windows so as to develop more effective measures of prevention and intervention.

There is accumulating evidence that both genetic and environmental factors play important roles in the etiology of allergic diseases including AR. Genetic factors are unlikely to explain the rapid increase in prevalence and therefore environmental factors have

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been found to be important contributors to the manifestations of allergic diseases. Mounting evidence suggests that long-term exposure to air pollution, particular the traffic-related air pollution, contributes to the development of allergic disease [4,5]. Surprisingly very few studies have addressed AR as an endpoint, although some recent short-term epidemiological studies have demonstrated a strong association between air pollution and outpatient visits for AR [6–8]. However, the limit evidences linking long-term exposure to air pollution and allergic rhinitis were equivocal [9–13], and hence warrant further studies.

Early-life exposure has recently been suggested to be an important determinant in the later development of allergic diseases throughout childhood and lifespan. Environmental challenges during pregnancy and early postnatal life have long been hypothesized to modulate the susceptibility to chronic diseases in later life, which is commonly referred to as “Barker hypothesis” or “theory of developmental programming” [14]. Due to the rapid development and immaturity of the immune and respiratory systems, the fetuses and infants are most vulnerable to the adverse effects of air pollution [10]. However, the effect of air pollution exposure during early life, particularly during pregnancy and the first year of life, on allergic diseases in later life has rarely been assessed [10,11,15–18].

Having in mind the inconsistent evidences linking AR with long-term exposure to air pollution and the scanty data on the effect of early life exposure, we engaged in a nationwide study “China-Children-Homes-Health” (CCHH) to examine the association between lifetime prevalence of AR in preschool children and early life exposure to air pollution during different time windows, with an objective to identify the key components of air pollution and time windows of exposure.

2. Methods

2.1. Study population

Between September 2011 and January 2012, we conducted a survey for childhood asthma and allergies in the kindergartens in Changsha, the capital city of Hunan Province in south–central China, having a population of 7.22 million and covering an area of 1909 km^2. The study protocol was approved by the Ethics Committee of the Central South University and also by the health department of each kindergarten. A Chinese version questionnaire combining a standard one designed by the International Study of Asthma and Allergies in Childhood (ISAAC) and another Swedish one about dampness in buildings and health (DBH) [2,19], with some changes to address the housing and cultural characteristics in China, was administered to collect information on health status and possible exposure to the indoor environmental factors of children and family members.

A total of 4988 questionnaires were randomly distributed to the children at 36 participating kindergartens. Children were instructed to have the questionnaire completed by parents and to return it to kindergartens within one week. We received 3897 completed questionnaires with overall response rate 78%. We first excluded 745 children from kindergartens having a response rate lower than 50%, as these kindergartens were mostly distributed in the suburban areas where the children are mainly from the far rural areas and thus the children's early-life exposure during pregnancy and first-year of life cannot be estimated using urban data. Then, we also excluded 162 children with low birth weight (<2.5 kg) and preterm birth (<37 weeks of gestation), and 10 children with multiple births, as these conditions may confound the association between air pollution and allergic diseases. We further excluded 80 children aged younger than 3 and older than 6 because of few data. The 302 children without information about health outcome were finally excluded. Therefore, in total 2598 responses of valid questionnaires were used.

2.2. Exposure assessment

We selected three pollutants, sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter $\leq$ 10 μm in diameter (PM_{10}) to represent ambient air pollution in Changsha where SO_2 was used as an indicator of industry–related air pollution, NO_2 as traffic–related air pollution, and PM_{10} as a surrogate of complex mixture of air pollutants [20]. Average daily concentrations of PM_{10}, SO_2 and NO_2 were obtained from 7 municipal air pollution monitoring stations. Measurements at the monitoring stations followed the standard methods set by the State Environmental Protection Agency of China: PM_{10} by a tapered element oscillating microbalance (TEOM1400, Rupprecht & Patashnick, USA), SO_2 by ultraviolet fluorescent method (ML/EC9850, Ecotech, Australia) and NO_2 by the chemiluminescent method (ML/EC9841B, Ecotech, Australia). The distribution of monitoring stations and surveyed kindergartens was provided in our previous study [16].

Individual exposure to air pollution was estimated by an inverse distance weighted (IDW) method described elsewhere [16]. Briefly, children's exposure was calculated in terms of the air pollutant concentrations at their kindergartens. At first, the average daily concentration at each kindergarten was calculated by the obtained daily concentrations from the nearest 4 monitoring stations. Then, the monthly mean concentrations of air pollutants at each kindergarten were computed as the averages of the daily concentrations at the kindergarten within each month.

We divided the lifetime exposure into prenatal and postnatal periods. Prenatal period during pregnancy was further divided into three trimesters, and the postnatal period was further divided into first-year of life and thereafter or after first-year (from the second year to the last year). The monthly mean concentrations of PM_{10}, SO_2, and NO_2 were used to estimate the exposure during above different timing windows.

2.3. Health outcome

Health outcomes are lifetime prevalence of doctor-diagnosed allergic rhinitis, which was defined as an affirmative answer to the question: “Has your child ever been diagnosed having hay fever or allergic rhinitis (AR)?”

2.4. Confounding covariates

Information on potential confounders was obtained from the questionnaire. The confounding variables in the present analysis included personal factors (child's sex, age, birth season, breastfeeding, parental atopy, and socioeconomic status (SES) indicated by house size) and indoor factors including environmental tobacco smoke (ETS) at home, new furniture, house redecoration, visible mold/damp stains, condensation on windows in winter, cockroaches noted, and household pets (Table 1). These confounders were related to the reporting of childhood allergies and may affect estimated associations between AR and air pollution [21]. Parental atopy was a measure of genetic predisposition and defined as an affirmative answer to the question: “Have you ever been diagnosed having atopic diseases (asthma, allergic rhinitis, or eczema)?” ETS was defined as an affirmative answer to the question: “Is there a family member smoking at home (Yes/No)?” and if yes: “Who is smoking?” In the present work, the family SES was estimed by using house size, as we haven't the information about parental education and occupation.