Prevalence of Diabetes Mellitus and Exposure to Suspended Particulate Matter

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Background. Evidence from various epidemiological studies has shown an association between particulate matter 2.5 ($PM_{2.5}$) and diabetes mellitus. A prospective study from the United States reported that exposure to $PM_{2.5}$ alters endothelial function, and leads to insulin resistance and reduction in peripheral glucose uptake. There is a paucity of data on the relation between air pollution and diabetes in low- and middle-income countries.
Objectives. To estimate the prevalence of type 2 diabetes among people living in areas with higher exposures of suspended $PM_{2.5}$ compared to people living in areas with lower exposures in Chennai, Tamil Nadu, India.
Methods. A cross-sectional study was carried out in two areas of Chennai city. The $PM_{2.5}$ affecting vulnerable areas were stratified from a list of air quality monitoring stations in Tamil Nadu Pollution Control Board and Central Pollution Control Board. The highest and lowest areas of exposure were selected from the list. Households were randomly selected for the study. A total of 201 (67 male, 134 female) individuals from a high exposure area (HEA) and 209 (76 male 133 female) individuals from a low exposure area (LEA) were recruited for the study. Adults over 18 years of age were screened for random capillary blood glucose (RCBG) by glucometer (OneTouch Ultra).
Results. The prevalence of diabetes (34.8% vs 19.6% p =0.001) was 77.5% higher among people living in areas of high particulate matter exposure compared to people living in less exposed areas. Multivariable logistic regression analysis showed that age, gender, residential area, and family history of diabetes were significantly associated with the prevalence of diabetes ($p<0.05$).
Conclusions. The present study indicates a link between high levels of exposure to $PM_{2.5}$ and diabetes mellitus. Further prospective studies on populations exposed to elevated pollution are needed to establish whether this association has a causative link.
Participant Consent. Obtained
Ethics Approval. The study was approved by the Ethics Committee of the Prof. M Viswanathan Diabetes Research Centre, Chennai, India
Competing Interests. The authors declare no competing financial interests.
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Introduction

The World Health Organization (WHO) reports 4.2 million annual deaths due to exposure to ambient outdoor air pollution worldwide.[1] Vehicular pollution and surface dust are the major sources of air pollution in urban areas. The air quality level of more than 80% of the people living in urban areas exceeds WHO limits.[1] According to WHO air quality guideline values, particulate matter 2.5 ($PM_{2.5}$) annual mean below 10 µg/m^3 and $PM_{2.5}$ 24-hour mean below 25 µg/m^3 are considered to be good air quality.[2] Unfavorable effects of air pollution on human health have been reported in many studies. Air pollution has been shown to indicate the major contributing factor for the development of cardiopulmonary disease.[3-6] The association between pulmonary diseases and air pollution is well known and air pollution is a major cause of mortality.[7-9] However, the association of air pollution with other metabolic dysfunctions is not yet known. Air pollution is an emerging contributing factor to the global burden of stroke in low- and middle-income countries.[10,11] Due to epidemiological transitions, patterns and causes of diseases change over the years. Global epidemiological studies have investigated the relationship between air pollution and the risk of developing diabetes.[12,13] The association with environmental pollution as a risk factor for diabetes has been neglected.[14] Studies report that ambient air pollution alters adipose inflammation, insulin resistance, and endothelial function in humans and animals and also causes a reduction in peripheral glucose absorption in humans.[15-18] Plasma inflammatory markers increase in response to higher $PM_{2.5}$ exposure in individuals with diabetes.[19] Multiple studies have demonstrated that long term exposure to $PM_{2.5}$ contributes to the incidence of diabetes along with insulin resistance, changes in β cell function, and adiposity.[20-23] As a main component of haze, smoke, and motor vehicle exhaust, $PM_{2.5}$ is dangerous because of its small size and ability to infect critical human organs in the respiratory and vascular systems.[24]

Abbreviations			
HEA	High exposure area	*RCBG*	Random capillary blood glucose
LEA	Low exposure area	*WHO*	World Health Organization
$PM_{2.5}$	Particulate matter 2.5	*BP*	Blood pressure
		HTN	Hypertension

The rapidly increasing Indian population, urbanization, and industrialization have adversely affected human health. A drastic increase in the number of vehicles has resulted in increased emission of air pollutants. According to the Chennai Metropolitan Development Authority, the motor vehicle population has increased at a very high rate over the last few decades. The total vehicle population has increased to 10.38 million in 2016 up from 2.4 million in 2009.[25]

A recent study from India reported that the prevalence of diabetes is higher in socio-economically deprived groups in urban areas of economically developed states in India.[26] There is also evidence that individuals living in polluted cities have a lower life expectancy and are more likely to die prematurely than those living in less polluted areas.[27] It is unclear what causes diabetes other than genetic and lifestyle factors. Hence, the present study aimed to evaluate the association between $PM_{2.5}$ and the risk of developing diabetes by assessing the prevalence among those living in areas with different levels of $PM_{2.5}$ exposure in Chennai, Tamil Nadu, India.

Methods

A cross-sectional study was carried out in two areas of Chennai city. A high exposure area (HEA) of $PM_{2.5}$ was used as the study area and compared with a low exposure area (LEA) of $PM_{2.5}$. Study criteria included residence in the high or low exposure areas for more than 7 years. The study was carried out between September 2017 to March 2018. The study areas were selected after analyzing the secondary data from the Tamil Nadu Pollution Control Board, Central Pollution Control Board and the National Air Quality Monitoring Programme. Air quality data of Chennai city was analyzed from a list of total air quality monitoring stations in the city from 2016 to 2017.[28] Under these three governing bodies, there are 12 monitoring stations in Chennai. According to the Indian Central Pollution Control Board, Ministry of Environment and Forest, National Ambient Air Quality Status 2009, particulate matter is measured gravimetrically with GFA/EPM 2000 filter paper using a respirable dust sampler. Out of 12 stations, 7 stations had $PM_{2.5}$ data. From this data, the highest and lowest areas of exposure were selected based on the National Ambient Air Quality Standard, Government of India. As per Indian governmental standards, a $PM_{2.5}$ annual average of 40 μg/m³ was permissible.

Among the many areas for which accurate pollution data was available, $PM_{2.5}$ exposed areas were stratified and two areas were selected according to the limits of $PM_{2.5}$ defined by the National Ambient Air Quality Standard of the government of India. Mapping of the study areas was done prior to data collection. Two areas were selected, Manali in north Chennai, which reported a $PM_{2.5}$ exposure level above 40 μg/m³ (study group), and Adyar in south Chennai which reported a $PM_{2.5}$ exposure level below 40 μg/m³ (comparison group). Adyar (Rukmini Nagar) the LEA, has an annual average $PM_{2.5}$ of 27.22 μgm/m³ and is a residential area located 2 km away from the main road. Manali, the HEA, has an annual average of 74.22 μgm/m³ and is an industrial area, with a petrochemical industry located within 2 km.

The sample size was derived using Equation 1:

Equation 1

$$n = Z \times p \times q/d^2$$

where Z represents the value of Z alpha, the type 1 error (P<0.05), and corresponding value of Z is 1.96. Since there are no previous studies on this topic, we considered the prevalence to be 50%, with a 5% precision. The calculated sample size was 384. A total of 154 households from the study area and 115 households from the control area were randomly selected to reach the sample size of 410 (143 males, 267 females). Every third house in the

Characteristics	High exposure area n= 201 (%)	Low exposure area n= 209 (%)
Age (years)*	44 ± 11	43 ± 13
Gender		
Male	67 (33.3)	76 (36.4)
Female	134 (66.7)	133 (63.6)
Education		
College and above	8 (7.9)	31(14.8)
Any formal schooling	124 (61.7)	125 (59.8)
No formal schooling	69 (34.3)	53 (25.3)
Occupation		
Skilled	40 (19.9)	55 (26.3)
Unskilled	74 (36.8)	58 (27.7)
Unemployed	87 (43.2)	96 (45.9)
Monthly household income (INR)**	10000 (6000)	13000 (9000)

Values are n (%); * values are mean ± SD; ** values are median (inter - quartile range)

Table 1 — Socio-Demographic Characteristics of the Study Groups

Characteristics	High exposed area n=201	Less exposed area n=209
Family history of DM	43 (21.4)	48 (23)
Behavioral	57 (28.4)	50 (23.9)
Risk factors		
Physical activity	11 (5.5)	29 (13.9)
Body mass index		
Normal	63 (31.3)	73 (34.9)
Overweight	40 (20.0)	48 (23.0)
Obese	98 (48.7)	88 (42.1)
Abnormal waist circumference		
Male	48 (23.9)	15 (7.2)
Female	132 (65.7)	143 (68.4)

Values are n (%).

Abbreviation: DM; diabetes mellitus.

Table 2 — Health Characteristics of the Study Groups

street or vertical building was included in the present study. After obtaining written consent, adults older than 18 years of age were screened for diabetes with random capillary blood glucose (RCBG) measurement. Individuals with self-reported diabetes were also tested for RCBG using a glucometer (One Touch Ultra). Two criteria for diagnosis of diabetes were utilized. The first was based on the RCBG cut off value: greater than or equal to 140 mg/dl (7.8 mmol/l). This RCBG value has shown the same sensitivity and specificity as venous blood to discriminate pre-diabetics with the impaired glucose tolerance test.[29] The second criteria were a RCBG >200 mg/dl (11.1 mmol/l) and reporting any one of the classic symptoms or weight loss; these cases were diagnosed as positive cases of diabetes mellitus.[30]

Blood pressure was measured using an OMRON automatic blood pressure monitor (HEM-7111). Hypertension was defined per the American Heart Association 2017 guidelines: systolic blood pressure greater than or equal to 140 mm mercury (Hg) and/or diastolic blood pressure more than or equal to 90 mm Hg and/or those who have reported a previous diagnosis of hypertension.[31] Investigators gathered data in a pre-tested structured interview schedule. Data on socio-demographics, personal habits, dietary pattern, physical activity, anthropometric measurements, blood pressure, years of living in the current place, and daily exposure to outdoor air were collected. The questionnaire can be found in Supplemental Material. Outdoor air exposures were collected three times a day. The short-term exposure limit was considered to be 15 minutes and the long-term exposure limit was considered to be more than 8 hours. Written informed consent was obtained from each participant. The institutional ethics committee Ethics Committee of the Prof. M Viswanathan Diabetes Research Centre, Chennai, India approved the study.

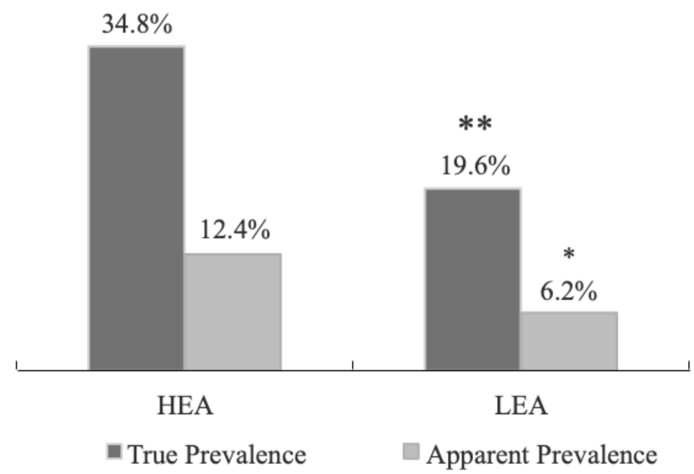
Statistical analysis

Statistical analysis was done using SPSS software version 20. Outdoor air exposures were divided into tertiles and prevalence of diabetes was reported in the HEA and LEA. Prevalence was expressed in percentages. Chi square test and independent t test were used to test statistical significance. Multivariable logistic regression analysis was performed to examine the association between dependent and independent variables. The variables of age, gender, area of living, family history of diabetes, behavioral risk habits, physical activity, body mass index, and waist circumference were included as the independent variables and random blood glucose was the dependent variable. A p value of less than 0.05 was considered to be statistically significant. The odds ratio and confidence interval at 95% were considered to examine the strength of the association between dependent and independent variables. Multi-collinearity was not considered in the study and may be a limitation.

Area of living	Elevated blood glucose (mg/dl)		
	<140	140-199	≥200
High exposed area (n=201)	138 (68.7)	29 (14.4)	34 (16.9)
Less exposed area (n=209)	173 (82.8)	21 (10.0)	15* (7.2)

Values are n (%); * values are p = 0.001 (significant) compared with high exposed area.

Table 3 — Distribution of Random Capillary Blood Glucose Across Study Areas



* p = 0.055 compared with LEA
** p= 0.002 compared with LEA
p ≤ 0.05 is considered to be significant

Figure 1— True and Apparent Prevalence of Diabetes

Results

Table 1 shows the socio-demographic characteristics of the groups. Both groups had similar socio-demographic characteristics.

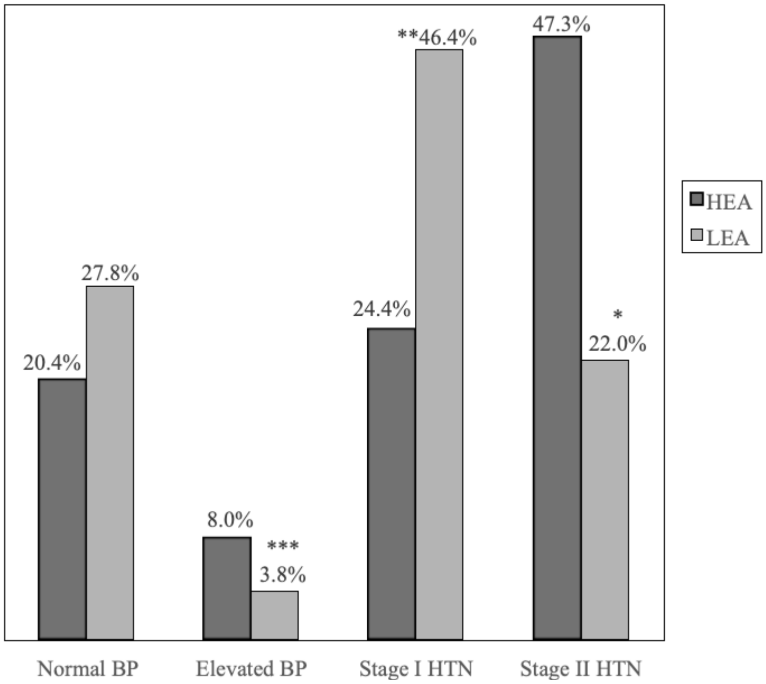
Table 2 shows the health characteristics of the study groups. The participants in the LEA were found to have a higher proportion (23%) of family history of diabetes compared to the HEA (21.4%), but this difference was not statistically significant (p = 0.702). Behavioral risk factors were also similar in both groups (p = 0.307). Subjects living in the HEA reported more physical inactivity compared to the LEA (p = 0.004). Body mass index was similar in both groups (p = 0.397). Abdominal obesity was higher among males in the HEA compared to the LEA (p <0.0001), but females did not show significant differences in abdominal obesity (p = 0.600).

Table 3 shows the distribution of RCBG in the HEA and LEA. There was a greater proportion of subjects with RCBG greater than 200 mg/dl among those living in the HEA compared to those living in the LEA (p = 0.001).

Figure 1 shows the true and apparent prevalence of diabetes in the groups. The proportion of total cases (true prevalence) reported during the study period was higher in the HEA than the LEA (p= 0.002) and the proportion of cases that were positive (apparent prevalence) during the study period was not statistically significant in either area (p = 0.055).

Figure 2 shows the prevalence of hypertension in the groups. The prevalence of stage 2 hypertension was also higher among the HEA than the LEA (p <0.001). Pre hypertension or elevated hypertension was also found to be higher among people living in the HEA than the LEA (p= 0.026)

Table 4 shows the prevalence of diabetes based on everyday exposure to outdoor air. The tertiles of everyday exposure to outdoor air (in hours) were calculated and the prevalence of diabetes was reported in the HEA and LEA. The prevalence of diabetes was higher among those who spent more time (>8 hours) outdoors in the HEA (40.6%) than individuals who spent more time outdoors in the LEA (6.5%), and this difference was statistically significant (p = 0.004). Similarly, the prevalence of diabetes was higher among people with medium exposure (2.1 hours to 8 hours) to everyday outdoor air in the HEA (26.9%) than the LEA (18.1%), but this was not statistically significant (p = 0.24). Subjects who were within the short-term exposure limit (≤ 2 hours) in the



* p = <0.001 LEA compared with HEA (Stage II HTN)
** p = 0.210 LEA compared with HEA (Stage I HTN)
*** p = 0.026 LEA compared with HEA (Elevated HTN)
p ≤ 0.05 is considered to be significant

Figure 2— Prevalence of Hypertension

Exposure to outdoor air	HEA (n=201)	Prevalence of diabetes	LEA (n=209)	Prevalence of diabetes	P-value
Short term exposure limit (≤ 2 hours)	63	14 (22.2)	101	20 (19.8)	0.7099
Medium exposure (2.1 – 8.0 hours)	52	14 (26.9)	77	14 (18.1)	0.2374
Long term exposure limit (≥ 8.1 hours)	86	35 (40.6)	31	2(6.5)	0.0043
Mean exposure (hours)*	7.2±5.9		4.2 ± 3.8		<0.0001

Values are n (%); * values are mean ± SD.

Table 4 — Prevalence of Diabetes Based on Everyday Exposure to Outdoor Air

HEA also showed higher prevalence of diabetes (22.2%) compared to subjects in the LEA (19.8%) (p = 0.71). Subjects in the HEA spent twice as much time (7.2±5.9 hours) in the outdoors compared to subjects in the LEA (4.2±3.8 hours), and this difference was statistically significant (p <0.0001).

Table 5 presents the results of the multivariable logistic regression analysis. Age, gender, residence in the HEA, and positive family history of diabetes were significantly associated with the prevalence of diabetes. A positive family history of diabetes was found to be significantly associated with the prevalence of diabetes (OR = 3.43, CI = 1.86-6.31, p <0.0001), as well as residence in the HEA (OR = 2.60, CI = 1.53-4.43, p <0.0001).

Discussion

This is the first study examining the association of $PM_{2.5}$ and the prevalence of diabetes in India. The findings suggest that the prevalence of diabetes was higher among people living in areas more highly exposed to $PM_{2.5}$ compared to those living in areas with lower exposures to $PM_{2.5}$. The present study showed a strong association between exposure to $PM_{2.5}$ and elevated random blood glucose level.[32] Physical inactivity was one of the biggest identified risk factors. Other risk factors such as family history of diabetes, body mass index, and waist circumference were similar in both groups. Another study from the United States reported that the prevalence of diabetes increased with increasing concentration of $PM_{2.5}$, an increase of 1% prevalence of diabetes with an increase in 10 µg/m^3 $PM_{2.5}$ exposure.[20] Weinmayr *et al.* followed subjects without diabetes in the general German population for three years and found the incidence of diabetes was 9.1%,[33] whereas in the current study the incidence of newly diagnosed cases among the high exposure group was 31.3%.

In the Study on the Influence of Air Pollution on Lung Function, Inflammation and Aging (SALIA) cohort, women older than 54 years followed for 16 years had a 10.5% incidence of diabetes. The current study reported a higher incidence rate of 14.9% of diabetes among women in the high exposure area than the SALIA cohort.[34] The current study reported that the prevalence of hypertension was also higher among people living in the HEA, almost double that of those in the LEA. A study by Lin *et al*. reported that long term exposure to $PM_{2.5}$ is associated with increased risk of developing hypertension and elevated systolic and diastolic pressures among adults older than 50 years.[35] The reported prevalence in that study was 61.4%. The present study reported a 46.3% prevalence of high blood pressure in the HEA. The present study showed a lower prevalence compared to the prevalence rate found by Lin *et al*., which may be due to age differences in the studied populations. A recent study by Bowe *et al*. suggested that a reduction in pollutants and exposures will yield large health benefits globally.[36] Bowe *et al*. estimated 206,105 deaths from diabetes attributable to PM exposure for the year 2016. There were about 3.2 million incident cases of diabetes and about 8.2 million years of healthy lives lost due to diabetes attributable to elevated concentrations of particulate matter.[36]

Significant variables	Odds ratio	(95% CI)	p value
Age (years)	1.07	(1.05-1.10)	<0.0001
Gender	1.97	(1.07-3.66)	0.03
Residence in HEA	2.60	(1.53-4.43)	<0.0001
Positive family history of DM	3.43	(1.86-6.31)	<0.0001
Behavioral risk factors	1.42	(0.79-2.57)	0.244
Dietary habits	1.31	(0.20-8.60)	0.778
Physical inactivity	2.03	(0.85-4.85)	0.110
BMI (kg/m^2)	1.55	(0.82-2.90)	0.177
Waist circumference (cm)	1.40	(0.66-2.94)	0.373

Abbreviations: DM; diabetes mellitus; BMI, body mass index.

Table 5 — Results of Multivariable Logistic Regression Analysis Dependent Variable Random Blood Glucose <140 vs ≥140 mg/dl

Limitations

The design of the present study did not allow us to differentiate between type 1 and type 2 diabetes. The cross-sectional nature of the study was not able to establish whether exposure to air pollution is a causative factor for diabetes. Additionally, due to logistical difficulties such as lack of quality-controlled laboratories and poor compliance to venous blood collection, the researchers used RCBG as a proxy measure. Several studies have compared RCBG measurements with venous plasma glucose measurements in screening for diabetes and prediabetes and have reported RCBG to be an appropriate alternative for screening in epidemiological studies in which collecting venous samples might be challenging. Monitoring of individual exposures to particulate matter would have yielded more relevant results and hence the use of indirect measurement (air quality monitor in the concerned areas) is another limitation. Socio-economic status and body mass index, and male/female participant ratio might also confound the results.

Conclusions

The results of the present study point toward the possibility that exposure to air pollution may be a new and therefore unrecognized contributing factor to the development of diabetes. In conclusion, the study highlights that exposure to $PM_{2.5}$ is positively associated with a higher prevalence of diabetes. Further prospective studies on populations with high exposures to pollution are required to establish whether this association has a causative link.

One important limitation of the present study is its low sample size, and further studies should include at least two areas in different pollution levels. It would also be beneficial to select areas with a gradient of pollution levels in order to demonstrate a trend in the prevalence of diabetes. From a public health perspective, cities in India are plagued by high pollution levels, and these results are important and provide the basis for a more comprehensive study with improved study design.

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