# Pathophysiological Effects of Cotton Dust Pollution on Blood Pressure in Textile Workers

SADAF ZIA1, SHIREEN JAVEED2, FOUZIA JAN3, KOMAL ATTA4, SHAISTA HUSSAIN5, SHAMA IQBAL6

# **ABSTRACT**

**Background:** Air pollution is a major occupational hazard in various industries including textile industry. The respiratory diseases are labeled as fourth leading cause of deaths in Asia with prevalence rate 6.3% and this results due to increase in environmental pollutants in last few years. Cotton fiber inhalation in very high concentration results in inflammation of respiratory epithelium along with shortness of breath and bronchospasm. **Aim:** To determine and correlate the harmful effects of cotton dust pollution on serum aldosterone and blood pressure in human beings.

**Method:** This study was conducted in a textile mill of Faisalabad. Eighty four conveniently selected workers participated in the research study who had been working in cotton industry for last 3-10 years. Data collection was done via questionnaire, blood pressure was measurement manually and serum aldosterone levels were estimated via ELISA.

Results: Highly significant difference was noted in systolic blood pressure and serum aldosterone levels in all study groups.

**Conclusion:** Exposure to cotton dust pollution for a prolonged period may damage the pulmonary endothelium which may lower the blood pressure in exposed persons.

Keywords: Cotton dust pollution, Airway hyper responsiveness, Endothelial dysfunction, Occupational Hazards

# INTRODUCTION

Air pollution is one of the majorhazardsin various industries including textileindustry. Due to rapid industrialization specially in textile sectorin the past few years, cotton dust induced lung diseases have become a global health issue1 Workers involved cotton industry, especially manufacturers of yarn, fabric and thread are mostly exposed to cotton dust<sup>2,5</sup> In Asian over half a million deaths per year is due to the diseases resulting from environmental pollution.3 According to a study carried out in India in 2017, the respiratory diseases are the fourth leading cause of deaths in Asia with prevalence rate 6.3% and if these environmental and occupational pollutants will not decrease, than it will become the third leading cause of death by the year 2040 and it is expected that its prevalence will increase to 8.6%4. Cotton fibers while growing in the fields are naturally colonized by different types of toxins producing microorganisms and during cotton processing these endotoxins are released in the atmosphere along with clouds of cotton dust<sup>6</sup>.

In human beings these invisible dust particles enter in lungsalveoli through inhalation in dusty environment and results in bronchospasm, shortness of breath and inflammation of respiratory passages<sup>7</sup>. These Ultra-fine

Correspondence to Dr. Sadaf Zia Email: drsadafzia9@gmail.com Cell: 0300-6617901 particulate matters aggregated on the surface of respiratory endothelium produce pro-inflammatory mediators such as leukotriene B4, interleukin-8 and tumor necrosis factor, theyinduce inflammation and necrosis ofrespiratory epithelial cells. This increases air way reactivity and ultimately leads to dysfunction of respiratory endothelium<sup>8</sup>.

Pulmonary endothelium acts as dynamic interface between flowing blood and vessel wall. It also produces a number of factors which regulate the blood flow. Pulmonary endothelium has numerous physiological, immunological, and metabolic functions<sup>9</sup>. The capillary endothelium is a major site for the conversion of enzyme angiotensin I in to angiotensin II by the pulmonary capillary endothelium bound (PCEB) angiotensin converting enzyme (ACE) through renin angiotensin system<sup>10</sup>. Angiotensin converting enzyme is expressed mainly by pulmonary capillaries therefore any damage to pulmonary endothelial results in shedding ofACE<sup>11</sup>.

Renin angiotensin aldosterone system is the main regulatorof blood pressure in human beings. It controls the release of aldosterone throughangiotensin II secretion by sensing intra vascular volume<sup>12</sup>. Aldosterone causes vasoconstriction of both afferent and efferent arterioles within minutes and regulates blood pressure<sup>13</sup>. In addition to blood pressure, aldosterone also helps in regulatingextracellular fluid volume and vascular tone.<sup>14</sup> The renin angiotensin system is not the only source, however aldosterone can also be synthesized by vascular smooth muscle cells, endothelial cells in brain, and myocardium<sup>15</sup>.

As no work has been done on this aspect of human health up till now. This study was designed to determine, compare and correlateserum levels of aldosterone with blood pressure in persons exposed to cotton dust for various durations.

<sup>&</sup>lt;sup>1</sup>Associate Professor, University Medical and Dental College, Faisalabad

<sup>&</sup>lt;sup>2</sup>Associate Professor, Aziz Fatimah Medical and Dental College, Faisalabad

<sup>&</sup>lt;sup>3</sup>Senior Demonstrator, University Medical and Dental College, Faisalabad

<sup>&</sup>lt;sup>4</sup>Assistant Professor Pathology, University Medical & Dental College, Faisalabad

<sup>&</sup>lt;sup>5</sup>Assistant Professor Physiology, Rahbar Medical & Dental College, Lahore

<sup>&</sup>lt;sup>6</sup>Assistant Professor Physiology, Sh. Zayed Medical College, Rahim Yar Khan

# **METHODOLOGY**

It was a correlational study, conducted in a textile mill of Faisalabad. The Sample size was 85 Subjects (calculated by formula N= [( $Z_{\alpha} + Z_{\beta}$ )/(1+r)/(1-r)]<sup>2</sup>+3,(  $Z_{\alpha}$  = 1.960at  $\alpha$  = 0.05,  $Z_{\beta}$  = 0.842 (power 80%) and r =0.3). <sup>16</sup>

One hundred mill workers were approached through convenient sampling. The selected age group was 18-40 years males.

#### **Control group**

- Male workers (18-40years of age)
- Newly inducted workers (having less than one month exposure)
- Subjects with no acute or chronic illness

#### Study group

- Same age (as control group)
- Males working from last 3 to 10years
- Subjects having no history of any acute or chronic illness

#### **Exclusion criteria**

Subjects with any chronic disease (e.g. hypertension, diabetes mellitus, COPD chronic pulmonary obstructive disease.)

- Subjects having any acute viral or bacterial infection.
- Subjects with history of major trauma/surgery in past six months.
- Smokers

Eighty four selected subjects were divided into three groups (n=26 for control group, n=29 for two study groups) depending on their duration of job in cotton industry.In control group the subjects having less than one month exposure to cotton dust were included. In group II, subjects have 3-5 years exposure and in group III subjects having exposure to cotton dust for 5-10 years were included. History and examination was recorded onstructured questionnaire. Blood pressure of all subjects was recorded in sitting position by using mercury sphygmomanometer, three readings were taken and then mean was calculated. Blood samples were taken under aseptic measures and serum aldosterone levels were estimated by using ELISA technique. All ethical considerations were specially observed. The collected data was analyzed by using SPSS version 17.0. All the quantitative variables were presented as mean±SD (Standard deviation). One way ANOVA was applied to observe the group mean differences. Post hoc was performed for multiple comparisons among the groups.Pearson correlation was applied to observe correlations between blood pressure, serum aldosterone and duration of cotton dust exposureP-value of <0.05 was considered as statistically significant.

# **RESULTS**

Table 1 is presenting the comparison of the mean systolic, diastolic blood pressure and serum aldosterone between the three study groups by ANOVA.Systolic blood pressure (SBP) was lower in group III as compared to group I and group II, this was significantly different (p value 0.04\*). However no significantly difference was obtained with respect to diastolicblood pressure (DSB)( P value 0.79). Mean ±SD for serum aldosterone(ηg/mI) values for control, group II and III are 1.90±0.08,1.76 ± 0.46and 1.30±0.09 respectively. These levels are lower in group II and lowest in group III as compare to group I. Highly significant difference was noted among the three study groups ( P value 0.000\*\*)

Pearson correlation was applied to observe correlations between aldosterone and blood pressure. No significant correlation of aldosterone was found with systolic (P value 0.730†) and diastolic blood pressure (P value 0.69†) (Table2) may be because of small sample size.

Figure 1 is indicating the multiple comparison of SBP among the groups bypost hoc tukytest. It is showing that the SBP of the group III is on the lower side(119±7.84)as compared to control group(123.46±5.79) and group II(123.62±4.79) and it is significantly different from other two groups. P value for control and groupIII was 0.04\*and p value 0.03\* was noted for comparison between group III and II. Group II and group I was also not significantly differ from each other (Pvalue 0.99)

Figure 2 is showing comparison of mean DBP among the threegroups by post hoc tuky test. The mean± SD diastolic blood pressure (DBP) values for control group, group II and group III are  $76.55 \pm 5.79$ ,  $79.14 \pm 5.52$ and 76.53 ± 5.24 respectively. These groups are not significantly differentfrom each other with respect to DBP. comparisons of mean aldosterone demonstrated in figure 3. t is indicating that the serum aldosterone of group III islowest than control group and group II and it is highly significant different from control (p value 0.001\*\*) as well as from group II (p value 0.001\*\*). Comparison between control group and group II is indicating relatively lower levels of aldosterone in group II but no significant difference is observed between these groups (P value 0.26)

Table 1: Comparison of parameters between the study groups by ANOVA

Study groups (n= 84)	SBP (mm Hg) Mean ±SD	DBP (mmHg) Mean ± SD	Aldosterone(ηg/ml) Mean ± SD
Control Group(n=26)	123.46 ±5.79	76.55 ± 5.79	1.90 ± 0.08
Group II(n=29)	123.62 ± 4.79	79.14± 5.52	1.76± 0.098
Group III(n=29)	119 ± 7.84	76.53± 5.24	1.3± 0.09
P value	0.041*	0.79	0.000**

Group1 (control group):: less than one month exposure to cotton dust, Group III: >5 years exposure,

\*\*p < 0.001 - highly significant

Group II: 3-5 years exposure \*p < 0.05 - significant

tp > 0.05 - non-significant

Table 2: Correlation coefficient (r) and p-value among study variables in whole population

Study variables	Correlation coefficient (r)	p-value
Blood pressure(systolic) – Blood pressure(diastolic)	0.512	<0.001**
Blood pressure(systolic) – Serum Aldosterone	0.038	0.730†
Blood pressure(diastolic) Serum Aldosterone	0.044	0.691†

Fig. 1: Comparison of systolic blood pressure (SBP) among the study groups

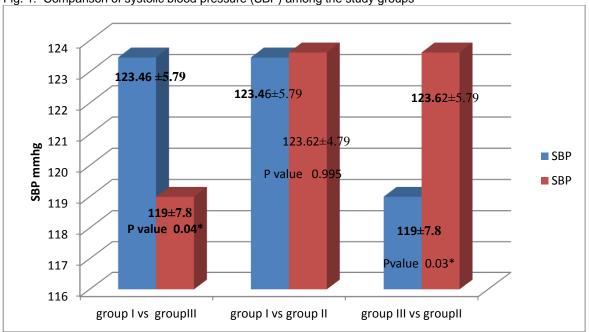
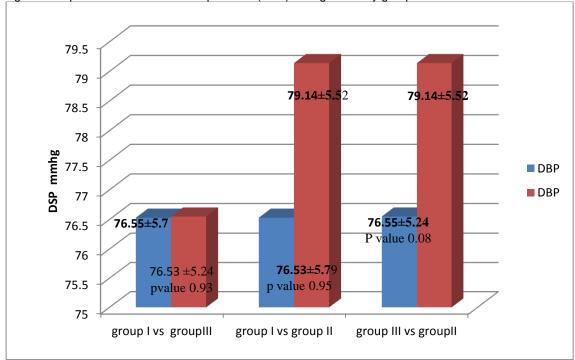


Fig. 2: Comparison of Diastolic blood pressure (DBP)among the study groups



Multiple comparisons by post hoc test

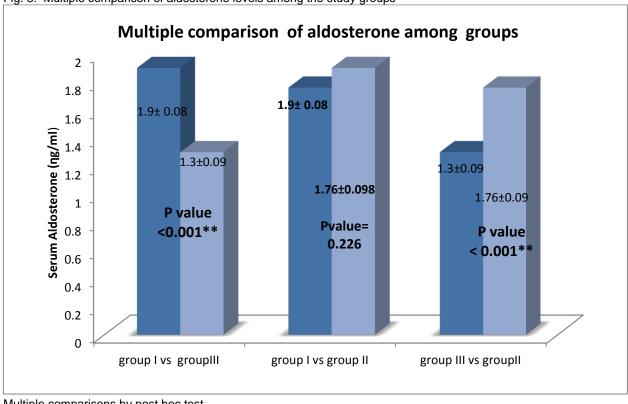


Fig. 3: Multiple comparison of aldosterone levels among the study groups

Multiple comparisons by post hoc test

### DISCUSSION

According to estimates from the WHO Global Health Observatory (GHO) data, about 30 deaths per 100 000 are attributable to indoor air pollution, while about 25 deaths per 100 000 are attributable to outdoor air pollution. 18 The cotton dust induced environmental agents, are the major contributors of chronic airway disease in textile workers. 1 Particulate matter activates the inflammatory process in airways and releases different cytokines which parenchyma and damage the lung pulmonary endothelium.17Endothelial damage ultimately leads to endothelial dysfunction. 12 Angiotensin converting enzyme (ACE), which is expressed mainly in endothelial cells is sensitive to hemodynamic stimuli such as shear stress. It participates in both short term and long term control of vascular structure and function 12,14. As pulmonary endothelium is a major site for producing membrane bound angiotensin converting enzyme, so any damage to endothelium results in reduction of angiotensin converting enzyme<sup>11</sup>.

At present sufficient data is not available on the effects of cotton dust exposure in relation to serum aldosterone levels and its subsequenteffect on arterial pressure. Moreover, no study was found on all these parameters in the same subject exposed to cotton dust for varving durations.

The present study was conducted to find out the effects of cotton dust exposure and its relationship with blood pressure in three different groups of subjects working in polluted environment of textile industry of Faisalabad.

On comparison of mean systolic and diastolic blood pressure with its effects on serum aldosterone between the three study groups showed that these levels are lower in group II and lowest in group III as compare to control group . Highly significant difference was noted among the three

study groupswith respect to systolic blood pressure. It is evident from this that that the environmental pollution has damaging effects on arterial pressure, 2,5 however no significant correlation was seen between serum aldosterone and systolic blood pressure. This may be due to small sample size or may be due to the fact that systolic blood pressure is under the control of many other parameters apart from blood aldosterone levels. 12

Similarly comparisons of mean aldosterone shows that levels are significantly lowest in groupIII which has maximum exposure of cotton dust pollution as compared to group II and control group. Which shows that exposure for five to ten years to cotton dust pollutionmay result in endothelial dysfunction which then lowers the serum aldosterone levels. However, the dusty environment of textile industry ultimately damages the pulmonary endothelium on prolonged exposure which may affects the blood pressure in human beings.

Limitations: Apart from cotton dust, many other factors such as noise pollution, smoking and obesity can also affect the human blood pressure in long run but because of our limited resources these parameters were not included in the study.

# CONCLUSION

This study shows that after a prolongedexposure from about five to ten years, the cotton dust may damage the pulmonary endothelium leading to lower levels blood pressure in exposed persons.

# **REFERENCES**

- Alemu K, Kumie A, Davey G. Byssinosis and other respiratory symptoms among factory workers in Akaki textile factory, Ethiopia. Ethiop. J. Health Dev 2010; 24(2):133-139.
- Khan, A. J., and Nanchal, R. Cotton dust lung diseases. Current Opinion in Pulmonary Medicine, 2007 march,13(2):137-141.
- Chung.K.F,Zhang.J and Zhong.N. Outdoor air pollution and respiratory health in Asia.Respirology (2011) 16, 1023–1026
- Rajkumar.P,Pattabi.k, Vadivoo.S, etal.A cross-sectional study on prevalence of chronic obstructive pulmonary disease (COPD) in India: rationale and methods. BMJ Open 2017;7:e015211. doi:10.1136/bmjopen-2016-01521
- Krstev S, Shu X, and GaoY.T, et al. Occupation and chronic bronchitis among Chinese women. JOEM 2008; 50: 64-71.
- Samantha R, Robret D and Sewell E,et al. Correlative measurement of four biological contaminants on cotton lint, and their implications for occupational health. IJOEH2006; 12: 120-125.
- 7. Farzaneh,M, Jamshidiha F, and Kowsarian, S.. Inhalational Lung Disease. IJOEM2009;1: 11-20.
- Nodari S, Corulli A, Manerba A, Metra M, Apostoli P, and Dei Cas, L. Endothelial damage due to air pollution. Heart Int 2006; 2: 115-125.
- Orfanos S, Mavrommati I, Korovesi I, and Roussos C. Pulmonary endothelium in acute lung injury: from basic

- science to the critically ill. Intensive Care Med 2009; 2: 215-227.
- Langleben D, Orfanos S and Giovinazzo M, et al. Pulmonary capillary endothelial metabolic dysfunction: severity in pulmonary arterial hypertension related to connective tissue disease versus idiopathic pulmonary arterial hypertension. Arthritis & Rheumatism April 2008; vol 58, No 4: pp1156-1164.
- Barauna V.G, Campos L, Miyakawa A, and Krieger J. ACE as a Mechanosensor to Shear Stress Influences the Control of Its Own Regulation via Phosphorylation of Cytoplasmic Ser1270. PLoS ONE 2011; 6(8): e22803.
- Nappi M, and SiegA. Aldosterone and aldosterone receptors antagonists in patients with chronic heart failure. Vasc health Risk Manag 2011; vol7:353-363.
- Rafiq K, Hitomi H, Nakano D and Nishiyama A. Pathophysiological Roles of Aldosterone and Mineralocorticoid Receptor in the Kidney. J pharmacolosci 2010; 115: 1-7.
- Tomaschitz A, MaerzW and Pilz S. Aldosterone/renin ratio determines peripheral and central blood pressure values over a broad range. JACC 2010; 55: 2171-2180.
- Sierra C, and Ruilope L. Review: Role of the selective aldosterone receptor blockers in arterial hypertension. JRAAS 2004; 5: 23-25.
- Hulley SB, Cummings SR, Browner WS, Grady D, Newman TB. Designing clinical research: an epidemiologic approach. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013. Appendix 6C, page 79
- Park H, Li. Z. Yang and X. O.chang et al.. A distinct lineage of CD4 T cells regulates tissue inflammation by producing interleukin. Nature immunol 2005. vol 6: 1133-1141.
- WHO. (2014). "Media Centre". Retrieved September 2016, from World Health Organization:
- http://www.who.int/mediacentre/news/releases/2014/air-pollution/en/.