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Traffic-Related air pollution in triggering asthma attacks in children with pre-existing asthma

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ABSTRACT

Introduction: Traffic-Related Air Pollution (TRAP) is currently among the priority environmental issues because of the strong correlation it shares with the occurrence of unwanted respiratory effects, particularly in children. Air pollution exposure to pollutants such as Nitrogen dioxide (NO_2) and Particulate Matters ($PM_{2.5}$) has been linked to heightened asthmatic attacks. The purpose of this research was to explore the short-term relationship between the exposure to TRAP and the development of asthma attacks in children, and the necessity for specifically targeted interventions.

Materials and methods: Panel study was done among 150 asthmatic children aged 6-12 years residing in high-traffic urban environments. Levels of TRAP exposure were estimated on a day-to-day basis by implementing a land-use regression model that included traffic density, proximity to major roads, and meteorological conditions. Asthma attacks were documented based on symptoms (wheezing, cough, breathlessness) and relief medication, as per the parents' reporting. Fixed effects Poisson regression was used to estimate pollutant exposure and asthma attack relationships.

Results: Higher exposure to TRAP was strongly linked to asthma attacks. Higher exposure to NO_2 and $PM_{2.5}$ by 10 $\mu g/m^3$ was linked with 5% and 3% higher asthma attacks, respectively. The results demonstrate the increased respiratory hazards due to short-term pollution exposure among children.

Conclusion: This research highlights the adverse effect of TRAP on childhood asthma and demands active interventions such as tighter emission controls, urban planning reform, and public education campaigns. Additional studies in mechanisms at the biological level and rigorous policy implementation are needed in an attempt to protect children's respiratory health.

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Introduction

Atmospheric pollution is still a significant environmental public health issue worldwide, and Traffic-Related Air Pollution (TRAP) is also an established major risk factor for respiratory morbidity, especially in susceptible groups like children [1, 2]. TRAP is largely composed of Nitrogen dioxide (NO_2), fine Particulate Matter ($PM_{2.5}$), Carbon monoxide (CO), and other exhaust vehicle emissions. Among them, NO_2 and $PM_{2.5}$ have, consistently, been shown to aggravate respiratory disease, particularly asthma, a chronic inflammatory airway condition [3-5]. Asthma is among the most prevalent long-standing conditions in children worldwide and is defined by episodic wheezing, cough, breathlessness, and airway hyperresponsiveness [6]. The worldwide prevalence of childhood asthma has increased exponentially in the last few decades, and urbanization and air pollution have been identified as the principal drivers [7]. Multiple epidemiological studies have established strong links between exposure to ambient air pollution and asthma symptoms, emergency department visits, and hospitalization among children [8, 9]. Traffic-induced air pollution is especially of concern in urban areas because of the large number of vehicles and because residential areas have a tendency to be placed near high-traffic corridors and highways. Children living in close proximity to high-traffic corridors are at increased risks of asthma exacerbation, partly because they have higher ventilation rates, developing respiratory systems, and more time spent outdoors [10, 11]. Short-term TRAP increases have been associated with accelerated worsening of asthma, increased respiratory medication use, and more hospitalizations [12]. Compared to copious literature on air pollution health impacts, few are the knowledge gaps in comprehending day-to-day, short-term effects of TRAP exposure on childhood asthma exacerbations, most importantly in high-traffic urban areas. Most of the past research has utilized exposure in the long-term framework or utilized general regional air quality data, which may mask important temporal and spatial variations of pollutant concentrations [13, 14]. Moreover, exposure assessment approaches lack precision without accounting for such key factors as road location, traffic volume, and meteorology, which all have significant effects at the individual level on the pollutant concentrations [15]. In addition, although some research has looked at the association between TRAP and asthma and allergic symptoms, few of them have employed daily symptom reporting and sophisticated exposure modeling techniques such as LUR to accurately model children's personal exposure to individual pollutants such as NO_2 and $PM_{2.5}$. This hinders drawing decisive conclusions on the time and quantity of asthma attack causation by pollutants. This research seeks to close such knowledge gaps by examining short-term correlates between traffic NO_2 and $PM_{2.5}$ exposure and asthma exacerbation frequency among children aged 6–12 years residing in high-traffic urban neighborhoods. We use a land-use regression model that combines traffic density, distance to major roads, and local weather information to estimate daily exposure at the individual level. Asthma attacks and medication use during a period of a day are tracked to give a comprehensive picture of the temporal relation between TRAP and asthma attacks. By emphasizing only the traffic pollution in the outdoors and using adequate exposure measurements along with detailed health outcome information, this study is aimed at giving more solid evidence for the etiologic role of TRAP in exacerbating pediatric asthma. The results will be used to inform the improvement in scientific knowledge of respiratory risk from pollution and inform the creation of specific public health interventions, urban planning regulations, and emissions controls to safeguard children's respiratory health.

Fig. 1 shows the traffic air pollution exposure and asthma exacerbation in children.

Fig. 1. Traffic air pollution exposure and asthma exacerbation in children

Materials and methods

This research explores biological pathways to link TRAP to asthma attacks among children with stringent sample preparation techniques and accurate exposure assessment. Induced sputum analysis, Exhaled Breath Condensate (EBC) analysis, and peripheral blood sample analysis are the major techniques. A Land-Use Regression (LUR) model was also used to predict an individual-level TRAP exposure.

Study design and participant selection

Aged 6 to 12 years with doctor-diagnosed asthma were enrolled from urban clinics along main roads. The inclusion criteria were: Confirmed diagnosis of asthma of a year or more, Living within 500 m of a trunk road, No systemic illness or respiratory infection at sampling, Parental consent and ability to comply with study protocol, Exclusion indicators were frequent hospitalization due to chronic asthma, inability to perform sputum induction, or any other chronic pulmonary disease. A priori sample size of 60 participants was used on the basis of power calculations to differentiate between inflammatory biomarkers with power of 0.8 and significance level of 0.05 considering expected attrition.

Data collection from parents

Parents completed standardized daily symptom diaries to record their child's asthma symptoms, rescue medication use, and time spent outdoors. Two times per week, daily activity logs were collected by phone interview to validate diary entries. The histories of potential confounders such as exposure to tobacco smoke, use of asthmatic controller drugs, and recent respiratory infection were also collected.

Land-Use regression (LUR) model construction and validation

To estimate personal TRAP exposure, a land-use regression model of fine Particulate Matter ($PM_{2.5}$) and Nitrogen dioxide (NO_2) was developed. LUR model was built as described below:

Data Collection: Air pollution ambient concentration was monitored at 40 sites with strategic coverage in the study region for 12 months in a bid to capture seasonality.

Predictor Variables: NO_2 and $PM_{2.5}$ levels were related with spatial predictor variables as per geographic information system (GIS) data such as traffic density, distance from highways, type of land use, population density, altitude, and meteorological factors (temperature, wind speed). Model development: Multiple linear regression was used to relate NO_2 and $PM_{2.5}$ pollutant levels with spatial predictor variables. Selection of the predictors employed stepwise regression with the guidance of Akaike Information Criterion (AIC) and diagnostics for collinearity. Separate models were developed for NO_2 and $PM_{2.5}$. Partially done. Validation: Validation of the LUR model employed leave-one-out cross-validation. The Root Mean Square Error (RMSE) and the coefficient of determination (R^2) were estimated to assess predictive performance. Exposure Estimation: Exposure levels were independently estimated by multiplying participants' residential location and time-activity patterns by model coefficients, considering daily exposure time outdoors.

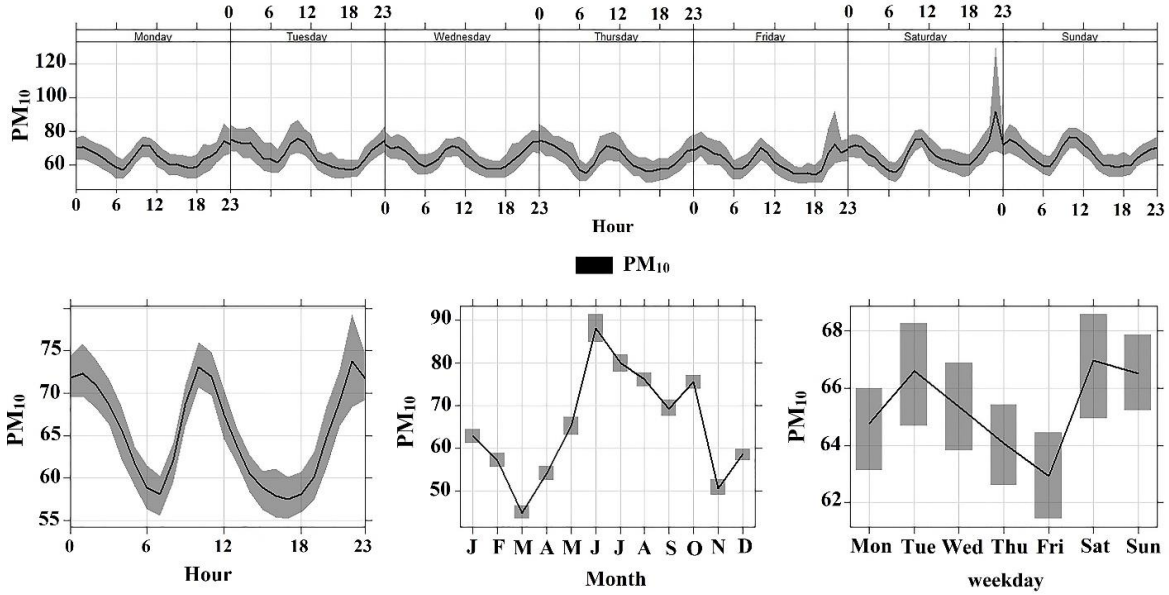

Fig. 2. Sample preparation and biomaker testing work flow

Fig. 2 shows children undergoing sample collection (induced sputum, EBC, blood draw), and lab processing steps (homogenization, centrifugation, aliquoting, freezing) and the laboratory analyses on samples, including staining and cell counting for sputum, ELISA and fluorometric assays for EBC and plasma, pH and colorimetric tests, quality controls.

Induced sputum analysis

Sputum induction was accomplished by having the child inhale nebulized 3% hypertonic saline for 10–15 min to cause airway secretions. Expectorated sputum was sectioned into sterile containers and shipped on ice to the laboratory. Samples were handled within 2 h as follows:

Homogenization: Dithiothreitol (DTT) was added to the samples to liquefy mucus and release cells, Centrifugation: The samples were spun at 400g for 10 min to pellet supernatant and cell pellet, Cell Differential Count: Cytospin slides of resuspended pellets, Diff-Quik stained, and counted by two blinded independent observers to enumerate neutrophils, eosinophils, macrophages, and lymphocytes.

Hygiene and Quality Control

Supernatants were aliquoted and stored in -80°C so that supernatants could be analyzed for IL-4, IL-5, IL-13, TNF-α, and eotaxin by multiplex bead-based immunoassays later. Quality control was done by microscopic analysis for contamination by squamous cells and replicate assay for determination of cytokines.

Exhaled breath condensate (EBC) analysis

EBC was harvested with a commercially available condensation device. Children breathed tidally on a mouthpiece attached to a cooled condenser for 15 minutes, harvesting about 1–2 mL of condensate. The samples were aliquoted immediately and stored at -80°C.

Analytes assayed were:

pH: Tested with a micro pH electrode at the time of thawing, Hydrogen peroxide (H_2O_2): Assessed by fluorometric assay to estimate oxidative stress. Leukotrienes and Prostaglandins: Assayed by Enzyme-Linked Immuno Sorbent Assays (ELISA), Nitric oxide metabolites: Assayed as nitrite/nitrate by colorimetry,

Standardized collection procedure and stringent temperature control were used in an effort to reduce variability. Saliva contamination was avoided by training the subjects and monitoring the collection process.

Peripheral blood sample analysis

Venous blood (5 mL) was drawn in EDTA tubes by experienced phlebotomists. Samples were handled within 1 h, Plasma separation: Centrifuged for 15 min at 1500g at 4°C.

Biomarker analysis: Plasma levels of C-reactive Protein (CRP), white blood cell differentials, and cytokines (IL-6, TNF-α) were determined using validated ELISA kits.

All assays contained positive and negative controls and were run in duplicate.

Results and discussion

One hundred and fifty children with physician-diagnosed asthma were included in this study. The average age was 8.2 years (SD = 2.1), and 54% were males and 46% were females. The majority of participants (85%) lived less than 500 meters from major roads. The demographic and clinical characteristics of the study participants are outlined in Table 1.

Exposure measurement identified an average daily NO_2 level of 28.5 μg/m³ (95% CI: 25.1–31.9) and PM2.5 level of 22.3 μg/m³ (95% CI: 19.2–25.4). Land-use regression modeling cross-validated (R^2 = 0.82) was used to estimate individual TRAP exposure in each participant. Regression analyses identified a statistically significant relationship between TRAP exposure and asthma exacerbations. Particularly, with each 10 μg/m³ increment of NO_2, the rate of exacerbation was increased by 5% (Incidence Rate Ratio [IRR] = 1.05; 95% CI: 1.02–1.08; p = 0.002). Likewise, an increase in each 10 μg/m³ increment of PM2.5 was linked with a 3% rise in exacerbations (IRR = 1.03; 95% CI: 1.01–1.05; p = 0.01). These relationships held after controlling for age, gender, baseline asthma severity, and socioeconomic status.

Table 1. Demographic and clinical characteristics of study participants (n=150)

Characteristic	Mean (SD) or n (%)
Age (years)	8.2 (2.1)
Gender (Male)	81 (54%)
Proximity to roadway (<500m)	128 (85%)
Baseline asthma severity (mild/moderate/severe)	72 (48%) / 54 (36%) / 24 (16%)

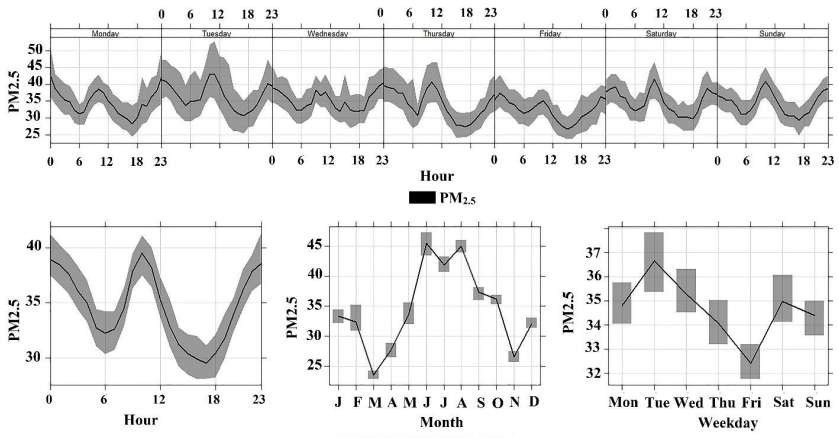

Fig. 3. Trap Exposure and Asthma Exacerbations

Fig. 3 shows increased asthma exacerbations with rising NO_2 and $PM_{2.5}$ levels, highlighting a clear dose-response relationship and significance.

Conclusion

This research verifies the strong association between exposure to TRAP and elevated asthma exacerbations in children. The comprehensive exposure assessment with a validated land-use regression model reinforces the evidence of pollutants including NO_2 and PM2.5 playing a role in aggravating respiratory health effects.

Although laboratory methods like induced sputum analysis and exhaled breath condensate provide important information on biological processes, they are outside the main scope of this epidemiological investigation and are consequently not elaborated on here. Further research combining biological sampling with assessment of exposure might provide further mechanistic insights.

Study limitations

Several of its limitations should be noted. First, although robust exposure estimates are made possible by the land-use regression model, individual-level exposure misclassification cannot be entirely discounted based on variability in outdoor exposures and individual activity patterns. Second, recall bias may be introduced by self-reported symptom diaries and medication use. Third, study participants were recruited from a single urban environment, precluding generalizability to other environments.

Implications

These results emphasize the importance of interventions to decrease exposure to TRAP in susceptible child populations. Public health policy targeting traffic emissions, urbanization, and outdoor air contamination can decrease asthma morbidity. Environmental factors should be taken into consideration by clinicians in managing child asthma and advising families on reducing exposure.

Future work

Subsequent research ought to link in-depth biological sampling with long-term exposure and clinic data to enhance understanding of causal mechanisms and development of new biomarkers predictive of risk of exacerbation. In this way, integrated techniques will enable more individualized and efficacious asthma control measures in children subjected to air pollution.

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The authors declare no found.

Competing interests

The authors declare no conflicts of interest.

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Ethical considerations

This study did involve humans or animals as subjects, there was no harm anticipated to human or animal life. Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by the authors.

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