

Home air-conditioning, traffic exposure, and asthma and allergic symptoms among preschool children

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Epidemiological data suggest that traffic exposures can influence asthma and allergic symptoms among preschool children; however, there is no information on risk reduction via home air-conditioning (AC). The aim of this study is to evaluate the associations of self-reported traffic densities with asthma and allergic symptoms among preschool children and determine whether AC is an effect modifier. A cross-sectional study adopting an expanded and modified ISAAC – International Study of Asthma and Allergies in Childhood conducted on randomly selected 2994 children living in homes without any indoor risk factors. Specific information on demographics, indoor home risk factors, and traffic variables were obtained. Adjusted prevalence ratios (PR) and 95% confidence interval (CI) were determined by Cox proportional hazard regression model with assumption of a constant risk period controlled for covariates. We found dose–response significant relationships between validated self-reported traffic densities and asthma and rhinitis symptoms. Among children sleeping in non-air-conditioned homes, there were stronger associations between asthma and rhinitis symptoms studied. PRs for heavy traffic density were 2.06 for wheeze (95% CI 0.97–4.38), 2.89 for asthma (1.14–7.32), 1.73 for rhinitis (1.00–2.99), and 3.39 for rhinoconjunctivitis (1.24–9.27). There were no associations found for children sleeping in air-conditioned homes. Our results suggest that AC in the bedroom modifies the health effects of traffic among preschool children. This finding suggests that attention should also be paid to ventilation characteristics of the homes to remediate health-related traffic pollution problems.

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Although there is a strong body of evidence that asthma and allergies prevalence among children are associated with traffic exposure (e.g., 1–3), not all surveys have shown similar relationships (4, 5). In most studies, exposure measurements are often conducted using outdoor sampling points that serve as surrogate exposure of the children (2, 6, 7). For epidemiologic studies that rely on outdoor information as a surrogate for exposure, total personal exposure must take into account its outdoor and indoor components.

Exposure studies have reported that home ventilation can affect personal–indoor–outdoor relationships (8). For pollutants of outdoor origin, it has been shown that personal and outdoor concentration ratios, and personal to outdoor regression slopes decrease with decreasing ventilation. Homes with air-conditioning (AC) can have low ventilation because of the fact that outdoor air is not deliberately introduced and that the air exchange rates (AER) because of leakage is typically quite small (9, 10).

In a summer study of 47 US homes in State College, Pennsylvania (11), the authors documented median AERs about six times higher for non-air-conditioned homes compared to air-conditioned homes. Secondary data gathered from a study by Wong and Huang (12) in four homes in Singapore revealed that the AER for homes under natural ventilation to be 4.3/h and AER for air-conditioned homes to be 0.6/h (13). In the same study (12), the amount of outdoor PM_{10} in naturally ventilated (NV) homes was found to be higher than those with AC units. These suggest that homes with AC are anticipated to have less outdoor-to-indoor transport and smaller exposures to indoor pollutant levels of traffic origin. Indeed, personal pollutant concentrations of outdoor origin correlate poorly with outdoor levels for home with AC compared to homes without (8).

Additional evidence found in the literature lend further support to the above-mentioned interpretation that allude to the role of home AC use in reducing health risks of traffic exposures. In the warm and humid climate areas in the US, where homes with AC are most prevalent, the dose-response rate for particulate matter (PM)-induced morbidity was found to be lower than in the milder climate areas, where open windows are used more for ventilation, indicating a modifying effect of AC (14). In the sub-tropics, allergic rhinitis among Taiwanese children was found to be associated with higher non-summer warmth and traffic-related air pollutants but not during the higher summer warmth period (1). Hospital asthma admissions of children under 2 yr old were reported lowest in the months with higher temperatures (15). In the hot and humid countries, lower temperatures seem to have a direct effect, resulting in exacerbations of asthma symptoms (16).

Epidemiologic information relating effect modification of AC on traffic exposure associations with asthma and allergic symptoms are sparse everywhere. The objectives of this study is two-fold; (i) to evaluate the associations of self-reported traffic densities with asthma and allergic symptoms among preschool children and (ii) to determine whether AC is an effect modifier for the association studied. We conduct this study in Singapore which has an increasing population (>4 million) with limited territory, adopting a high-density and high-rise residential strategy integrated within a well-developed transportation system characterized by interweaving roads and highways. It has been estimated that traffic in Singapore is responsible for over 50% of the total pollution.

Methods

The study population was taken from 1.5- to 6-yr-old children attending child care centers (CCC) in Singapore. Drawing from the Ministry of Community Development, Youth and Sports database of 687 CCCs throughout the island, we recruited preschool children from 120 randomly selected CCCs. Details of the questionnaire study have been reported elsewhere (17–19). The selection criteria for this study is all the preschool children in the sampling population ($N = 6794$) who are not exposed to environmental tobacco smoke (ETS), dampness, and wallpaper. ETS, dampness, and wallpaper exposures have been found to be associated with asthma and/or allergic symptoms in earlier studies (17–19). Further, as pet exposure has been shown to interact with outdoor air pollution (20), children with pets at home were excluded from the study. Of the 4759 children (response rate 70.0%) in the sampling population, 3071 children were included in this study.

The parents or guardians of the children were the respondents for the questionnaire survey. The International Study of Asthma and Allergies in Childhood (ISAAC) written questionnaire was used (21). Here, we focus on doctor-diagnosed asthma, current symptoms of wheeze, rhinitis, rhinoconjunctivitis, and eczema. Parents who answered positively to the following questions: 'Has your child ever had asthma?' or 'Has your child had wheezing or whistling in the chest in the past 12 months?' or 'In the past 12 months, has your child ever had a problem with sneezing or a runny or blocked nose when he/she did not have a cold or a flu?' or 'In the past 12 months, has this nose problem been accompanied by itchy-watery eyes?' or 'Has your child had an itchy rash which was coming and going for at least 6 months in the last 12 months' were classified, respectively, as having asthma, wheeze, rhinitis, rhinoconjunctivitis or eczema.

Personal and demographic factors were collected by the questionnaire. These include gender, age, race (Chinese, Malay, Indian, or other), and socioeconomic status (SES). We assess SES by including questions on household income (17–19). Information on traffic exposure was obtained from the questionnaire which includes a self-evaluation of the traffic density outside the residence (low, medium, heavy). We also asked question on the type of ventilation strategies use in the children's bedroom – AC or NV and whether the children lives in the current home since birth (lifetime residence). Information on possible confounders include food

allergy, maternal and paternal atopy, preterm birth, breastfeeding, deliberate avoidance of AC use, respiratory infection (usual cough with colds or a flu), and CCC characteristics.

Empirical measurements of traffic pollutants concentrations have been reported to be higher near roads but declined beyond 150–200 m (3). Thus, we evaluate the validity of reported proximity of residence to roads (within 100 m) by calculating actual distance between the residence and the nearest road using an on-line street directory (<http://www.streetdirectory.com>). We also evaluate the validity of reported traffic densities by measuring traffic pollutants concentrations outside randomly selected homes that was reported as low ($N = 30$), medium ($N = 30$), and high ($N = 30$). One day measurements from 9- to 6-PM for fine particle mass concentration ($PM_{2.5}$) and benzene (BZN) were performed. Measurement of $PM_{2.5}$ was made using DustTrak monitor (TSI, St. Paul, MN) recording at every minute. BZN was sampled actively and analyzed using thermal desorption–gas chromatograph–mass detection (ATD-GC-MSD) techniques. The statistical significance of the differences between the three categories was tested using one-way analysis of variance (ANOVA).

The data were initially analyzed by cross-tabulation and evaluated where appropriate using the chi-square test. We determined prevalence ratios (PR) (the relative risk of having symptoms associated with traffic exposures) and 95% confidence intervals (95% CI) to determine the relationship between traffic variables and prevalence of asthma and allergic symptoms among the children using the modified Cox proportional hazard regression model, with the assumption of a constant risk period. The use of PR and using the modified Cox proportional hazard regression model for cross-sectional studies has been discussed in epidemiologic literatures and detailed elsewhere (22). Core models were adjusted for the child's age, gender, race, SES in multiple regression analysis. Confounding was evaluated by assessing whether the coefficients of prevalence ratio for exposure changed by more than 10% after including an additional covariate to the core model. Effect modification by lifetime residence and AC was assessed by examining the effects of exposure by strata.

Results

Among the 4759 children who responded, there were 3071 children who were not exposed to ETS, dampness, wallpaper, and pets. Table 1 summarizes data on demographics, home envi-

Table 1. Demographics, home characteristics, health, and residential traffic exposures of study participants

	No. (%)†		
	Natural ventilation (NV)‡ (N = 1625)	Air-conditioning (AC)‡ (N = 1398)	All (N = 3071)
Gender			
Male	837 (52)	735 (53)	1572 (51)
Female	788 (48)	663 (47)	1499 (49)
Age* (yrs)			
2	166 (10)	213 (15)	379 (12)
3	304 (19)	292 (21)	596 (19)
4	411 (25)	325 (23)	736 (24)
5	436 (27)	347 (25)	783 (25)
6	218 (13)	171 (12)	389 (13)
Ethnicity**			
Chinese	1283 (79)	1147 (82)	2430 (80)
Malay	166 (10)	75 (5)	241 (8)
Indian	104 (6)	58 (4)	162 (5)
Others	36 (2)	95 (7)	131 (4)
Total monthly income**			
<\$2K	284 (17)	121 (9)	405 (13)
\$2–4K	524 (32)	421 (30)	945 (31)
\$4–6K	358 (22)	288 (21)	646 (21)
>\$6K	372 (23)	495 (35)	867 (28)
Type of housing**			
Public apartment	1416 (87)	1028 (74)	2444 (80)
Private apartment	153 (9)	259 (19)	412 (13)
Single house	29 (2)	87 (6)	116 (4)
Family history			
Maternal atopy	206 (13)	177 (13)	412 (13)
Paternal atopy	183 (11)	165 (12)	348 (11)
Health characteristics			
Wheeze*	225 (14)	223 (16)	448 (15)
Asthma	113 (7)	80 (6)	193 (6)
Rhinitis*	372 (23)	369 (26)	741 (24)
Rhinoconjunctivitis*	92 (6)	115 (8)	207 (7)
Eczema	184 (11)	189 (14)	376 (12)
Food allergy	946 (58)	794 (57)	1740 (57)
Home characteristics			
Lifetime residence	70 (4)	82 (6)	152 (5)
Traffic density			
Light	457 (28)	448 (32)	935 (30)
Medium	554 (34)	475 (34)	1050 (34)
Heavy	117 (4)	101 (7)	226 (7)
Resident height*			
Low	589 (36)	446 (32)	1035 (34)
Mid	462 (28)	383 (27)	845 (28)
High	187 (12)	248 (18)	435 (14)

* $p < 0.05$, ** $p < 0.001$ by chi-square test in comparing the ventilation categories with characteristic/symptoms/exposures.

†The total may not add up because of missing values.

‡Percentage of total in each ventilation category.

ronmental factors, health status, and traffic exposures. The largest group of children was from the 3- and 4-yr olds. The majority of the children were Chinese, lived in high-rise apartments (residential and private) and about two-thirds were lifetime residence. Of those, 15% of all the children had wheeze, 7% had asthma, 24% had rhinitis, 7% had rhinoconjunctivitis

and 12% had eczema. Comparing the different variables with ventilation strategies, significant differences were found for age, ethnicity, SES, type of housing, wheeze, rhinitis, and rhinoconjunctivitis symptoms and height of residence. Of the 3071 respondents, 2994 reported that their residences are not more than 100 m away from nearby road while the remainder 77 did not answer this question (i.e., missing).

From the measurements of distance between residences to the nearby road using the on-line street directory, the mean and median distances were found to be 45 and 35 m, respectively. A total of 52 out of the 2994 residence (1.7%) who reported that their residences are not more than 100 m away from a nearby road were actually more than 100 m. The mean and median distances from residence to nearby road for these misclassified data were 150 and 130 m, respectively. These misclassified data have been removed from further analyses.

The plot of $PM_{2.5}$ and BZN concentrations for reported traffic densities is given in Fig. 1. It appears that the objective measurements are in agreement with self-reported traffic densities. Average and median (in parentheses) $PM_{2.5}$ concentrations were 87.5 (74.2), 74.7 (67.3), and 62.6 (65.6) $\mu g/m^3$ for self-reported traffic densities described as heavy, medium, and low, respectively. For BZN concentrations ($\mu g/m^3$), the corresponding levels were 56.1 (47.0), 42.0 (34.1), and 26.6 (20.4) $\mu g/m^3$ for heavy, medium, and low reported traffic densities, respectively. Differences were statistically significant for BZN ($p = 0.019$) while $PM_{2.5}$ approached significance ($p = 0.068$).

The adjusted PR for the self-reported traffic densities with symptoms are given in Table 2.

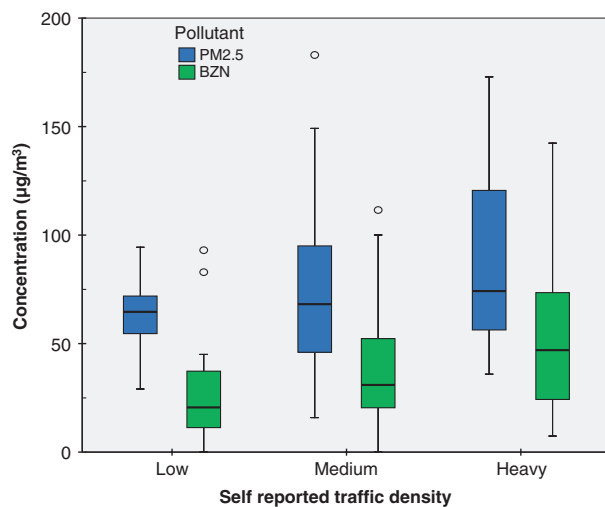


Fig. 1. Box plots of $PM_{2.5}$ and benzene concentrations for self-reported traffic densities.

There is a weak linear exposure–response relationship between self-reported traffic densities and asthma and rhinitis symptoms for both groups: all children, and those with lifetime residence. However, strong linear trends in PR were observed for rhinoconjunctivitis. Among lifetime residents, the PRs for different symptoms were larger compared to corresponding PRs for all children. No associations were observed for prevalence of eczema symptom with traffic densities.

Table 3 showed the adjusted PR for the associations of symptoms with traffic densities stratified into ventilation strategies and lifetime residence. Among children who use AC, there was little evidence of increased risk associated with asthma and allergic symptoms with traffic densities. No linear trends relationship between traffic densities and asthma and allergic symptoms was found. However, strong linear exposure–response trends in PR were observed for asthma and rhinitis symptoms among children using NV. The associations were significant at high traffic densities for asthma, rhinitis and rhinoconjunctivitis with wheeze approaching significance for lifetime residents. Comparing the categories of ‘heavy’ and ‘medium’ traffic densities exposure, respectively, with ‘low’, the adjusted PRs (95% CIs) for wheeze were 1.13 (0.66–1.94) and 2.06 (0.97–4.38); for doctor-diagnosed asthma 1.36 (0.64–2.88) and 2.89 for (1.14–7.32); for rhinitis 1.30 (0.86–1.82) and 1.73 (1.00–2.99) and for rhinoconjunctivitis 1.58 (0.70–3.59) and 3.39 (1.24–9.27). For all children, the associations were significant for rhinitis and rhinoconjunctivitis symptoms only.

Discussion

This study shows that asthma and rhinitis symptoms were associated with self-reported traffic densities. These associations were among the highest for those children exposed to high traffic density and had lived in the current addresses since birth. Significantly, the PR for the associations between asthma and rhinitis symptoms with traffic densities were lower in children using AC compared to those using NV. The latter results provide indication that the use of AC modifies the effect of exposure to traffic pollutants to asthma and rhinitis symptoms. This suggests that AC use reduced the exposure of outdoor traffic pollutants via low ventilation rates and/or some exposure associated with NV may be important in augmenting the effect of traffic pollution.

Generally, preschool children spend majority of their time in homes. Thus, for epidemiologic

Table 2. Adjusted PRs and 95% CI for asthma and allergic symptoms according to traffic exposures

	All children					Lifetime residents				
	Low	Medium		Heavy		Low	Medium		Heavy	
		PR	95% CI	PR	95% CI		PR	95% CI	PR	95% CI
Wheeze*	1	0.89	0.69–1.16	1.08	0.73–1.61	1	1.03	0.72–1.45	1.59	0.96–2.63
Asthma†	1	0.97	0.65–1.44	1.11	0.61–2.03	1	1.06	0.62–1.81	1.58	0.75–3.32
Rhinitis‡	1	1.07	0.88–1.31	1.30	0.97–1.74	1	1.22	0.94–1.58	1.25	0.83–1.88
Rhinoconjunctivitis§	1	1.33	0.92–1.92	1.92	1.17–3.16	1	1.74	1.03–2.93	2.07	1.00–4.30
Eczema¶	1	0.74	0.55–1.00	0.75	0.46–1.25	1	0.78	0.52–1.15	0.53	0.24–1.17

PR, prevalence ratios. Results are in bold when $P < 0.05$.

*Adjusted for gender, age, race, socioeconomic status (SES), type of house, parental atopy, breastfeeding, food allergy, and resident height.

†Adjusted for gender, age, race, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

‡Adjusted for gender, age, race, SES, type of house, parental atopy, breastfeeding, food allergy, and resident height.

§Adjusted for gender, age, race, SES, type of house, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

¶Adjusted for gender, age, race, SES, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

Table 3. Adjusted PRs and 95% CI for asthma and allergic symptoms according to traffic densities stratified by ventilation strategies

	All children					Lifetime residents				
	Low	Medium		Heavy		Low	Medium		Heavy	
	PR	PR	95% CI	PR	95% CI	PR	PR	95% CI	PR	95% CI
NV (natural ventilation)										
Wheeze*	1	1.00	0.68–1.48	1.18	0.64–2.19	1	1.13	0.66–1.94	2.06	0.97–4.38
Asthma†	1	1.59	0.89–2.84	1.68	0.72–3.91	1	1.36	0.64–2.88	2.89	1.14–7.32
Rhinitis‡	1	1.12	0.84–1.51	1.58	1.04–2.39	1	1.30	0.86–1.82	1.73	1.00–2.99
Rhinoconjunctivitis§	1	0.98	0.56–1.72	2.34	1.18–4.67	1	1.58	0.70–3.59	3.39	1.24–9.27
Eczema¶	1	0.70	0.45–1.08	0.84	0.42–1.68	1	0.63	0.37–1.09	0.50	0.17–1.42
AC (air-conditioning)										
Wheeze*	1	0.81	0.56–1.16	1.06	0.62–1.82	1	0.89	0.54–1.47	1.39	0.68–2.86
Asthma†	1	0.54	0.29–1.00	0.96	0.39–2.36	1	0.62	0.27–1.42	0.78	0.17–3.59
Rhinitis‡	1	1.00	0.76–1.31	1.11	0.72–1.71	1	1.10	0.76–1.60	0.98	0.52–1.86
Rhinoconjunctivitis§	1	1.49	0.91–2.46	1.62	0.77–3.40	1	1.57	0.78–3.18	1.38	0.44–4.34
Eczema¶	1	0.76	0.50–1.15	0.66	0.31–1.39	1	0.93	0.52–1.63	0.53	0.16–1.78

PR, prevalence ratios. Results are in bold when $P < 0.05$.

*Adjusted for gender, age, race, socioeconomic status (SES), type of house, parental atopy, breastfeeding, food allergy, and resident height.

†Adjusted for gender, age, race, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

‡Adjusted for gender, age, race, SES, type of house, parental atopy, breastfeeding, food allergy, and resident height.

§Adjusted for gender, age, race, SES, type of house, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

¶Adjusted for gender, age, race, SES, parental atopy, breastfeeding, preterm birth, food allergy, and resident height.

studies that rely on outdoor information as a surrogate for exposure, total personal exposure must take into account its outdoor and indoor components. Exposure studies have reported that home ventilation can affect personal–indoor–outdoor relationships (23). For pollutants of outdoor origin, it has been shown that personal and outdoor concentration ratios, and personal to outdoor regression slopes decrease with decreasing ventilation. Use of AC is associated with low ventilation and without AC, residents are more likely to open their windows especially during periods when temperatures are elevated. Therefore, homes with AC are anticipated to

have less outdoor-to-indoor transport and smaller exposures to indoor pollutant levels of traffic origin. Personal pollutant concentrations of outdoor origin correlate poorly with outdoor levels for home with AC compared to homes without (11). Indeed, homes with AC in Singapore typically do not have ventilation at all.

In addition, the ingress of outdoor allergens (e.g., pollen, fungi) would also lead to higher exposures to aeroallergens in NV rooms resulting in co-exposures with traffic pollutants. In a local study of hospital-attending children aged between 2 and 14, the authors found patients living in households without AC are at increased

risk of mold sensitization and polysensitization (24). One way of interpreting this present study's observation is that developing individuals need exposure to both allergen and some adjuvant-like cofactors to develop sensitization to allergens. Interactions of aeroallergens with traffic pollutants (adjuvants) have been shown in experimental studies to induce allergic disease in the airways in mice and sensitized humans (25, 26). It is highly plausible that these conditions were more favorable to occur in the homes of children which are NV and near traffic sources. Indeed, other epidemiological studies have shown that sensitization and symptoms prevalence were higher among residents near traffic sources (7).

This study considered stratified analysis for children who are lifetime residents because it was anticipated that exposure within this stratum would have been more precisely assigned for the period during which asthma and allergies developed than for children moving later. This is more prominent for asthma symptoms as it has been hypothesized that children may be more vulnerable to traffic exposure during prenatal or infancy period (2, 3). For rhinitis and rhinoconjunctivitis symptoms, the associations were also stronger for children who are lifetime residents. This concurs with the results of Duhme et al. (27) who has reported that associations of allergic rhinitis symptoms among adolescents with traffic were stronger for those who had lived for more than 5 yr in their current dwellings as opposed to those with shorter duration. However, they did not observe similar trends for wheeze.

We did not find any association of traffic exposure with eczema. Although this result coheres with results reported from some studies, others have documented contradictory findings. For example, Brauer et al. (28) did not find an increased risk for Canadian children exposed to higher levels of traffic pollutants, but Lee et al. (29) found an association with traffic-related air pollution in Taiwan. Our finding is unclear, but we hypothesize that this has to do with the tropical hot and humid climate in Singapore. In hot and humid environment, the water content of the stratum corneum is higher (30), which could reduce itching symptoms (31).

A limitation of the study is the lack of indoor data providing evidence that AC reduces the ingress of traffic pollution. Although, it has been shown elsewhere that PM_{10} concentrations were higher for AC compared to NV homes (12), the measurements only included four residences and there was only a small difference in the concentration levels. Future research looking at the effects of AC on asthma and allergies should

include indoor and outdoor air monitoring as well as ventilation rate measurements.

Turner et al. (32) demonstrated an increased risk of a near fatal asthma attack in asthmatics if they owned an AC. One of the conclusions drawn was that decreased ventilation could lead to a build-up of pollutants and allergens indoors. To remove this confounding variable in our study, we have excluded children who are currently exposed to known indoor risk factors of asthma and allergies (17–19).

There was consistency among self-reported traffic exposure from the questionnaire with objective pollutant levels data in this study. This suggests that the collected traffic exposure information is satisfactorily reliable. Still, reporting bias cannot be excluded with certainty. Parents of symptomatic children could have been more worried about the possible negative effects of traffic pollution than parents of children who were asymptomatic and therefore be more prone to overestimate traffic density. This was unlikely because of several reasons; (i) the study was conducted among random samples of child care center children where no specific concerns about traffic pollution near homes were specifically an issue; (ii) there is also no general awareness of potential role of AC to reduce risk of symptoms associated with traffic exposure; and (iii) evidence of linear dose-response trend whereas bias in reporting is expected to affect this relationship. Because this is a cross-sectional study, our findings lacked temporality in that a time relationship between the development of symptoms and traffic exposure could not be ascertained. It will be important to proceed with a follow-up study to establish causal relationships between traffic exposure and asthma and allergies in our population.

Conclusion

These results suggest that AC in homes modifies the health effects of traffic among preschool children; consistent with ventilation theory that AC reduces indoor pollutant exposure of traffic origin and/or interaction effects between co-exposure of allergens and traffic pollutants in NV rooms. This finding suggests that attention should be paid to ventilation characteristics of the homes to remediate health-related traffic pollution problems.

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