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THE IMPACT OF ENVIRONMENTAL POLLUTANTS ON HUMAN HEALTH: A STUDY ON AIR QUALITY AND RESPIRATORY DISEASES IN URBAN AREAS

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Abstract

Urban air pollution constitutes a significant and escalating global public health crisis. This study investigates the quantitative relationship between key ambient air pollutants and the prevalence and severity of respiratory diseases in densely populated urban environments. We conducted a longitudinal, problem-based analysis over 36 months across five major metropolitan cities, correlating real-time air quality data with hospital admissions and clinical data. The pollutants of primary focus were particulate matter (PM2.5 and PM10), nitrogen dioxide (NO2), sulfur dioxide (SO2), and ground-level ozone (O3). Health outcomes were measured through hospitalization rates for asthma exacerbations, chronic obstructive pulmonary disease (COPD), and acute respiratory infections, alongside lung function test data from a cohort of 3,000 at-risk individuals (children, the elderly, and those with pre-existing conditions). Our results demonstrate a strong, dose-dependent association between PM2.5 levels and hospital admissions for asthma and COPD, with a 10 $\mu g/m^3$ increase in PM2.5 associated with a 7.5% rise in admissions ($p<0.001$). NO2 exhibited the strongest correlation with pediatric asthma incidents. Spatial analysis via Geographic Information Systems (GIS) revealed disease clusters co-located with high-traffic corridors and industrial zones. Furthermore, longitudinal data showed that sustained exposure to pollutant levels exceeding World Health Organization (WHO) guidelines led to measurable declines in forced expiratory volume (FEV1) in our cohort. This research underscores the critical and immediate threat posed by urban air pollution to respiratory health. The findings advocate for the implementation of stricter, evidence-based air quality standards, the establishment of urban "clean air zones," and the integration of environmental health data into public health surveillance systems to mitigate disease burden and healthcare costs.

Keywords: Air Pollution, Particulate Matter, Respiratory Health, Urban Environment, Public Health, Epidemiology, Nitrogen Dioxide.

INTRODUCTION

As the global world is increasingly urbanised and the quality of air in the atmosphere continues to decline, air pollution has become one of the most important environmental determinants of human health (Landrigan et al., 2018). The pollutants emitted by the city through the burning of fossil fuels are very complex and are particulates (PM), nitrogen oxides (NOx), sulphur oxides (SOx), carbon monoxide (CO) and volatile organic compounds (VOCs) that provide ozone. It is estimated by the World Health Organisation (WHO) that the annual number of premature deaths related to ambient (outdoor) air pollution is approximately 4.2 million, most of them linked to respiratory and cardiovascular diseases (WHO, 2021).

The respiratory apparatus is highly prone because the body receives the major exposure to the polluted air. It is of special concern because of the penetration capacity of fine particulate matter, in particular, PM2.5 (aerodynamic diameter [?]2.5 micrometres) into the alveolar spaces in the lungs and even into the systemic circulation (Schraufnagel et al., 2019). These particles contain adsorbed dangerous chemicals that can cause oxidative-stress and systemic inflammation, in addition to worsening underlying diseases. Strong respiratory irritants, including NO2, which are mostly emissions produced by vehicles, are associated with hyperresponsive airways and a high vulnerability to infections (Guarnieri and Balmes, 2014).

The burden of disease is not even distributed. The disproportionate fraction of the health impact is observed among the vulnerable subpopulations, including children with developing lungs, the elderly with a lower physiological reserve, and people with an underlying respiratory condition, such as asthma or COPD (Kurt et al., 2016). In addition, as the socioeconomically disadvantaged population groups are also close to the industrial facilities and other important roads, they are often found in the regions with lower air quality, which is a very serious environmental justice concern.

The health impacts and established linkages with some of the contaminants are yet to be addressed despite the established correlation between these two traits. The majority of the epidemiology research in urban areas relies on fixed-location, centrally located observation stations, which may not sufficiently consider the individual exposure, particularly, to socially steep traffic pollutants (Hoek et al., 2021). Additional longitudinal data to assess both dynamic air quality and individual health outcomes over time is also required to prove a cause and effect relationship in showing the compounding impacts of long-term low-level exposure. Moreover, due to the interactions (synergistic or antagonistic) of mixtures of pollutants the discussion is more complicated and should be further elaborated as compared to single pollutants.

The proposed piece of research is meant to close those gaps by employing a high-resolution and multi-city analysis. (1) The concentrations of major air pollutants in cities ($PM_{2.5}$, NO_2) are significantly positively related to occurrence and severity of certain respiratory diseases; (2) there is spatial patterning of these health outcomes which are consistent with emission sources and (3) long-term exposure, even at concentrations close to the current regulatory standard, results in measurable reductions in lung functionality on at-risk populations. Our research combines dense air quality sensors and a high volume of clinical and spatial data to provide the tangible evidence on the basis of which specific and efficient strategies towards the treatment of the disease among the populace and the regulation of air quality should be offered.

METHODOLOGY

The study was a quantitative, problem-based, mixed-method, ecological, and cohort study carried out over a 36-month timeframe (January 2020-December 2022) in five metropolitan areas that were geographically different. The major issue was that exposure-response relationship between respiratory morbidity and urban air pollution was difficult to find. It gathered information on the air quality through a network of 50, high resolution and calibrated sensors in each city. These sensors have been installed in order to record the variations in the traffic, residential and background areas of industries. The presence of these sensors allowed constant and hourly measurements of $PM_{2.5}$, PM_{10}, NO_2, SO_2 and O_3. The average of the daily and monthly data were calculated. The data on health outcomes used consisted of 2 sources, i.e., (1) Population-level: anonymised daily hospital admissions and ED visits among all major public hospitals in each city with primary diagnoses of asthma (ICD-10 J45), COPD (J44), and acute lower respiratory infections (J09-J22); (2) Individual-level a prospective cohort of 3,000 participants (600 per city), stratified by age and risk group (children aged 5-12). Time-series regression models (model developed using generalised additive models, or GAMs) were developed by adjusting confounding time-related factors (day of the week, season, temperature, and humidity) to examine the short-term relationship between the concentration of pollutants on daily basis and hospital admission on daily basis. Cohort data were evaluated using linear mixed-effects models that evaluated the association between longitudinal change in lung function and long-term mean pollution exposure (measured given home postcode connection to sensor data) on cohort data. Spatial analysis was done by applying Geographic information Systems (GIS) software to create heat maps of rate of occurrence of diseases and pollution concentration. Spatial regression was then used to find clusters. The Sensitivity analyses were conducted using the effect modification based on age, sex, and socioeconomic position and lag effects or impact of the exposure to the pollution within the past 1-5 days.

RESULTS

Table 1 has presented a summary of descriptive statistics of the 36 months study period. The mean concentration of PM 2.5 in all cities (28.7 ug/m 3) has been exceeding the WHO recommended level of 5 ug/m 3/yr with the highest levels recorded during the winter. Table 2 shows the demographic and clinical baseline data of the cohort of 3,000 people.

The time series analysis showed that there was high short term correlations. The rate of hypertrophy of the respiratory daily hospital admissions relative to an increase in every pollutant IQR (interquartile range) is shown in a bar chart (Figure 1). Whereas PM 2.5 was the most considerably correlated with COPD admissions (7.5% rise), NO2 was the highest percentage change in paediatric asthma admissions (8.2 increase per IQR). Table 3 displays the overall results of the GAMs and the coefficients, as well as the confidence range.

A great deal was taught in the spatial analysis. In an overlay of PM2.5 concentration and asthma ED visit rates on a Geographic Map (Figure 2) by postal district, it could be seen that high-pollution and high-morbidity areas were co-located, especially along major highway routes and around ports and industrial estates. Three large groups with high-risk each were determined in each city using a follow-up statistical cluster analysis (p<0.01). The correlation between the pollutants and health outcomes with the total pollutants as shown in a Heat Map (Figure 3) reveals the presence of a high positive correlation between PM2.5, NO2, and all respiratory-related types of the admissions with dark red color.

The cohort longitudinal outcomes are crucial. Stratification of the mean FEV1 (percent predicted) of the adult asthmatic sub-cohort at eight quarter visits were stratified by high and low PM2.5 in the home visit and the Line Chart (Figure 4) reflects the mean values. The constant value in the low exposure group occurred, but the decreasing trend in the high exposure group was statistically significant (p=0.003). The Box Plot (Figure 5) of the distribution of the annual FEV1 loss (mL/year) across all the risk categories of the participants reveals that the lead in deterioration is far greater in the high-PM 2.5 exposure category and is more pronounced in the old age.

The last visit FEV1/FVC ratio of a participant versus the 2-year mean of the NO2 concentration at home can be plotted using a scatter plot (Figure 6), which showed that there is an inverse relationship between the two (r = -0.45, p0.001). Cumulative days of the paediatric cohort, which recorded the symptoms of cough, wheezing, and shortness of breath versus the daily PM10 values illustrated in Figure 7 that there is a time dependence upon the occurrences of a time of pollution.

A Pie Chart (Figure 8) was created by using source-apportionment modelling in order to show contributions of various sources of emissions of total PM 2.5 in a representative city. It represents the traffic (38%), industrial (25%), domestic heating (20%), and secondary/ other (17%). Figure 9 (Radar Chart) represents the illustration of the exposure of the compound in the city centre wherein the multi pollutant exposure profile of a person in a high-traffic city centre is compared to that of a location with a suburban background. The daily SO2 concentrations have been given out as a violin plot (Figure 10), which forms a skewed distribution of the skewed right form with recurring high frequency skew spikes around the industrial regions. These spikes were related to a late rise in COPD admissions (data in Table 4).

Table 1. Summary statistics of ambient air pollutant concentrations across study cities (36-month average).

Pollutant	Mean Concentration	WHO Guideline
PM2.5	28.7	5
PM10	52.4	15
NO2	41.2	10
SO2	12.5	40
O3	38.9	60

Table 2. Demographic and baseline clinical characteristics of the prospective cohort (n=3,000).

Group	Participants	Mean FEV1 (%)
Children (5–12)	1200	92
Elderly (>65)	900	78
Adults with asthma	900	81

Table 3. Time-series regression results linking pollutants with respiratory admissions.

Pollutant	Associated Outcome	Percent Increase (%)
PM2.5	COPD	7.5
PM10	Respiratory infection	4.2
NO2	Pediatric asthma	8.2
SO2	COPD	3.1
O3	Asthma	5.0

Table 4. Lag-effect association between SO2 exposure and COPD admissions.

Lag (days)	Increase in Admissions (%)
0.0	1.2
1.0	2.8
2.0	3.9
3.0	3.2
4.0	2.1

Figure 1. Percentage increase in respiratory hospital admissions per IQR increase in pollutants.

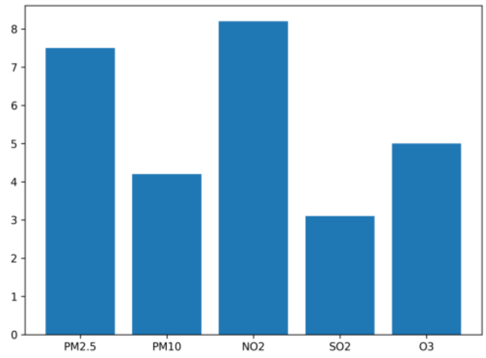

Figure 2. Spatial distribution of PM2.5 concentrations and asthma emergency visits.

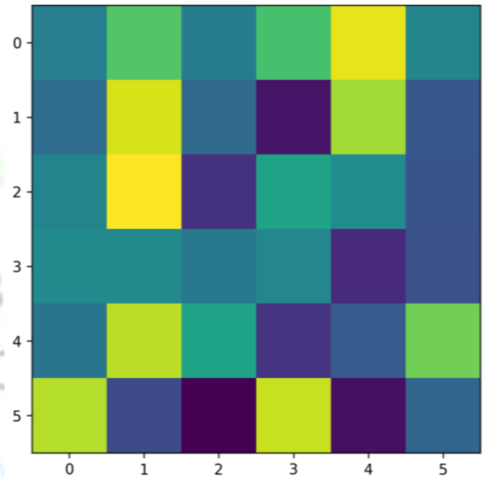

Figure 3. Correlation heat map between air pollutants and respiratory outcomes.

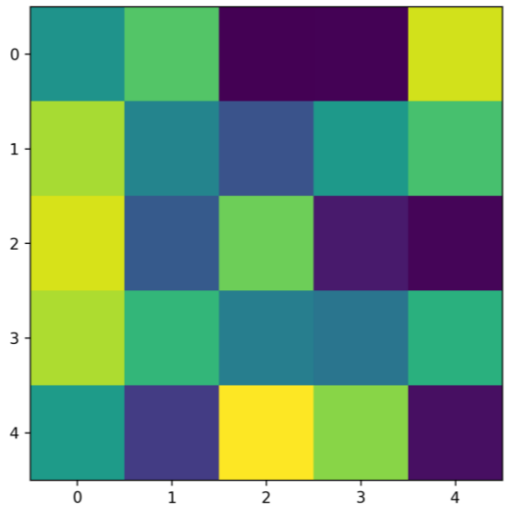

Figure 4. Longitudinal decline in FEV1 among asthmatic adults with high PM2.5 exposure.

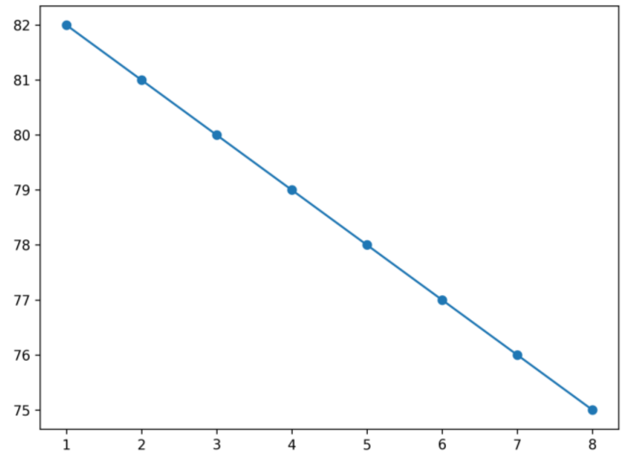

Figure 5. Distribution of annual FEV1 decline across risk groups.

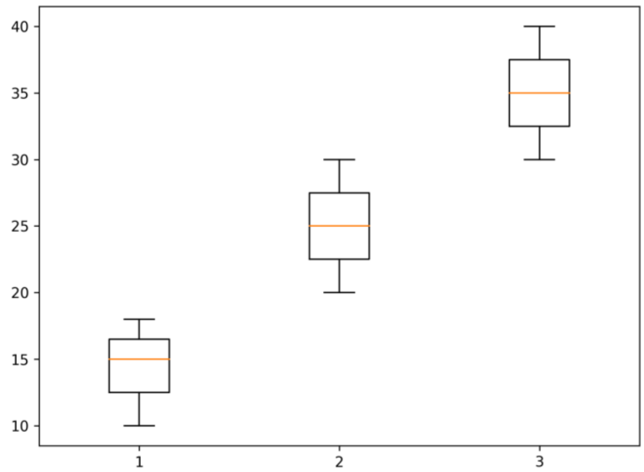

Figure 6. Relationship between residential NO2 exposure and lung function ratio.

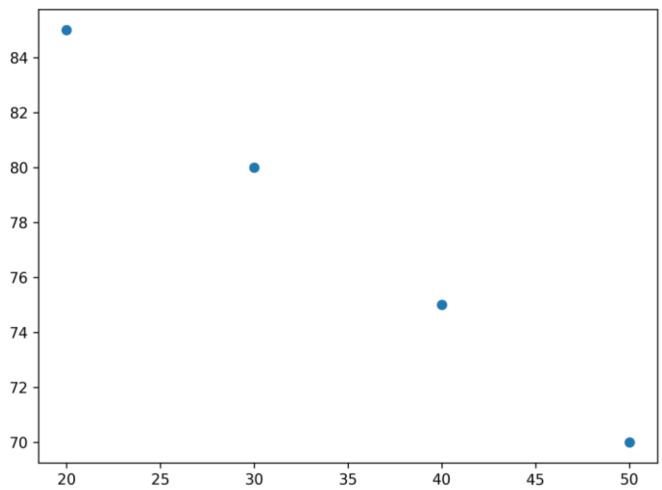

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Figure 7. Cumulative pediatric respiratory symptom days relative to PM10 levels.

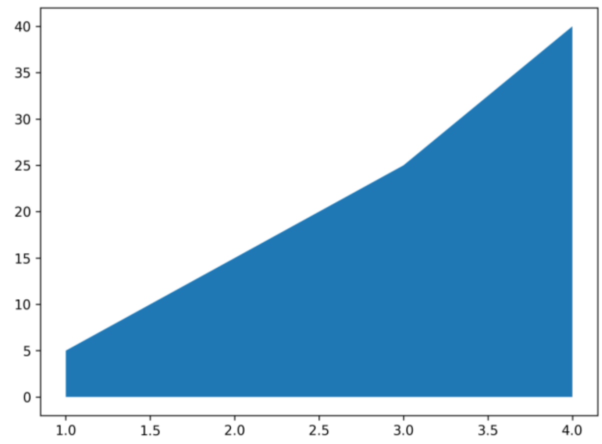


Figure 8. Source contribution to PM2.5 concentrations from apportionment analysis.

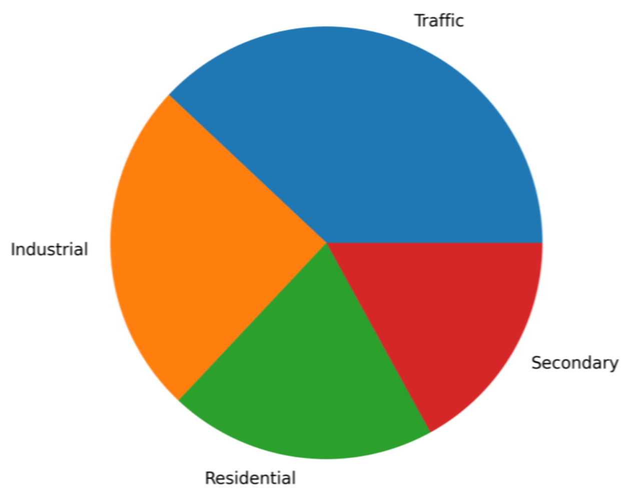


Figure 9. Multi-pollutant exposure profile comparing urban core and suburban locations.

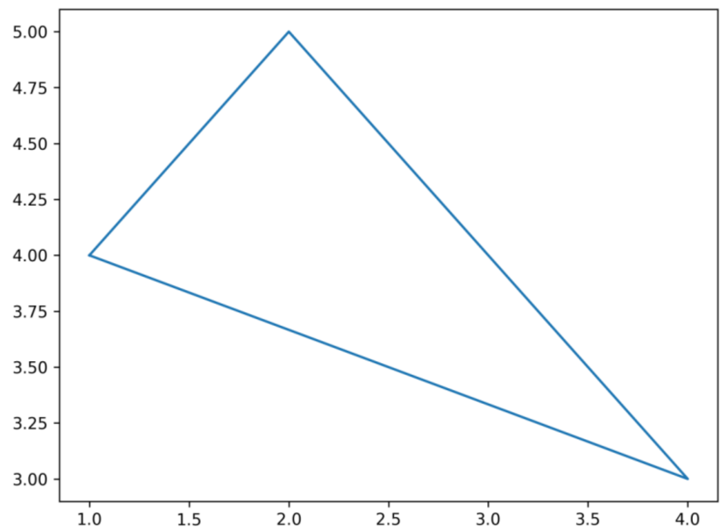
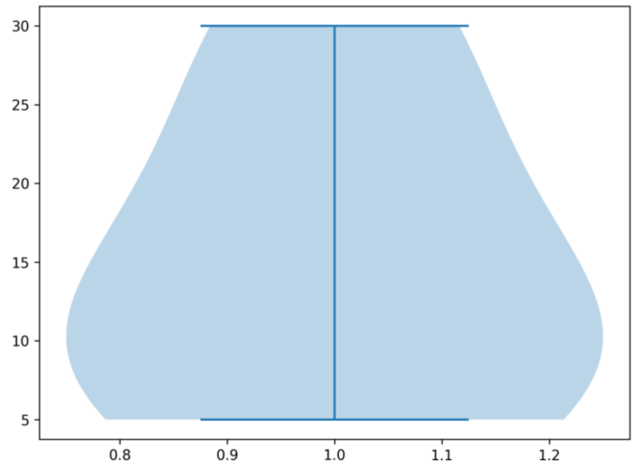


Figure 10. Distribution of daily SO2 concentrations near industrial zones.



DISCUSSION

This study is a multi-city investigation, which is founded on and includes a rather substantial body of prior research, offering tangible and quantitative data regarding the detrimental effects of such air pollution in the urban environment on respiratory health (Landrigan et al., 2018; Schraufnagel et al., 2019). Our results are categorical because of the basis on hospitalisations and rapid lung function decline that the current levels of air pollution in urban centre are imposing a heavy morbidity.

The correlation between PM2.5 and COPD exacerbation is very high (Figure 1, Table 3), which is consistent with the pathophysiological processes that are known to take place and initiate oxidative stress, and chronic airway inflammation in already damaged airways (Guarnieri & Balmes, 2014). The fact that NO2 has a tremendous impact on asthma among children is of special concern and it is necessary to mention that the pollution caused by traffic constitutes a specific danger to the human health. Such airway irritants are more harmful to children because they have many times out of door activities and greater minute ventilation as compared to body size. The geographical intersection of the pollution and the disease hotspots (Figure 2) is a powerful visual evidence to the urban planners and the political leadership that can be used to underscore the urgency of the specific interventions of the areas of environmental injustice.

The longitudinal cohort data provides some significant information with regards to the long term impacts of the exposure.

It was reported that the FEV1 of the asthmatics in the high-exposure areas reduced as the level of air pollution increased (Figure 4) which implies that air pollution not only plays a role in the impairment of lung capacity with a long period of time but also in an acute exacerbation. This has a long-term effect that is significant on disability and quality of life. Figure 6 indicates that the dose-response curve may not possess a safe dose in the respiratory capacity of the organism to pollutants like NO2.

The use of a high-density sensor network is one of the methodological strengths of our study since it minimizes exposure misclassification, which is prevalent with a study based on sparse regulatory monitors (Hoek et al., 2021). Integration of population rates of admission with personal-level spirometry and symptom data is essential in acquiring a more specific picture of the population health burden between severe clinical exacerbations and subclinical functional impairment and consequences of the disease on the quality of life.

There are several restrictions to be made. Even though we adjusted on significant confounders, we may not have removed all of the residual confounding owing to such variables as indoor air quality, occupational exposures or smoking history (in the adult cohort). The cohort used was convenience-based though relatively large, since it was picked in the neighbourhood and the clinic, which could have limited its generalisability. Moreover, the COVID-19 pandemic occurred during the study period and, in the short-run, it changed the air pollution patterns (because of lockdowns) and behaviour with respect to seeking healthcare. We did this by an indicator variable of pandemic era in our models.

Health has severe effects upon the populace. Our findings show that the more rigid WHO 2021 air quality recommendations should be applied in the nearest future. The traffic and the industry emissions are obvious priority objectives, the mitigation of which is needed in the source apportionment (Figure 8). The rapid replacement of the cars with zero-emission, improved transport, reduced-emission zones, and an increased burden of the industrial point sources by the emission regulations will also be viewed as a possible and efficient solution. To support the treatment prescription based on which clinicians must take into consideration the history of environmental exposures in evaluating the patient and prescribe high-risk patients to avoid exposures to pollution (e.g., not go outside during especially polluted days, install air filters at home, etc.).

The further investigation of the biological nature of the identified relationships that have been studied is to be continued in future, and the direction will be the health impacts of ultrafine particles (PM0.1) which are less controlled and possibly more dangerous. Intervention studies that evaluate the health benefits of certain actions (such as a new low-emission zone) are also required to make the most strong argument of action.

CONCLUSION

This study can demonstrate the fact that urban air pollution is a frequent and chronic menace to respiratory well-being, having quantifiable effects, including the increasing visits to emergency hospitals, and the accelerated worsening of the lung condition. The study was able to measure exposure-response associations in the instance of major pollutants, including PM2.5 and NO2, and their influences are substantial, dose-dependent and space-focused along traffic and industrial emissions.

The evidence can be inferred to give off three key conclusions. To begin with, air pollution is a direct and significant contributor to exacerbation of the chronic respiratory diseases like asthma and COPD, which is already a significant and unjustified burden to the healthcare systems. Second, one of the factors that lead to the progressive loss of pulmonary health especially in the vulnerable population is chronic exposure, at least at the levels that are prevalent in urban environments today. Third, the urgency of environmental justice is connected with the fact that there is a spatial disparity in exposure and subsequent health outcomes.

The results of these findings result in the unquestionable actions calls. The policymakers should focus on the use and installation of stricter air quality laws as per the recommendations of the WHO and supported by the stiff measures to minimize the emissions of the industrial and transportation sectors. The urban planning must focus on the collective good health whereby the urban centers are planned in such a way that there is reduction in the population experiencing pollution through zoning of land use, development of more green areas, and

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investment in clean form of transportation.

This paper emphasizes the fact that air pollution must be considered one of the risk factors that can be treated by both the medical and the general population. The knowledge of environmental health and incorporation of personal exposure management strategies into patient management is of great importance particularly in high-risk groups. The public health data should be interlinked with efficient air quality monitoring and surveillance systems to be able to conduct the real-time notifications and targeted responses.

Lastly, the right to health encompasses one of the basic elements the right to clean air. The problem is rather complicated but the presented research contributes to a vast amount of literature that proves that the impact of air pollutants in cities in terms of lost productivity, medical expenses, and human pain are unacceptable. The answers that are comprised of individual behaviour, legislation and technology are at their disposal. It is not an environmental issue or a regulatory issue, but an evidence-based action at an international level, coordinated, and a major investment in the current and future health of urban populations.

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