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Quantification of increase in blood pressure due to urban air pollution with reduction in peak expiratory flow rate of lungs

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Abstract: *Urban air pollution is one of the main causes of respiratory problems and selected cardiovascular diseases. Due to damage to respiratory system various pulmonary function values are reduced. Persons with lower pulmonary functions have a higher risk for developing cardiovascular diseases including hypertension. We took a study to evaluate association and develop a relationship between increase in blood pressure due to air pollution and reduction in lung capacity in terms of reduction in Peak expiratory flow rate of lungs. Five locations at Jodhpur were selected for taking observations for various pollutants including PM_{10}, SO_2, NO_2 and CO to evaluate AQI. Study was undertaken on exposed population with exposure to air pollution for 1-5 years. Persons with the same socio-economic background but not exposed to urban air pollution were selected as control population for comparison. Data related to cardiovascular risk including age, body mass index, weight and height, systolic and diastolic blood pressure was measured. Reduction in Peak expiratory flow rate of lungs was measured and index was developed to find out the extent of damage in terms of percentage of volume reduction in fraction (IPEFR). Blood pressure was measured with the subject seated and percentage increase in systolic blood pressure (ISBP) and percentage increase in diastolic blood pressure (IDBP) was calculated. Regression analysis with the help of SPSS was done to find out the statistical parameters. It is clear from the statistical analysis that independent variable IPEFR accounts for the variation in ISBP up to 83.9% and remaining 16.1% is due to other reasons. It also indicates that independent variable IPEFR accounts for the variation in IDBP up to 79.2% and remaining variation is due to other reasons. Estimated equations for showing relationship of ISBP/IDBP with IPEFR were developed by multiple regression analysis. It can be said from the analysis of data that reduction in peak expiratory flow rate of lungs is related to the increase in blood pressure.*

Key Words*: AQI, DBP, PEFR SBP, PM_{10}*

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I. Introduction

Exposure to ambient air pollution has been linked to various health effects including impaired cardiopulmonary function, respiratory and cardiovascular diseases, cancers, and all-cause mortality[4,10,18,21]. Air – borne particulate matter(PM) is a complex mixture of solid and liquid particles of various sizes and compositions ,including polycyclic aromatic hydro carbons(PAH),elemental carbon ,organic carbon compounds ,transition metals and reactive components[19] .These particles are able to penetrate deeply into the respiratory tract and therefore constitute a risk to health by increasing mortality from respiratory infections and diseases like lung cancer and selected cardiovascular diseases[5]. High respiratory vulnerability has been widely acknowledged as a major component of adverse effects of air pollution[7].Several studies have shown an association between lung function decline and long term exposure to air pollution in adults[2,9,15].Lately air pollution induced cardiovascular toxicity has become the focus of intensive studies among cardiologists and specialists in environmental medicine[5,6]. As the chemical composition of ambient particles varies greatly between different geographical areas, it is difficult to identify specific components that elicit cardiovascular toxicity. Air pollution exposure results in significant changes in many cardiovascular indexes. Some of the effects i.e. changes in heart rate, blood pressure, develop acutely in response to increased level of ambient particles[5]. Choi, Xu, Park in their study showed the association between ambient air pollutant concentrations and blood pressure[14]. Engstrom showed that cardiovascular disease and death associated with hypertension is increased in the presence of reduced lung function[8]. Studies have suggested that air pollution is linked to cardiovascular events, frequent hospitalizations, exacerbation of pre-existing cardiac diseases and cardiac related mortality[10,12]. Studies indicates that blood pressure is also a risk factor for future cardiovascular diseases[11,23]. Lina Mu in their study found that short-term exposure to different air pollution levels has significant effects on respiratory function measured by

peak expiratory flow and breath rate .The effects of different air pollution exposure on heart rate ,blood pressure and other physical examinations were not clear[19].Adienbo O loghaguo Macstephen in their study to determine the impact of solid waste exposure in cardio-Pulmonary parameters of municipal solid waste workers in Nigeria found that there was significant increase in systolic & diastolic blood pressure, pulse rate as well as decrease in peak expiratory flow rate of subjects when compared with control subjects[1]. Manas Ranjan Ray and others in their study during 2007-10 found that chronic exposure to vehicular pollution of Kolkata reduces lung function ,increases blood pressure ,suppresses immunity , & enhances cancer risk in the lungs[17].Banna Ram Panwar & others in their study on sand stone mine workers found that the decrease in PEFR is responsible for increase in blood pressure[3]. Exposure to a high concentration of air pollution has been linked to changes in PEFR, especially among Asthma patients[13,16,22]. All most in all studies either pollutant concentration as variable or exposure duration as a variable was considered and extent of damage were reported as increase of blood pressure or mild/severe damage of respiratory system. Quantification of increase of blood pressure due to urban air pollution is not done in relation to decrease of pulmonary function. In this study efforts have been made to quantify increase in blood pressure in relation with decrease in peak expiratory flow rate of lungs due to urban air pollution in terms of Indian AQI as per Central Pollution Control Board (CPCB) of India[20]. Keeping all this in view, the present cross sectional study is designed to monitor the ambient air quality and correlate it with the respiratory and cardiovascular health status of the general population in Jodhpur. Jodhpur is the second largest city of Rajasthan and is situated in the western part of Rajasthan (India). It represents hot and arid climate. It has high amount of dust load and sand in the ambient atmosphere. Due to arid condition in Jodhpur there are frequent dust storm events in pre monsoon season. This leads to long range transport and spread of airborne suspended particulate matter. The aerosols originating naturally (mineral dust) is also one of the important parameter. According to report by W.H.O in 2014 Jodhpur has the highest concentration of PM_{10} in Rajasthan[24].Also Jodhpur is Rajasthan's most polluted city as per May 2016 report of World Health Organisation[25].

II. Methodology & Observations

Various observations for PM_{10}, SO_2, NO_2 & CO were taken at five locations of Jodhpur. From these concentrations of the pollutants, value of sub indices of respective pollutants was calculated to predict AQI as per CPCB guidelines[20]. The highest value of sub indices for various pollutants for that sampling station is considered as AQI for that particular station. The respiratory parameter considered in this study was peak expiratory flow rate of lungs (PEFR) and it was measured with the help of computerized Spirometer. Subjects having exposure to air pollution for duration of 1-5 years were selected for the study. Also control subjects were selected who were not exposed to air pollution but are of same socio-economic background for comparison purpose. Data related to cardiovascular risk including age, body mass index, weight and height, systolic and diastolic blood pressure was measured. Blood pressure was measured with the subject seated. Cigarette smoking, alcohol consumption, medical history including respiratory and cardiovascular conditions and subject's use of medications were ascertained by questionnaire. As it is very difficult to calculate increase of blood pressure in absolute terms, hence Indices were developed for systolic blood pressure and diastolic blood pressure and are represented as ISBP and IDBP i.e. percentage increase in systolic blood pressure in fraction and percentage increase in diastolic blood pressure in fraction. The PEFR of a person depends upon its age, height and weight, and the reduction in PEFR depends upon extent of air pollution. Therefore, it is not possible to find out the generalised amount of reduction due to pollution. So index was developed to find out the extent of damage in terms of percentage of reduction in fraction (IPEFR). Linear regression analyses were conducted to assess the association between increase in blood pressure and decrease in Peak expiratory flow rate of lungs due to air pollution. For the analysis the multiple logistic regression analysis in SPSS was used. IPEFR, IDBP and ISBP were calculated from the equations given below. Predicted values of PEFR were calculated by ERS-93 equations which were inbuilt in spirometer and are named as $PEFR_P$.

$IPEFR=(PEFR_P - PEFR)/PEFR_P$

$IDBP = (DBP-80)/80$

$ISBP = (SBP -120)/ 120$

Where:

PEFR: Measured value of Peak expiratory flow rate of lungs

$PEFR_P$: Predicted value of Peak expiratory flow rate of lungs

DBP: Measured Diastolic blood pressure

SBP : Measured Systolic blood pressure

Table-1 Mean value of indices for exposure duration 1-5 years

Station NO.	AQI	Exposure Duration in years	IPEFR	ISBP	IDBP
1	85	1	0.0805	0.063	0.021
		2	0.0993	0.065	0.034
		3	0.2095	0.137	0.114
		4	0.2107	0.182	0.169
		5	0.2625	0.227	0.182
2	104	1	0.110	0.072	0.036
		2	0.1788	0.134	0.080
		3	0.2125	0.220	0.131
		4	0.2348	0.226	0.182
		5	0.2648	0.303	0.222
3	125	1	0.1776	0.137	0.058
		2	0.1845	0.175	0.138
		3	0.2487	0.250	0.183
		4	0.2686	0.284	0.197
		5	0.3081	0.296	0.223
4	184	1	0.1791	0.170	0.104
		2	0.1983	0.185	0.151
		3	0.2779	0.291	0.185
		4	0.3158	0.308	0.200
		5	0.3606	0.308	0.231
5	236	1	0.1702	0.209	0.176
		2	0.3054	0.261	0.211
		3	0.3375	0.263	0.215
		4	0.3819	0.308	0.216
		5	0.3919	0.309	0.229
6		Control population	0.0371	0.0468	0.0292

Table-2 AQI values at various sampling stations

Sampling Stations NO.	Parameter				AQI
	SO_2 (µg/m³)	NO_2 (µg/m³)	PM_{10} (µg/m³)	CO (mg/m³)	
1	9.64	29.82	138	0.64	125
2	7.62	34.56	226	1.09	184
3	9.47	38.60	85	0.85	85
4	5.58	19.82	106	0.59	104
5	8.77	31.28	286	0.86	236

Concentration of various pollutants at five locations and corresponding AQI is given in Table-2. Percentage reduction in IPEFR with percentage increase in SBP / DBP in fraction is given in Table-1. It is clear from the observations that if AQI is increasing, reduction in PEFR is increasing and blood pressure is also increasing. Observations also indicate that if AQI is constant but exposure duration is increasing, reduction in PEFR is increasing and blood pressure is also increasing.

III. Analysis

(a) Statistical Analysis: Statistical parameters and coefficients for statistical verification and to establish relationship between IPEFR and IDBP/ ISBP are tabulated in table-3 & table-4. F-test and t-test is also done to check the significance of independent parameter IPEFR on dependent parameter IDBP/ISBP

Table No. 3 Statistical Constants for IDBP

Model Summary					
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	
1	0.895(a)	0.800	0.792	0.03025	
ANOVA					
	Sum of Squares	Df	Mean Square	F	Sig.
Regression	0.084	1	0.084	92.147	0.000
Residual	0.021	23	0.001		
Total	0.105	24			
Coefficients					
	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta	B	Std. Error
(Constant)	-0.012	0.018		-0.653	0.520
IPEFR	0.702	0.073	0.895	9.599	0.000
Note: Predictors: (Constant), IPEFR Dependent Variable: IDBP Independent Variable: IPEFR					

F-Test:
$H_0 : \beta_1 = \beta_2 = 0$ against H_1 : not all $\beta_k = 0$:(k = 1,2)
Anova table gives the value of calculated 'F' = 92.147
and $F_{n-k-1, \alpha} = 4.2793$(From standard tables)

Hence $F_{CAL} > F_k, F_{n-k-1, \alpha}$

Hence, reject H_0 at α=0.05 level of significance , therefore significance of individual β's be tested by 't' test

t-Test:
$H_0 : \beta_J = 0$ against $H_1 : \beta_J \neq 0$ (for j=1)
The calculated value for't' statistics for β_1 :
t (for β_1) = 9.599
The value of $t_{n-k-1;\alpha/2}$=2.069
The value of t is > $t_{n-k-1;\alpha/2}$ therefore reject H_0
Hence $\beta_1 \neq 0$

Thus the estimated multiple regression analysis equation for IDBP can be expressed as:

IDBP = 0.702 IPEFR - 0.012

Table No. 4 Statistical Constants for ISBP

Model Summary					
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	
2	0.920	0.845	0.839	0.03238	
ANOVA					
	Sum of Squares	Df	Mean Square	F	Sig.
Regression	0.132	1	0.132	125.853	0.000
Residual	0.024	23	0.001		
Total	0.156	24			
Coefficients					
	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta	B	Std. Error
(Constant)	0.006	0.020		0.280	0.782
IPEFR	0.878	0.078	0.920	11.218	0.000
Note: Predictors: (Constant), IPEFR Dependent Variable: ISBP Independent Variable: IPEFR					

F-Test:
$H_1 : \beta_1$ against H_1 : not all $\beta_k = 0$:(k = 1)
Anova table gives the value of calculated 'F' = 125.853
and $F_{k, n-k-1, \alpha} = 4,2793$ (From standard tables)

Hence $F_{CAL} > F_k, F_{n-k-1, \alpha}$

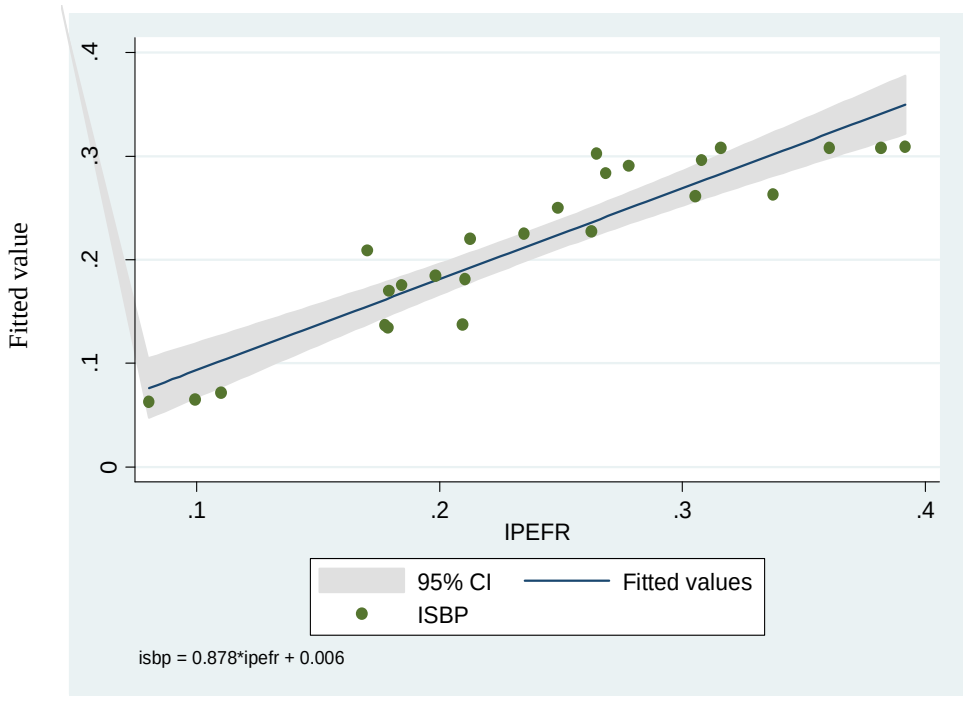
Hence, reject H_0 at α=0.05 level of significance , therefore significance of individual β's be tested by 't' test

t-Test:
$H_0 : \beta_J = 0$ against $H_1 : \beta_J \neq 0$ (for j=1)
The calculated value for't' statistics for β_1
t (for β_1) = 11.218

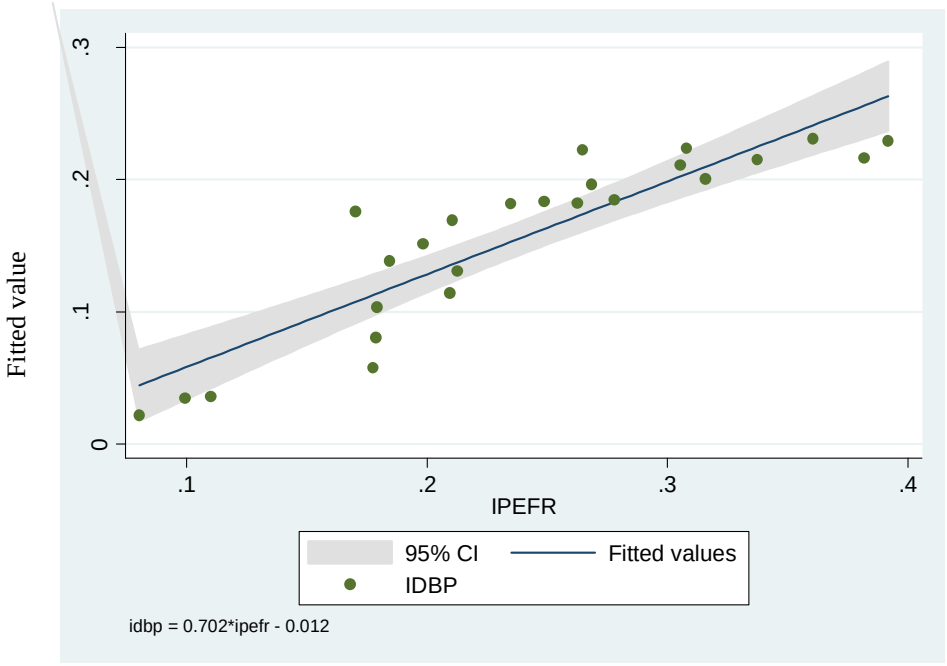
The value of $t_{n-k-1;\alpha/2}$=2.069
The value of t is > $t_{n-k-1;\alpha/2}$ therefore reject H_0
Hence $\beta_1 \neq 0$
Thus the estimated multiple regression analysis equation for ISBP can be expressed as:

ISBP = 0.878 IPEFR + 0.006

(b) Graphical Analysis: Statistical analysis and trends of calculated values of IPEFR, IDBP and ISBP indicates that best fit curve is linear one. Graph-1 represents observed values and fitted values in estimated relationship between ISBP and IPEFR and shows that both values are lies within close limits. Similarly Graph-2 represents observed values and fitted values in estimated relationship between IDBP and IPEFR and shows that both values are lies within close limits.



Graph-1



Graph-2

Graphical and statistical analysis clearly indicates that reduction in IPEFR is associated with increase in SBP/DBP. Line of best fit clearly shows relationship between IPEFR and IDBP/ISBP. Dots are scattered plot between actual values of IDBP/ISBP and IPEFR to evaluate the departure of regressed value. Most of the actual values of IDBP/ISBP are in close range of fitted values as most of the dots are in the shaded regions which is the 95% confidence level

IV. Conclusion

It can be established from the findings of the study that increase in SBP and DBP due to urban air pollution is closely associated with reduction in PEFR and persons having lower pulmonary functions have a higher risk for developing cardiovascular diseases including hypertension. It is clear from the statistical analysis that independent variable IPEFR accounts for the variation in ISBP up to 83.9% and remaining 16.1% is due to other reasons. It also indicates that independent variable account IPEFR accounts for the variation in IDBP up to 79.2% and remaining variation 20.8% is due to other reasons. Further estimated equations for showing relationship of ISBP/IDBP with IPEFR were developed. Hence it is clear that long exposure duration in urban pollution is not only affecting the respiratory tract but also increasing the blood pressure.

References

[1]. Adienbo, Njoku and Asara, "An Assessment of some Cardiovascular and Lung Function Parameters of Municipal Solid Waste Workers in Port Harcourt, South-South Nigeria". Greener Journal of Human Physiology and Anatomy. Vol. 1 (1),pp. 001-006, October 2013.

[2]. Andersen ZJ, Hvidberg M, Jensen SS, Ketzel M, Loft S, Sorensen M, et al. Chronic obstructive pulmonary disease and long-term exposure to traffic-related air pollution: a cohort study. Am J Respir Crit Care Med. 2011; 183: 455-461

[3]. Banna Ram Panwar , Anil Vyas , Suresh Kumar Singh, "Effect of Reduction in Peak Expiratory Flow Rate on Blood Pressure on Sand Stone Mine Workers". Journal of Geoscience and Environment Protection, 2016, 4, 14-19

[4]. Boffetta P. Human cancer from environmental pollutants: the epidemiological evidence. Mutat Res. 2006; 608:157-162. [PubMed:16843042]

[5]. Boris Z. Simkhovich, Michael T. Kleinman, Robert A. Kloner, "Air Pollution and Cardiovascular Injury" Journal of the American College of Cardiology, Vol.52, No.9, 2008;719-726

[6]. Brook RD . Cardiovascular effects of air pollution . clinical science . 2008; 115:175-187.[PubMed:18691154]

[7]. Dockery DW, Pope CA III. "Acute respiratory effects of particulate air pollution" Annual Rev Public Health 1994;15:107-32.

[8]. Engstrom, G., Headblad, B., Valind, S., and Janzon, L.(2001).Increased incidence of myrocardial infraction and stroke in hypertensive men with reduced lung function. Journal of Hypertension, 19, 295-301

[9]. Forbes LJ, Kapetanakis V, Rudnicka AR, Cook DG, Bush T, Stedman JR, et al. Chronic exposure to outdoor air pollution and lung function in adults. Thorax.2009; 64: 657-663.

[10]. Franchini M, Mannucci PM. Short- term effects of air pollution on Cardiovascular diseases : outcomes and mechanisms . J Thromb Haemost. 2007;5:2169-2174. [PubMed:17958737]

[11]. Hans Orru, Rain Jogi, Marko Kaasik "Chronic Traffic-Induced PM Exposure and Self-Reported Respiratory and Cardiovascular Health in the RHINE Tartu Cohort" Int. J. Environ, Res. Public Health 2009, 6, 2740-2751

[12]. Hoek G, Brunekreef B, Fischer P, van Wijnen J. The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other Cardiovascular causes of death in a time series study. Epidemiology. 2001:12:355-357. [PubMed:11337606]

[13]. Hong YC, Pan XC, Kim SY, Park K, Park EJ, et al. Asian Dust Storm and pulmonary function of school children in Seoul. The Science of the total environment. 2010;408:754-759.[PubMed:19939437]

[14]. Ji-Ho Choi, Qing-Song Xu, So-Yeon Park, Jin-Hee Kim "Seasonal Variation of effect of air pollution on blood pressure" J Epidemical Community Health 2007; 314-318

[15]. Kan H, Heiss G, Rose KM, Whitsel E, Lurmann F, London SJ, et al. Traffic exposure and lung function in adults: the Atherosclerosis Risk in Communities study. Thorax.2007; 62: 873-879.

[16]. Ma L, Shima M, Yoda Y, Yamamoto H, Nakai S, et al. Effects of airborne particulate matter on respiratory morbidity in asthmatic children . J Epidemiol. 2008;18:97-110. [PubMed:18490839]

[17]. Manas Ranjan Ray , Twisha Lahiri, "Air Pollution and its effects on health-Case studies, India"

[18]. Mannino DM, Buist AS. Global burden of COPC: risk factors , prevalence, and future trends. Lancet. 2007;370:765-773.[PubMed:17765526]

[19]. Mu, L., Deng, F., Tian, L., Li, Y., Swanson, M., Ying, J., … Bonner, M. R. (2014). Peak Expiratory Flow, Breath Rate and Blood Pressure in Adults with Changes in Particulate Matter Air Pollution during the Beijing Olympics: A Panel Study. Environmental Research, 133, 4–11. http://doi.org/10.1016/j.envres.2014.05.006

[20]. National Air Quality Index , Central Pollution Control Board(CPCB), Ministry of Environment and Forest & Climate change Publication.

[21]. Pervin T, Gerdtham UG, Lyttkens CH. Societal costs of air pollution-related health hazards: A review of method and results . Cost effectiveness and resource allocation: C/E.2008;6:19.[PubMed:18786247]

[22]. Qian Z, Lin HM, Chinchilli VM, Lehman EB, Stewart WF,et al. Association between air pollution and peak expiratory flow among patients with persistent asthma. Journal of toxicology and environmental health Part A. 2009;72:39-46.[PubMed:18979353]

[23]. Vasan, R.S., Larson, M.G., Leip, E.P., Evans, J.C., O'Donnell, C.J., Kannel, W.B., and Levy, D. Impact of High-Normal Blood Pressure on the Risk of Cardiovascular Disease. The New England Journal of Medicine, 345(18), 1291-1297, 2001

[24]. WHO Global Urban Ambient Air Pollution Database,2014

[25]. WHO Global Urban Ambient Air Pollution Database,2016

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