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Prenatal exposure to perfluoroalkyl acids and allergic diseases in early childhood☆,☆☆

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ABSTRACT

Perfluoroalkyl acids (PFAAs) are persistent organic pollutants that are detected in humans worldwide. Laboratory animal studies have shown that PFAAs are associated with immunotoxic effects. However, epidemiological studies investigating the role of PFAAs, in particular PFAAs with longer chains than perfluorooctanoic acid, are scarce. We investigated associations between prenatal exposure to PFAAs, including long-chain compounds, and infant allergic diseases at 12 and 24 months in a large study population. The participants included mothers and their infants who enrolled in the Hokkaido Study on Environment and Children's Health 2003–2009. Eleven PFAAs were measured in maternal plasma taken at 28–32 weeks of gestation using ultra-performance liquid chromatography coupled to triple quadrupole tandem mass spectrometry. Characteristics of participants and information on infant allergic diseases were obtained from self-administered questionnaires and medical records. At 24 months, the adjusted odds ratio (OR) (first vs. fourth quartiles) for eczema in association with higher maternal perfluorotridecanoic acid (PFTrDA) levels was 0.62 (95% confidence interval (CI) 0.45, 0.86). After stratification by gender, the adjusted ORs in female infants from mothers with higher maternal perfluoroundecanoic acid (PFUnDA) and PFTrDA levels were also statistically significant (PFUnDA: OR = 0.50; 95% CI, 0.30, 0.81; PFTrDA: OR = 0.39; 95% CI, 0.23, 0.64). Our findings suggest that lower prenatal exposure to PFTrDA may decrease the risk of developing eczema in early childhood, only in female infants.

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1. Introduction

Perfluoroalkyl acids (PFAAs) are used in a broad range of consumer products because of their surface properties, which include insulation and water resistance. These compounds are persistent organic pollutants that are widespread within the environment, wildlife, and humans (Lau et al., 2007). Contamination of drinking water and foodstuffs such as seafood, leaching from food packaging and non-stick cookware, and household dust are major known routes of human exposure (Fromme et al., 2009). Potential health effects associated with PFAA exposure in humans are worsened by both bioaccumulation and persistence.

PFAA exposure has been suggested to have immunotoxic effects in laboratory animals including altered inflammatory responses, production of cytokines, and adaptive and innate immune responses (Dewitt et al., 2009). Cytokine expression and signaling related to inflammation and T-helper cell responses are altered in PFAA-exposed animals (Dewitt et al., 2012). PFAAs cross the placental barrier and are transferred to the fetus in humans (Midasch et al., 2007; Monroy et al., 2008). Previous epidemiological studies have shown a positive or negative association between perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) and levels of cord blood immunoglobulin (Ig) E (Okada et al., 2012; Wang et al., 2011a). Moreover, these studies have reported no association between prenatal PFOS, PFOA, or perfluorononanoic acid (PFNA) exposure and allergic and infectious diseases as health outcomes in children (Fei et al., 2010; Okada et al., 2012; Wang et al., 2011a). In the C8 Health Project, which was a cross-

Abbreviations: PFAAs, perfluoroalkyl acids; PFCAs, perfluorinated carboxylic acids; PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid; PFOA, perfluorooctanoic acid; PFNA, perfluorononanoic acid; PFDA, perfluorodecanoic acid; PFUnDA, perfluoroundecanoic acid; PFDoDA, perfluorododecanoic acid; PFTrDA, perfluorotridecanoic acid; PFTeDA, perfluorotetradecanoic acid; PFHxS, perfluorohexane sulfonate; PFOS, perfluorooctane sulfonate; MDL, method detection limits; CI, confidence interval; OR, odds ratio; Ig, immunoglobulin; ETS, environmental tobacco smoke; ISAAC, International Study of Asthma and Allergies in Childhood.

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☆☆ Ethics approval: This study was conducted with written informed consent from all participants and was approved by the institutional ethical board for epidemiological studies at the Hokkaido University Graduate School of Medicine.

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sectional, immune biomarker study that investigated residents in the vicinity of a PFOA plant, IgA, IgE, and C-reactive protein levels significantly decreased with increasing PFOA levels in blood samples (Fletcher et al., 2009).

In 2002, after 50 years of production, the 3M Company phased out the manufacture and distribution of PFOS (Renner, 2001). PFOS was also included in Annex B of the 2009 Stockholm Convention on Persistent Organic Pollutants (UNEP, 2007; Wang et al., 2009). The Environmental Protection Agency of the United States (2006) launched a 2010/2015 PFOA Stewardship Program to voluntarily reduce PFOA emissions. Recent studies indicate that concentrations of PFOS and PFOA are declining in the general human population (Olsen et al., 2012; Sundström et al., 2011; Wang et al., 2011b). In contrast, concentrations of PFNA and perfluorodecanoic acid (PFDA), which are long-chain perfluorinated carboxylic acids (PFCAs), are increasing in the general human population (Wang et al., 2011b). However, the effects of prenatal exposure to other PFAAs, particularly PFCAs, which generally have longer chains than PFOA with a carbon chain length of eight (e.g., PFDA, perfluoroundecanoic acid (PFUnDA), perfluorododecanoic acid (PFDoDA), and perfluorotridecanoic acid (PFTrDA)), have not been characterized. PFCAs with chains longer than those of PFOA have high bioconcentration factors, suggesting that they are environmentally persistent (Martin et al., 2003). Furthermore, between 2003 and 2011, we reported increased PFNA and PFDA in maternal plasma levels in Japanese, whereas levels of PFOS and PFOA decreased (Okada et al., 2013). Epidemiological determination of whether exposure to long-chain PFCAs affects immunity and allergic responses in humans is critical.

In this study, we explored associations between maternal PFAA levels, including long-chain compounds, and allergic diseases in early childhood using a prospective birth cohort study.

2. Methods

2.1. Study population

This prospective ongoing birth cohort study (Hokkaido Study on Environment and Children's Health) includes mothers who gave birth at hospitals in Hokkaido, Japan and their infants. The study was initiated in February 2003, and details have been described elsewhere (Kishi et al., 2011; Kishi et al., 2013). Briefly, participants were considered eligible if they were indigenous Japanese women who had received antenatal care at one of 37 participating hospitals within Hokkaido during their first trimester of pregnancy. Of the 33,500 eligible women invited to participate in the study from 2003 to 2009, 17,869 agreed to join (participation rate 53.3%). These participants signed informed consent forms, completed a baseline questionnaire, and also mailed follow-up questionnaires. From all participants ($n = 17,869$), we selected 12,847 who had submitted a baseline questionnaire and from whom we had obtained a third trimester blood sample and hospital birth records. From these, we excluded cases of miscarriage and stillbirth ($n = 19$), congenital malformation ($n = 143$), and multiple births ($n = 162$), because these are common exclusion criteria for studies investigating allergies, infectious diseases, mental development, and endocrine metabolic disorders. From the selected 12,523 participants, we then extracted 6335 participants who had completed all three self-administered questionnaires (at 4, 12, and 24 months after birth) for long-term follow-up of child development. Finally, from these 6335 participants, we randomly extracted 300 participants per year from 2003 to 2008 and 295 participants in 2009 to give a total of 2095 participants selected for the PFAA analysis of maternal plasma. Of these participants, we excluded cases of congenital malformations that became apparent from the follow-up questionnaire at 12 months ($n = 17$) and those whose maternal blood samples were taken before 26 weeks of gestation ($n = 15$) because the time of blood sampling during pregnancy may have affected the concentrations due to increased maternal blood volume during gestation. Thus, a final total of 2063 study participants met the specific exclusion and inclusion criteria for this study (Fig. 1). The protocol used in this study was approved by the institutional ethical board for epidemiological studies at the Hokkaido University Graduate School of Medicine.

2.2. Data collection

Participants completed a self-administered baseline questionnaire during the first trimester of pregnancy. The baseline questionnaire included maternal and paternal information related to age, pre-pregnancy height and weight, previous medical history, educational level, household income, alcohol intake during pregnancy, and parity. Medical birth records from hospitals included the gestational age, infant gender, and birth weight, as well as miscarriage and stillbirth, multiple births, and congenital malformations. At 4 months post-delivery, participants completed a self-administered questionnaire including information about birth size, maternal complications during pregnancy, and maternal smoking status in the third trimester. At 12 and 24 months post-delivery, participants completed another self-administered questionnaire, which included information related to breast feeding, infant weight, length, head and chest circumferences, smoking status of parents, environmental tobacco smoke (ETS) exposure, pets in the home, day care attendance, infant vaccination, and previous or current medical history of infant allergic diseases (eczema, wheezing, and allergic rhinoconjunctivitis symptoms), infectious diseases, and other diseases. ETS exposure was defined as a self-reported positive response of whether a smoker was in the place where children lived their daily life at both 12 and 24 months of age.

2.3. Assessment of infant allergic diseases

Infant allergies that developed during the first 12 months of life and from months 12–24 were assessed based on the mothers' self-administered questionnaires that were obtained twice, at 12 and 24 months post-delivery. Allergic diseases were defined using a modified part of the Japanese version of the International Study of Asthma and Allergies in Childhood (ISAAC) Phase Three questionnaire. In this study, we estimated eczema based on positive answers to all three of these questions: “Have you (has your child) had this itchy rash at any time in the past 12 months?”, “Have you (has your child) ever had a skin rash which was coming and going for at least 6 months?”, and “Has this itchy rash at any time affected any of the following places: the folds of the elbows; behind the knees; in front of the ankles; under the buttocks; or around the neck, ears, or eyes?” Wheezing was based on a positive answer to the question: “Have you (has your child) had wheezing or whistling in the chest in the past 12 months?” Current allergic rhinoconjunctivitis symptoms were based on all positive answers to both of these questions: “In the past 12 months, have you (has your child) had a problem with sneezing or a runny or blocked nose when you (he/she) did not have a cold or the flu?” and if yes, “In the past 12 months, has this nose problem been accompanied by itchy watery eyes?” (Asher et al., 2006). We also defined total allergic diseases as cases with at least one of the following symptoms: eczema, wheezing, allergic rhinoconjunctivitis symptoms.

2.4. Measurement of PFAA concentrations in maternal plasma

Detailed sampling and laboratory methods for analysis of PFAAs have been previously described (Okada et al., 2013). In brief, a 10-mL blood sample was taken from the maternal peripheral vein between 28 and 32 weeks of pregnancy. Maternal plasma was analyzed using ultra-performance liquid chromatography coupled to triple quadrupole tandem mass spectrometry instrumentation (Waters, Tokyo, Japan).

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