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Effect of Inhaled Allergens and Air Pollutants on Childhood Rhinitis Development

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Keywords

Allergen sensitization; Traffic-related air pollution; allergic rhinitis

Rhinitis is a common pediatric disease with upper airway infection in which allergic rhinitis (AR) affecting 9.7% children in the United States [(1)]. Some rhinitis symptoms are related to allergen exposures. Both epidemiologic and experimental studies have documented that exposure to traffic-related air pollutant (TRAP) increases the risk of allergen sensitization and allergic diseases [(2–4)], which suggested that allergens and TRAP may have synergistic effects on the development of rhinitis. TRAP refers to exposure to primary emissions from motor vehicles including carbon monoxide, nitrogen oxides (NOx) and particulate matter (PM). However, it is not feasible to measure all the components of TRAP, residential distance to the nearest freeway or main road has been accepted as one surrogate of TRAP using methods described previously [(5)]. We previously reported that children living near major roads had increased risk of respiratory diseases, including wheeze [(5)], respiratory hospital admission, as well as significant deficits in lung function growth rate [(6)]. These

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findings were also supported by biological evidence, which showed DEP enhances the allergen-specific IgE response in animal studies [7]. In the present study, using data from Southern California Children's Health Study (CHS), we assessed the individual and joint effects of air pollutants with allergen sensitization on development of rhinitis in children.

From 5,277 CHS participants enrolled during 2002–2003, 695 children with and without asthma were selected for a sub-study [5]. With parents' consent, 232 underwent skin prick test (SPT) for selected common allergens including pollens (olive, coast oak, ragweed, thistle, timothy, and bermuda) and indoor allergens (dust mite mix, cat, dog, mouse, cockroach, and aspergillus). Sensitization was defined by positive SPT to any of allergens mentioned above. Using validated ISAAC questionnaire [8], we excluded children who provided affirmative answer to "In the past 12 months, has your child had a problem with sneezing or a runny or blocked nose when he/she DID NOT have a cold or flu" at baseline. 67 children were kept and followed for up to 8 years for new onset rhinitis identified by affirmative answer to the same question in ISAAC questionnaire updated annually. Other information including demographic characteristics, personal and family history of asthma and other respiratory conditions were obtained from questionnaire completed at baseline.

Based on results from a previous CHS cohort showing respiratory health associations on the spatial scale, residential distance to the nearest freeway was categorized as <500m and ≥500 m. Ambient levels of NO_2, particulate matter with aerodynamic diameter <10 micrometer (PM_{10}) and 2.5 micrometer ($PM_{2.5}$), and O_3 were measured continuously at monitor station in each community and annual average levels prior to baseline were calculated. Two times of standard deviation of ambient air pollutants (NO_2-12 ppb; $PM_{2.5}$-10 μg/m^3; PM_{10} - 21 μg/m^3; O_3; 14 ppb) were used as the unit to measure the hazard ratio.

Cox proportional hazards models were used to investigate the association between allergen sensitization, or/and air pollutants, and the risk for developing rhinitis. All models were adjusted for age, sex, and asthma status at the time of SPT assessment. For those showing evidence for a potential interaction, joint modeling of sensitization and TRAP exposure was further performed.

Among the 67 participants, 17 (25.4%) were identified as sensitized to at least one allergen in the tested SPT panel including 5 were sensitized to both pollens and indoor allergens. Eleven children were sensitized to more than one tested allergens. During follow up, 36 (53.7%) children developed rhinitis. Thirteen of the 17 (76.5%) sensitized children developed rhinitis. It was found that children who had any allergen sensitization were 2.52 times (95% CI: 1.23–5.15) as likely to develop rhinitis as those who had no allergen sensitization. The risk of rhinitis increased with the number of allergens to which a child showed sensitization (P_{trend}<0.001). No statistically significant associations were observed between the occurrence of new onset rhinitis with ambient PM_{10} (HR=1.21; 95% CI: 0.54–2.71), $PM_{2.5}$ (HR=1.19; 95% CI: 0.54–2.59), NO_2 (HR=0.95; 95% CI: 0.48–1.89), or O_3 (HR=0.93; 95% CI: 0.48–1.81).

A joint model was further performed to evaluate the effects of sensitization and proximity to roadways. After adjustment for age, sex and asthma, children who had any allergen

sensitization and lived within 500m of a freeway were 8.13 times (95% CI: 2.81–23.53) as likely to develop rhinitis as compared with non-sensitized children residing more than 500m from a freeway (Table 1). Children with sensitization to any indoor allergen and who lived within 500m to a freeway were 7.64 times (95% CI: 2.55–22.87) as likely to have new-onset rhinitis as non-sensitized children who lived more than 500m from a freeway. No other significant interactions on multiplicative scale were found between allergen sensitization and ambient air pollutants.

The results from this study indicate that the risk of developing new-onset rhinitis increased among children with allergen sensitization and even higher among those with both allergen sensitization and living closer to freeway in Southern California. This type of rhinitis is most likely to be allergic rhinitis[8]. The finding is consistent with other studies showing TRAP is associated with the development and exacerbation of respiratory disease, especially among children [9, 10], suggesting that increasing levels of TRAP may, in part, be responsible for the global increasing trend of allergic respiratory disease.

However, there were some limitations. In this study, only 67 children were eligible to be followed for new onset rhinitis and the SPT test was only performed at baseline due to limited funding. NO_2, $PM_{2.5}$, PM_{10} and O_3 were collected at community level, which could not reflect the actual person level exposure. Further analysis of associations with specific allergens is needed in a larger population.

In conclusion, this prospective study supports the hypothesis that TRAP and aeroallergen exposure during childhood may increase the risk of new-onset rhinitis. More research is needed to support public health policy interventions to reduce TRAP and individually environmental control for the primary prevention of allergic rhinitis.

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Abbreviations

AR	Allergic Rhinitis
TRAP	Traffic-Related-Air-Pollution
NOx	nitrogen oxides
NO_2	Nitrogen Dioxide
O_3	Ozone
DEP	Diesel Exhaust Particles

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Table 1

Joint Effect of Air Pollution Indicated by Proximity to the Nearest Freeway and Allergen Sensitization on Onset of AR among Study Population during up to 8 Years of Follow-up

Sensitization	Residential distance from the nearest freeway			
	>500m		≤500m	
	N	HR* (95% CI)	N	HR* (95% CI)
Any allergen†				
None	41	Reference	9	1.29 (0.45, 3.70)
Yes	11	1.67 (0.68, 4.09)	6	8.13 (2.81, 23.53)
Pollen‡				
None	44	Reference	11	1.68 (0.68, 4.18)
Yes	8	1.32 (0.49, 3.60)	4	10.99 (2.95, 40.94)
Any indoor allergen§				
None	47	Reference	10	1.50 (0.57, 3.94)
Yes	5	3.06 (0.83, 11.26)	5	7.64 (2.55, 22.87)

*Adjusted for age, sex and asthma

†Any allergen included positive SPT to any of allergens mentioned in pollen and indoor allergens

‡Pollen sensitization was defined by positive SPT to one of the following pollens: olive, coast oak, ragweed, thistle, timothy, and bermuda.

§Indoor allergen sensitization was defined as sensitization to any one of the following indoor allergens: dust mite mix, cat, dog, mouse, cockroach, Aspergillus.

HR: Hazard Ratio; CI: Confidence Interval