Evaluation of the effects of air pollution on serum inflammatory markers in young adults

Hina Riaz[1,2], Binafsha Manzoor Syed[2], Zulfiqar Laghari[3]

ABSTRACT

Objective: To evaluate the effects of exposure of air pollution (traffic pollutants) on markers of inflammation in apparently healthy vehicles drivers (high exposure) and compare it with (less exposed) students and to evaluate correlation with the duration of exposure of traffic pollution.

Study Design: cross sectional (Descriptive) study.

Place and Duration: This study conducted in Physiology department of Liaquat University of Medical and Health Sciences (LUMHS) from 3rd February to 5th December 2017.

Methodology: This study evaluated the effects of traffic pollution in markers of inflammation markers including C-reactive protein (CRP), Leukocytes count, interleukin-6 (IL-6), tumor necrotic factor-α (TNF-α) and tumor necrotic factor-β (TNF-β) of apparently healthy volunteers. One hundred seventy-four (174) non-smoking, apparently healthy automobile vehicle drivers (high exposed group, n=87) and the same number of hostel resident students (less exposed group, n=87) were recruited for this study, their serum inflammatory markers were compared. Further automobile vehicle drivers categorized into five groups according to their duration of exposure by using 5-year cut-off.

Results: Levels of inflammatory markers including IL-6 ($p < 0.001$), TNF-β ($p < 0.021$), Leukocytes ($p < 0.03$), Neutrophils ($p < 0.001$), Eosinophils ($p < 0.001$), Basophils ($p < 0.001$) and CRP ($p < 0.001$), were significantly higher among drivers group than student's groups. TNF-α, TNF-β, IL-6, CRP, neutrophils, lymphocytes, eosinophils, basophils and monocytes were positively correlated with duration of driving exposure. While total leukocytes did not show any significant correlation.

Conclusion: Exposure to traffic pollutants causes rise in the circulatory markers of inflammation in healthy volunteers.

Keywords: Traffic pollutants, Markers of inflammation, Leukocytes, Non-smokers, Healthy students, Healthy drivers.

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INTRODUCTION

Several studies reported that particulates, especially particulate matter$_{2.5}$ ($PM_{2.5}$), is the most common cause of mortality. As revealed by world health organization (WHO) air quality database Pakistan was ranked among the most polluted countries for having the elevated concentration of $PM_{2.5}$, while Karachi ranked 2nd for the levels of PM_{10}[1]. It is confirmed by numerous studies that in Pakistan road transportation and rise in urban population abolishing environmental quality and cause the production of pollutants like Sulphur Dioxide (SO_2) in the atmosphere[2]. Giannadaki (2016), conducted study to evaluate the relation of particulate matter ($PM_{2.5}$) with early demises; results reported 105 thousand/ year early demises in Pakistan, 1.33 million/ year in China and 575 thousand/ year in India[3]. Population residing in less privileged areas of the world experience the burden of ambient air contamination out of proportion with an 88% of the 370,0000 early demises happening in low and middle income parts of the world. The highest affliction has been observed in the WHO defined South-East Asia and Western Pacific areas. According to the current estimates, air pollution observed to play a significant role in early demises & cardiovascular ailments putting a lot more burden than was formerly imagined by scientists[4]. The particles with the greatest health implications are ones with a diameter of ten microns or lesser, (≤ PM10), which can enter and stay

1. Lecturer of Physiology, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan.
2. Director Medical Research Centre, Director Clinical Research Division, Director ORIC, LUMHS, Jamshoro, Pakistan.
3. Professor of Physiology Department, University of Sindh. Jamshoro, Pakistan.

Correspondence:
Dr. Hina Riaz
Lecturer of Physiology, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan.
Email: hina_riaz81@yahoo.com

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inside the depths of the lungs. Long term contact with such particles adds to the danger of contracting respiratory and cardiovascular illnesses, and the cancer of the lungs[5,6].

The available literature suggests that the exposure to the automobile fume produces deleterious effects on health. In addition, long-term exposure also provokes inflammatory response. However, the effect of the duration of exposure of pollutants and level of exposure not answered yet. Thus, this study is a preliminary study in Pakistan; aimed to evaluate the effects of exposure of traffic exhaust and its effects on serum inflammatory markers among high exposure group and the low exposure group. Give that the concept that long the duration causes more detrimental effects, this study also aimed to look at the duration of exposure with the rise in the markers of inflammation. Thus, this study hypothesized that daily exposure of automobile exhaust fume causes rise in systemic inflammatory markers of apparently healthy volunteers. This stud aimed to assess the effects of exposure traffic pollutants on markers of inflammation in apparently healthy vehicles drivers and compare it with less exposed students and to evaluate correlation with the duration of exposure of traffic pollution.

METHODOLOGY

This study evaluated the markers of inflammation in high automobile pollution exposed group and less exposed group of apparently healthy volunteers. One hundred seventy-four (174), non-smoking, apparently healthy (not suffering from any systemic diseases) volunteers included, while persons having BMI ≥ 30, suffering from any systemic or autoimmune disorders excluded from study. Exposed group consist of eighty-seven (87), apparently healthy, non-smoking automobile vehicle drivers (must be having daily contact of traffic exhaust for at least six hours), aged between 18-40 years. Based on duration of exposure this group was divided into five categories: first who have been driving for at least five years (≤ 5), second who have been driving experience for ten years (≥10), third who have been driving experience for fifteen years (≥15), fourth who have been driving experience for twenty years (≥20) and fifth who have been driving experience for twenty-five years (≥25). The sample of the drivers was taken from the areas of heavy traffic intersections of the Jamshoro and Hyderabad city. While less exposed group comprised of hostel resident students (aged 18-25 years) of LUMHS, as hostel are located comparatively less polluted vicinity and surrounded by so many trees. Non-probability purposive sampling techniques was used.

Serum Inflammatory markers: This study evaluated the effects of traffic pollutants on markers of inflammation including C-reactive protein (CRP), Leukocytes count (i.e., neutrophils, lymphocytes, eosinophils, monocytes and basophils) interleukin-6 (IL-6), tumor necrotic factor-α (TNF-α) and tumor necrotic factor-β (TNF-β). CRP analyzed by "C-reactive protein Hitachi 902 turbidimetry "and total leukocytes count by "Automated Analyazer"(sysmex)". While IL-6, TNF-α and TNF-β were analyzed by "human instant ELISA (enzyme linked immunosorbent assay) kits" (Koma Biotech). For analysis of markers, 200 ul of washing solution added to each well. Wells were aspired and excessive liquid removed 100 ul of standard (sample) then incubated at room temperature for 2 hours. Well aspirated and washed. 100 ul of diluted detection antibody (0.4 ug/ml for TNF-α, 0.1ug/ml for TNF-β and for IL-6 0.25ug/ml) to each well covered with the plate sealer then incubated for 2 hours then diluted Color Development Enzyme (1:20 dilute) each well was added. Incubated at room temperature until the appropriate color development at least for 17-27 min, plate read at 450 nm wavelength.

Statistical Analysis: Data analyzed by using IBM statistical program for social sciences (SPSS), (IBM Corp. Released 2012. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp. version) Mann-Whitney-U test applied to determine the difference of markers of inflammation in both groups. Spearman rank correlation used to determine the correlation of duration of driving exposure with inflammatory markers in drivers group. The results of all analyses evaluated for statistical significance using p-value < 0.05 and the 95 % confidence intervals (CI).

RESULTS

During study period, one hundred seventy-four (N=174) apparently healthy subjects included, that consisted of eighty-seven students and the same number of drivers. Base line features of volunteers described in Table-I. Mean age of students was 23 (±2.2) years and drivers were 31(±7.3) years. While mean height and mean weight of students and drivers were 171cm (±4.5), 174cm (±4.8) and 70 kg (±3.6), 75 kg (±6.3) respectively. Mean Body mass index (BMI) of students and drivers were 22.1 (±2.6), 24.3 (±2.4).

Table-I: Level of markers of Inflammation between students and drivers group (N=174)

Markers of inflammation	Students (Median)	Drivers (Median)	p –Value
TNF-α pg/ml	6.7	7.7	0.75
IL-6 pg/ml	27.1	29.0	< 0.001
TNF-β pg/ml	26.9	27.7	0.021
CRP mg/dl	0.28	0.50	< 0.001
Leukocytes	7.1	9.3	0.03
Neutrophils%	48.6	66	< 0.001
Eosinophils %	0.32	5.0	< 0.001
Basophils%	0.03	2.1	< 0.001
Lymphocytes%	24	33	< 0.001
Monocytes %	6.1	10.1	0.33

Comparison of serum markers of inflammation: Mann Whitney-U test, (Table-I) Shows that levels of markers of inflammation such as IL-6 ($p < 0.001$), TNF-β ($p < 0.021$), Leukocytes ($p < 0.027$), Neutrophils ($p < 0.04$), Eosinophils ($p < 0.001$), Basophils ($p < 0.03$) and CRP ($p < 0.001$) were significantly higher among drivers. Whereas TNF-α ($p > 0.75$),

Lymphocytes (p > 0.73) and Monocytes (p > 0.33) did not differ significantly were same and lower among the both groups.

Correlation of serum markers of inflammation with duration of driving: Duration of driving exposure showed positive correlation with almost all markers of inflammation, i.e. TNF-α (r_s = 0.61, p = 0.005), TNF-β (r_s = 0.39, p = 0.001), CRP (r_s = 0.20, p = 0.04), neutrophils (r_s = 0.31, p-value = 0.04), lymphocytes (r_s = 0.28, p-value = 0.007), eosinophils (r_s = 0.76, p-value = 0.001), monocytes (r_s = 0.19, p-value = 0.07), basophils (r_s = 0.21, p-value = 0.06). While no association found with total leukocytes count (r_s = 0.09, p-value = 0.36), as shown in Table-II.

Table-II: Spearman Correlation among markers of inflammation and driving duration of traffic pollutant (N= 87)

Inflammatory markers	Duration of traffic exposure	
	r_s	p –Value
TNF-α pg/ml	0.61	0.005
IL-6 pg/ml	0.13	0.23
TNF-β pg/ml	0.39	0.001
CRP mg/dl	0.20	0.04
Leukocytes	0.09	0.36
Neutrophils%	0.31	0.04
Eosinophils %	0.76	0.002
Basophils%	0.21	0.06
Lymphocytes%	0.28	0.007
Monocytes %	0.19	0.78

DISCUSSION

In regard of effects of traffic pollution on human health susceptible population (suffering from systemic diseases) always have been a ground of interest for researchers thus, limited literature is available concerning effects of pollution on health status apparently healthy population. Our study results showed positive association with prolong duration of driving exposure; altered level of markers of inflammation at subclinical level are worrisome indication of health issues in future. In our study monocytes are positively correlated with duration of driving consistent findings are reported by another study[7]. Our study findings are similar with findings of Lee et.al, who reported that long term exposure of pollutants causes alteration in leukocytes count and fibrinogen[8].

According to results of our study, markers of inflammation were raised in exposed group, Similarly, Karoly reported rise in proinflammatory markers and amplification of IL-6 gene in pulmonary vessels due to exposure of PM[9]. consistent findings observed by Hajat A, who reported indication of relationship between pollutant exposure and IL-6[10]. Likewise, prolong exposure of traffic pollutants and rise in IL-6, found in former study[11]. Similarly, black carbon exposure causes increase in serum concentration of cytokines like IL-6, IL-8 and TNF-α and potentiate the phagocytic ability of monocytes [12]. However, another study suggested no relation of neutrophils and allergic cells (eosinophils, basophils) with traffic exhaust[13].

In addition, few studies have shown particulate issues of instance exposure and have positively mentioned an association with CRP levels[14]. Even though exposure intensity of pollutants is over all reduced with time duration, over long periods (10 years and longer) have been stayed stable[15].

On contrary, traffic pollutant with markers of inflammation (leukocytes, CRP, TNF-α) and red blood cells showed inconsistent relationship in another study, while dissimilar findings observed in our study[16]. Studies also reported prolong exposure of traffic pollutants is associated with rise in CRP concentration and cardiovascular disorders and exacerbation if inflammatory cascade[17,18]. Similarly, traffic generated NO_x and CO showed positive association with TNF-α, IL-6 and CRP [19,20].

CONCLUSION

The findings of our study suggest that prolong exposure of traffic pollutants causes increase in the distribution of inflammatory blood cells in apparently healthy subjects.

Recommendation: In such an alarming situation when the automobile pollution is at rise, there is dire need to conduct large scale research projects to look into details long term effects of the automobile pollution. Such a data will help to constitute policies regarding health status of peoples who have been exposed to traffic exhaust fumes on daily basis.

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AUTHOR'S CONTRIBUTION

Riaz H: Conceived idea, Designed research methodology, Statistical analysis, Manuscript writing, Data collection.
Syed BM: Manuscript final reading and approval.
Laghari Z: Manuscript final reading and approval.

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REFERENCES

1. Norbert B. Contribution of air pollution to COPD and small airway dysfunction. Respirology. 2016;21(2):244. DOI.org/10.1111/resp.12644.
2. Baloch MA. Dynamic linkages between road transport energy consumption, economic growth, and environmental quality: evidence from Pakistan. Environ Sci Pollut R. 2018;25(8):7552.
3. Giannadaki D, Lelieveld J, Pozzer A. Implementing the US air quality standard for PM2.5 worldwide can prevent

millions of premature deaths per year. Enviro Health 2016, 23;15(1):88.
4. World Health Organization. An estimated 12.6 million deaths each year are attributable to unhealthy environments. 2016. Website: [www.who.int] Retrieved on 4th June 2016.
5. Cohen AJ, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. Lancet. 2017;389(10082):1918. DOI: org/10.1016/S0140-6736(17)30505-6
6. World Health Organization. Ambient (outdoor) air quality and health. 2018. Website: [www.who.int] Retrieved on 2nd August 2018
7. Su TC Hwang JJ Yang YR Chan CC. Association between Long-term Exposure to Traffic-related Air Pollution and Inflammatory and Thrombotic Markers in Middle-aged Adults. Epidemiol. 2017; 28: 81. DOI: 10.1097/EDE.0000000000000715.
8. Lee H W Myung. "Short-and long-term exposure to ambient air pollution and circulating biomarkers of inflammation in non-smokers: A hospital-based cohort study in South Korea." Environ Int. 2018. 119: 273. DOI: org/10.1016/j.envint.2018.06.041.
9. Karoly ED, Li Z, Dailey LA, Hyseni X, Huang YCT. Upregulation of tissue factor in human pulmonary artery endothelial cells after ultrafine particle exposure. Environ. Health Perspec. 2007;115(512234):540.
DOI: org/10.1289/ehp.9556.
10. Hajat A, Allison M, DAV, Jenny NS, Jorgensen NW, Szpiro AA, et al. Long-term Exposure to Air Pollution and Markers of Inflammation, Coagulation, and Endothelial Activation. Epidemiol. 2015;26(3):320
DOI: org/10.1097%2FEDE.0000000000000267.
11. Chuang KJ, Yan YH, Chiu SY, Cheng TJ. Long-term air pollution exposure and risk factors for cardiovascular diseases among the elderly in Taiwan. Occup. Environ. Med. 2011;68(1):68. DOI: org/10.1136/oem.2009.052704.
12. Sahu D, Kannan G, Vijayaraghavan R. Carbon black particle exhibits size dependent toxicity in human monocytes. Int J Inflam. 2014. DOI: org/10.1155/2014/827019.
13. Larsson N, Brown J, Stenfors N, Wilson S, Mudway IS, Pourazar J, et al. Airway inflammatory responses to diesel exhaust in allergic rhinitics. . Inhal. Toxicol. 2013;25(3):167. DOI: org/10.3109/08958378.2013.765932.
14. Wang R, Henderson SB, Sbihi H, Allen RW, Brauer M. Temporal stability of land use regression models for trafficrelated air pollution. Atmos. Environ. 2013;64:319. DOI: abs/10.1002/bdrc.20097 .
15. Gulliver J, DK, Hansell A, Vienneau D. Development and Back-Extrapolation of NO 2 Land Use Regression Models forHistoric Exposure Assessment in Great Britain. Environ. Sci. Technol. 2013;47(14):7811. DOI: org/10.1021/es4008849
16. Cesaroni G, Porta D, Badaloni C, Stafoggia M, Eeftens M, Meliefste K, et al. Nitrogen dioxide levels estimated from land use regression models several years apart and association with mortality in a large cohort study. Environ Health 2012;11(1):64.
17. Tsai D-H, Riediker M, Berchet A, Paccaud F, Waeber G, Vollenweider P, et al. Effects of short-and long-term exposures to particulate matter on inflammatory marker levels in the general population. Environ Sci Pollut R. 2019:8. (19):19704. DOI: 10.1007/s11356-019-05194-y.
18. Pilz V, Wolf K, Breitner S, Rückerl R, Koenig W, Rathmann W, et al. C-reactive protein (CRP) and long-term air pollution with a focus on ultrafine particles. Int. J. Hyg. Environ. Health. 2018;221(3):518. DOI: org/10.1016/j.ijheh.2018.01.016.
19. Wittkopp S, Staimer N, Tjoa T, Gillen D, Daher N, Shafer M, et al. Mitochondrial genetic background modifies the relationship between traffic-related air pollution exposure and systemic biomarkers of inflammation. PloS One. 2013;8(5). DOI: org/10.1371/journal.pone.0064444.
20. Mostafavi N, Vlaanderen J, Hyam MC, Beelen R, Modig L, Palli D, et al. Inflammatory markers in relation to long-term air pollution. Environ Int. 2015;81:7.
DOI: org/10.1016/j.envint.2015.04.003.