Egyptian Journal of Occupational Medicine, 2012; 36 (1) : 107-121

OXIDATIVE STRESS AND IMMUNOGLOBULIN LEVELS IN WORKERS EXPOSED TO COTTON DUST

By

Shaker DA*

** Department of Occupational and Environmental Medicine,*

Faculty of Medicine, Cairo University, Egypt.

Abstract:

Background: Among the different occupational hazards to which cotton industry workers are exposed such as accidents, fire, disabilities, noise and heat, of all, the largest health hazard is due to inhalation of cotton dust. Prolonged cotton dust inhalation causes various known diseases in textile industry such as byssinosis and chronic obstructive pulmonary disease. These disorders seems at least partly, be mediated by oxidative stress and altered immune system. **Objective:** To assess oxidative stress and immunoglobulin levels (IgG & IgM) in workers exposed to cotton dust. **Setting:** This study was conducted in Misr Helwan Industry for Spinning and Weaving on cotton dust exposed workers in the spinning department. **Subjects and methods:** Forty two workers exposed to cotton dust were included in this study together with 30 non exposed apparently healthy subjects taken as controls. Every participant of this study was subjected to occupational history taking, detailed questionnaire, thorough clinical examination, assessment of serum malondialdehyde (MDA), nitric oxide, superoxide dismutase (SOD), glutathione peroxidase, catalase, and total antioxidant status, and measuring of immunoglobulin levels (IgM & IgG). Both groups were matched as regards age, sex, smoking habit, duration of employment and socioeconomic status.

Results: The levels of oxidants biomarkers (nitric oxide and MDA) were significantly elevated, and antioxidants (glutathione peroxidase, catalase, SOD and total antioxidant) were significantly decreased in exposed workers compared with the controls. Statistically significant increase in the immunoglobulin levels was observed in cotton exposed workers (vs. controls). No significant difference was found in oxidant /

antioxidant status and immunoglobulin levels between exposed smokers and exposed non-smokers. Smoker exposed workers showed statistically significant elevation in their levels of oxidant biomarkers (MDA & nitric oxide) and immunoglobulin M when compared to those in controls. Age and duration of employment in exposed workers were significantly negatively correlated with total antioxidant status while no significant correlation was found with other antioxidants nor with oxidant parameters.

Conclusion: Workers exposed to cotton dust were under great oxidative stress as manifested by a rise in oxidant biomarkers and a reduction in antioxidants. Cotton dust stimulates humoral immunity as detected by the increases in the immunoglobulin levels. Smoking may have synergistic effect with cotton dust in induction of oxidative stress. These bio-functional markers might be useful in screening and surveillance for occupational hazard.

Key Words: cotton dust, oxidative stress, immunoglobulin levels

Introduction

The current literature supports the possibility that occupational dust exposure results in increased reactive oxygen species (ROS) or free radical generation (Banerjee et al., 2001) and may causes alteration of the immune system (Sultana et al.,. 2012).

Cotton dust has a complex nature and is heavily contaminated by Gram negative bacteria, soils, chemicals, allergens and fungi, making the mechanism of induced respiratory disorders a complicated issue (Holt, 1990).

Epidemiologic studies have shown that chronic occupational exposure to cotton dust may induce chronic airway disease. Among many agents present in cotton dusts, there is substantial evidence to suggest that bacterial endotoxin is a major causative agent that contributes to airway inflammation and airflow obstruction (Christiani et al., 2001). The concentration of inhaled endotoxin in bioaerosol is associated strongly with the development of airway disease in cotton and agricultural workers (Wang et al., 2003). Exposure to endotoxin may result in endothelial cell damage, influx of blood leukocytes into the airway, production of different cytokines, as well as increased production of oxygen metabolites, such as reactive oxygen species that may induce oxidative lung injury (Victor and De la Fuente, 2003).

Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. Disturbances in the

normal redox state of cells can cause toxic effects through the production of peroxides and free radicals that damage all components of the cell, including proteins, lipids and DNA. Further, some reactive oxidative species act as cellular messengers in redox signaling. Thus, oxidative stress can cause disruptions in normal mechanisms of cellular signaling (Marnett, 2002).

Lipid peroxidation is one of the most widely used indicators of free radical formation, a key indicator of oxidative stress. Unsaturated fatty acids such as those present in cellular membranes are a common target for free radicals. Reactions typically occur as a chain reaction where a free radical will capture a hydrogen moiety from an unsaturated carbon to form water. This leaves an unpaired electron on the fatty acid that is then capable of capturing oxygen, forming a peroxy radical. Lipid peroxides are unstable and decompose to form a complex series of compounds, which include reactive carbonyl compounds, such as malondialdehyde (MDA) (Pryor et al., 1976).

The immune system uses the lethal effects of oxidants by making production of oxidizing species a central part of its mechanism of killing pathogens; with activated phagocytes producing both reactive oxygen species (ROS) and reactive nitrogen species. These include superoxide, nitric oxide and their particularly reactive product, peroxynitrite (Valko et al., 2007). Although the use of these highly reactive compounds in the cytotoxic response of phagocytes causes damage to host tissues, the non-specificity of these oxidants is an advantage since they will damage almost every part of their target cell. This prevents a pathogen from escaping this part of immune response by mutation of a single molecular target (Perricone et al., 2009).

Aim of the work:

The aim of this study was to assess oxidant/antioxidant status and immunoglobulin levels (IgG, IgM) in workers occupationally exposed to cotton dust in the spinning department in textile industry.

Subjects and methods

The current study was carried out in Misr Helwan industry for spinning and textile in Cairo governorate. The study population consisted of 72 subjects who were divided into exposed and control groups.

Inclusion criteria:

- Exposed group: Forty two male workers employed in the cotton industry located

in Helwan district were selected. Prior to the sample collection, subjects were interviewed for information of age, tobacco habits, detailed occupational history and medical history. Their ages varied between 31-59 years (44.7 ± 5.85). None of the exposed workers used any protective equipment during working hours.

- Healthy controls: Thirty male healthy volunteers not occupationally exposed to any occupational dust were matched for age that ranged from 38-57 yrs (45.4 ± 5.66), sex, smoking habits and socio-economic status. Only those proved to be in a good state of health and free from signs of chronic diseases or disorders were included.

Exclusion criteria:

The subjects having history of diseases which induce oxidative stress such as diabetes mellitus, hypertension, respiratory diseases, cardiovascular diseases etc. were excluded from the study.

All workers were interviewed using a special questionnaire including general history, detailed occupational history along with full clinical examination were carried out. The purpose of our study was explained to the subjects and their consent was taken. After obtaining prior consent, venous blood samples were collected from the subjects under aseptic precautions. Out of 8 ml blood collected, 4 ml was poured in sterile heparinized bulb and 4 ml allowed to clot. Serum and plasma were separated by centrifugation at 3000 rpm for 10 minutes at room temperature. All the samples were analyzed on the same day of collection. Serum MDA levels were measured by reacting it with thiobarbituric acid at high temperature to form pink colored complex as Kei Satoh method (Satoh, 1978).

Nitric oxide was determined by the addition of zinc sulfate to deproteinate serum samples and then nitrate is reduced to nitrite by vanadium III and chloride followed by the addition of 100 µL of Griess reagent (equal mixture of 1% sulphanilamide in 5% phosphoric acid and 0.1% N-(1-naphthyl) ethylenediamine hydrochloride in distilled water). The plates were incubated for 30 minutes at room temperature and the optical density was measured at 540 nm using the ELISA (Enzyme-Linked Immunosorbent Assay) reader (Bio-Tek Instruments INC, USA) (Ghasemi et al., 2007).

SOD was measured with a commercially available Ransod kit (Randox Laboratories, Ltd, UK). Briefly The SOD

activity was assessed by the inhibition of the reduction of nitroblue tetrazolium by superoxide anion. The assay mixture was composed of 0.05 M phosphate buffer, pH 8.60,10.2 M EDTA, and 10.4 M nitroblue tetrazolium; dimethyl sulfoxide were added under stirring to the above mixture. The absorbance of each solution was read at 550 nm using Beckman spectrophotometry (USA). One unit of enzymatic activity is defined as the amount of enzyme causing 50% inhibition of the reduction to formosan observed in the blank (Arthur and Boyne, 1985).

Glutathione peroxidase was measured by a Ransel kit (Randox Laboratories, Ltd, UK). Its function is to catalyze the oxidation of reduced glutathione (GSH) to oxidized glutathione (GSSG) by using t-butyl hydroperoxide as the substrate for the assay of the enzyme. The rate of formation of GSSG is measured by means of the glutathione reductase reaction. The oxidation of NADPH is measured at 340 nm using Beckman spectrophotometry (USA) (Paglia and Valentine, 1967).

Catalase was measured by a Ransel kit (Randox Laboratories, Ltd, UK). Catalase catalyzes the breakdown of H2O2 into 2 moles of water and 1 mole of oxygen. The rate of decomposition of H2O2 by catalase is measured using Beckman spectrophotometry (USA)at a wavelength of 230 nm. Ethanol is added to stabilize the hemolysate by breaking down the "complex II" of catalase and H2O2 (Aebi, 1973).

Total antioxidant capacity was measured using Randox total antioxidant status kit (UK) in which ABTS (2, 2-Azino-di [3- ethyl benzthiazolin sulphanate]) is incubated with a peroxidase and H2O2 to produce the radical cation ABTS+. This has a stable blue green color, which is measured at 600 nm. Antioxidants in the added sample cause suppression of this color production to a degree which is proportional to their concentration (Miller et al., 1993).

Immunoglobulin levels (IgM and IgG) were detected in serum samples using Abcam`s Humann kit according to manufacturers instruction.

Statistical methods:

Data were checked, coded, entered and analyzed using computer based statistical package for social sciences (SPSS) for windows 16.0 program. Comparison between quantitative data of the study groups was done using student`s t-test, while comparison between qualitative data was done by chi-square test. Pearson correlation coefficient was used for testing

the association between two continuous variables. Probability value of $p < 0.05$ was considered the limit below which the difference of the values would be statistically significant (Daniel, 1987).

Results

The exposed group consisted of 42 male workers occupationally exposed to cotton dust; their mean age was 44.7 ± 5.85 years with mean duration of employment of 24.6 ± 7.35 years. The control group included 30 male workers not occupationally exposed to cotton dust. Their mean age was 45.4 ± 5.66 years. There was no statistically significant difference between exposed and control (non-exposed) group as regards age, sex, duration of employment and smoking habit ($p > 0.05$). The frequency of smokers among exposed workers and control group were 15 (35.7%) and 12 (40.1%) respectively, and there was no statistically significant difference between the 2 groups as regards smoking index. The two groups studied had similar demographic characteristics (table 1).

Table 2 shows levels of serum MDA, serum nitric oxide, superoxide dismutase activity, glutathione peroxidase, catalase and total antioxidants status in cotton industry workers and control group. Serum MDA and nitric oxide levels were highly significantly increased in cotton exposed group ($p < 0.001$) as compared to controls. There were significant decreases in superoxide dismutase, glutathione peroxidase, catalase and total antioxidant status as compared to control group ($p < 0.05$).

There were statistically significant differences between exposed and control groups as regards immunoglobulin levels (IgM & IgG) ($p < 0.05$) which were highly increased in cotton exposed workers (table 3).

As shown in table 4 there were no statistically significant differences between smokers and non-smokers in cotton industry workers as regards levels of oxidants parameters & antioxidants and immunoglobulin levels ($p > 0.05$).

There were statistically significant differences between smokers in both exposed and control groups as regards levels of oxidants biomarkers (MDA & nitric oxide) which were increased in exposed smokers ($p < 0.05$) while antioxidants were not significantly decreased in exposed smokers as compared to smokers in control group ($p > 0.05$). As regards levels of immunoglobulins (IgM & IgG), there was statistically significant increase in immunoglobulin M in exposed smokers

when compared to those in controls ($p < 0.05$) while no significant difference between both groups as regards IgG level ($p > 0.05$) (table 5).

Age and duration of employment in cotton exposed group were significantly negatively correlated with total antioxidant status ($r = -0.337$; $r = -0.240$ respectively) ($p < 0.05$) while other antioxidants (SOD , glutathione peroxidase and catalase) and oxidants (MDA & nitric oxide) showed no significant correlation ($p > 0.05$). As regards immunoglobulin levels (IgM & IgG), there was significant negative correlation between age and IgG level ($r = -0.334$) ($p < 0.05$) while no significant correlation was found between duration of employment and immunoglobulin levels ($p > 0.05$) (table 6).

Table 1: Demographic characteristics of the studied exposed and control groups.

Parameter		Exposed N = 42		Control N = 30		t-test	P
Age (years)	Range	31-59		38-57		-.479	> 0.05
	Mean	44.7		45.4			
	SD	5.85		5.66			
Duration of employment (years)	Range	9 - 43		10 - 37		1.240	> 0.05
	Mean	24.6		22.4			
	SD	7.35		8.04			
S.I. † (pack. year)	Range	2.5 - 60		2.5 - 45		0.007	> 0.05
	Mean	19.83		19.79			
	SD	14.83		13.91			
Smoking habit		No	%	No	%	χ^2	> 0.05
	Smokers	15	35.7	12	40.1	0.137	
	Non smokers	27	64.3	18	59.9		

† Smoking Index (Pack. year);

$P < 0.05$* and $p < 0.001$** were considered statistically significant and highly significant, respectively.

Table 2 : Comparison between cotton exposed workers and controls as regards oxidants biomarkers and antioxidants levels.

Parameter	Exposed group Mean ± SD	Control Group Mean ± SD	t-test	P
Serum MDA (nmol/ml)	6.56 ± 3.23	1.59 ± 0.56	8.307	< 0.001**
Serum nitric oxide (umol/L)	31.7 ± 8.07	18.5 ± 2.41	8.630	< 0.001**
Superoxide dismutase (U/ml)	1.37 ± 0.77	5.36 ± 1.87	-12.414	< 0.05*
Glutathione peroxidase (U/ml)	23.75 ± 6.27	50.56 ± 8.90	-14.994	< 0.05*
Blood catalase (U/ml)	176.8 ± 34.6	337.7 ± 67.8	-13.176	< 0.001**
Plasma total antioxidant (mmol/L)	0.76 ± 0.31	2.67 ± 0.52	-19.265	< 0.001**

P < 0.05* statistically significant and p < 0.001** highly significant.

Table 3: Comparison between cotton exposed workers and controls as regards immunoglobulin Levels (IgM and IgG).

Parameter	Exposed group Mean ± SD	Control Mean ± SD	t-test	P
IgM (mg/dl)	115.98 ± 49.8	69.28 ± 7.3	5.076	< 0.001**
IgG (mg/dl)	1051.4 ± 219.6	683.61 ± 68.9	8.849	< 0.05*

P < 0.05* statistically significant and p < 0.001** highly significant.

Table 4 : Comparison between smokers and non-smokers in cotton exposed workers as regards levels of oxidants & antioxidants and immunoglobulin levels.

Parameter	Exposed smokers Mean ± SD	Exposed non-smokers Mean ± SD	t-test	P
Serum MDA (nmol/ml)	7.36 ± 2.91	6.12 ± 3.36	1.195	> 0.05
Serum nitric oxide (umol/L)	30.22 ± 7.62	32.52 ± 8.33	-0.883	> 0.05
Superoxide dismutase (U/ml)	1.35 ± 0.73	1.39 ± 0.81	-0.152	> 0.05
Glutathione peroxidase (U/ml)	22.90 ± 5.85	24.23 ± 6.55	-0.652	> 0.05
Blood catalase (U/ml)	172.4 ± 32.8	179.3 ± 35.9	-0.618	> 0.05
Plasma total antioxidant (mmol/L)	0.75 ± 0.32	0.77 ± 0.31	-0.132	> 0.05
IgM (mg/dl)	142.67 ± 65.8	101.15 ± 30.7	2.790	> 0.05
IgG (mg/dl)	1044.0 ± 150.6	1055.5 ± 252.6	-0.160	> 0.05

Table 5 : Comparison between smokers in both exposed group and control group as regards levels of oxidants & antioxidants and immunoglobulin levels.

Parameter	Exposed smokers Mean ± SD	Control smokers Mean ± SD	t-test	P
Serum MDA (nmol/ml)	7.36 ± 2.91	1.51 ± 0.57	6.81	< 0.05*
Serum nitric oxide (umol/L)	30.22 ± 7.62	19.85 ± 1.18	4.64	< 0.05*
Superoxide dismutase (U/ml)	1.35 ± 0.73	6.16 ± 0.95	-14.83	> 0.05
Glutathione peroxidase (U/ml)	22.90 ± 5.85	51.78 ± 8.27	-10.61	> 0.05
Blood catalase (U/ml)	172.4 ± 32.8	356.2 ± 34.1	-14.20	> 0.05
Plasma total antioxidant (mmol/L)	0.75 ± 0.32	2.70 ± 0.33	-15.08	> 0.05
IgM (mg/dl)	142.67 ± 65.8	66.70 ± 9.97	3.942	< 0.001**
IgG (mg/dl)	1044.0 ± 150.6	666.78 ± 72.12	7.957	> 0.05

P < 0.05* statistically significant and p < 0.001** highly significant.

Table 6: Pearson`s correlations between age & duration of exposure and oxidant / antioxidant status & immunoglobulin levels (IgM & IgG) among exposed group.

Parameter	Age		Duration of exposure	
	r	P	r	P
Serum MDA	0.024	> 0.05	0.048	> 0.05
Serum Nitric oxide	0.017	> 0.05	0.009	> 0.05
Superoxide dismutase	-0.011	> 0.05	-0.023	> 0.05
Glutathione peroxidase	-0.031	> 0.05	-0.060	> 0.05
Blood Catalase	-0.051	> 0.05	-0.147	> 0.05
Plasma total antioxidant	-0.337	< 0.05*	-0.240	< 0.05*
IgM	0.028	> 0.05	-0.056	> 0.05
IgG	-0.334	< 0.05*	-0.156	> 0.05

P < 0.05* statistically significant.

Discussion

Occupational exposure to cotton dust has been shown to cause airway disease characterized by acute changes in airflow and by chronic obstructive lung disease. Endotoxin is the principal component of cotton responsible for the development of inflammation of the airway and airflow obstruction. In vitro, lipolysaccharides intraperitoneal injection results in a significant increase of reactive oxygen species production by peritoneal leukocytes, generating an oxidative stress situation (Victor and De la Fuente, 2003). The oxidant/antioxidant theory postulates that an excess of oxidants in the lung promotes cellular and tissue damage, and is the major initiator of the disease process (Tsuji et al., 1998). Previous researches have suggested that chronic occupational endotoxin exposure is associated with faster decline of lung function (Victor and De la Fuente, 2003).

It is already known that the exposure to various toxic substances such as cotton dust increases production of reactive oxygen species (ROS). The increase of ROS production with the consequent disturbance of the oxidative balance in the cell, called oxidative stress, disturbs the metabolism

of macromolecules. The oxidative stress causes the damage to membrane lipids which manifests as the increase of the malondialdehyde (MDA) concentration (Marnett, 2002).

Cells have a variety of defense mechanisms to ameliorate the harmful effects of ROS. Superoxide dismutase (SOD) catalyzes the conversion of two superoxide anions into a molecule of hydrogen peroxide (H2O2) and oxygen (O2). In the peroxisomes of eukaryotic cells, the enzyme catalase converts H2O2 to water and oxygen, and thus completes the detoxification initiated by SOD. Glutathione peroxidase is a group of enzymes containing selenium, which also catalyze the degradation of hydrogen peroxide, as well as organic peroxides to alcohols (Halliwell, 1996).

Our results demonstrate highly significantly increased levels of oxidants biomarkers (serum MDA and NO.) in exposed group when compared to controls (table 2). Increased lipid peroxidation in the blood of exposed subjects warns that oxygen free radicals have increased in the body and thus might attack cells, which, in the long-term, results in multi-organ damage.

In the present study, serum glutathione peroxidase (GPX) which is a selenoenzyme responsible for elimination of ROS, was significantly lowered in cotton exposed workers in comparison to their controls (23.75 ± 6.27, 50.56 ± 8.90, respectively, $P < 0.05$) (table 2). Decrease in the activity of antioxidants may be due to exhaustion of the enzymes by reactive oxygen species (Perricone et al., 2009).

Our results are in agreement with that of Suryakar et al. (2010) who reported highly significant increases in serum MDA and nitric oxide in cotton industry workers as compared to controls. The activities of SOD, glutathione peroxidase, glutathione reductase were found to be significantly decreased in cotton industry workers. Their study demonstrated significant decreases in catalase activity and total antioxidant status in exposed workers. They concluded that cotton dust exposure induces oxidative stress among textile industry workers which may contribute to respiratory disorders.

The negative effect of either passive or active smoking on the level of antioxidants and the positive effect on the level of oxidants were discussed in many studies. Sayyed et al. (2008) reported an increase in the level of oxidants with a simultaneous decrease in the level of antioxidant enzymes

in association with smoking. This goes in accordance with the slight increase in oxidant biomarkers (MDA & nitric oxide) and the decrement in SOD, glutathione peroxidase, catalase and total antioxidant status in exposed smokers as compared to exposed non-smokers ($P > 0.05$; table 4). Lack of statistically significant differences between smoker and non smokers exposed persons may be due to the fact that the non-smokers might be passive smokers.

However, our findings demonstrate statistically significant increases in the oxidant biomarkers ($P < 0.05$) and decreases in antioxidants status (although not significant) ($P > 0.05$) in exposed smokers when compared to those in controls (table 5). This may be explained by the additional exposure of smokers in the cotton industry to organic dust and its constituent of endotoxins which may have synergistic effect to cotton dust in inducing oxidative stress. This observation strongly supports the concept that increased production of oxygen derived free radicals contributes to lipid peroxidation in cotton exposed workers (Suryakar et al., 2010).

In the present study, we selected IgM and IgG as markers of the humoral immunity. The results showed statistically significant higher levels of immunoglobulin M and immunoglobulin G in cotton exposed workers compared to control group (table 3; $P < 0.05$).

Byssinosis is a chronic respiratory disease of cotton, flax and soft hemp workers. In accordance with our findings, Karnik et al. (1987) reported that workers exposed to cotton dust in textile mills showed significant rise in IgG with or without byssinosis; IgA and IgM increased (non significantly) only in byssinosis. Similar changes in immunoglobulins have also been described by other research workers (Massoud and Taylor, 1964 ; Kamat et al., 1979).

Our results are also consistent with that obtained by Sultana et al. (2012) who found that occupational exposure to cotton dust causes a wide range of physical and psychological health problems in the garment workers that may also affect their immune function as evident by the significant increase in the mean IgG level in cotton exposed workers when compared to the controls. They conclude in their research that garment workers constantly inhale fiber particles which can probably stimulate antibody production (including IgG); these antibodies could kill the bacterial cells.

Also, immunoglobulin levels were elevated in exposed smokers when compared to those in control group with only significant elevation in IgM level (table 5), meaning that smoking has no significant effect in immunoglobulin level in our studied population.

Although, age and duration of exposure are important factors in inducing elevations of oxidants and reductions in antioxidants, yet the present work detected no significant relationships between duration of exposure and oxidant / antioxidant status apart from total antioxidant status which was significantly negatively correlated with age and duration of employment. We attribute those results to individual susceptibility, which led to affection of the tested parameters even with long duration of exposure.

Our findings showed significant negative correlation between age and immunoglobulin G ($r = 0.334$; $p < 0.05$) (table 6) despite the known positive effect of age on IgG level. This may be explained by the fact that states that serum concentration of the immunoglobulin G may be affected by smoking which has a negative effect on its level (Gonzalez-Quintela, et al., 2007).

The levels of oxidant biomarkers and total oxidant status were determined to demonstrate the role of oxidative mechanisms. Since it was shown that endotoxin exhibited an increase of MDA and total oxidative status, the overall conclusion was that cotton dust may play a role in induction of oxidative damage by increasing lipid peroxidation and oxidative stress. Smoking may have a synergistic effect with cotton dust in induction of oxidative stress. In addition, cotton dust may alter the immune system. Consequently, protection measures to avoid the cotton dust exposure, periodical medical examinations, measurements of endotoxin concentrations and anti-smoking campaigns are desirable measures to protect workers against cotton dust-induced oxidative stress. Proper nutrition with diet rich in antioxidants e.g. (vitamin C, vitamin A). Also, antioxidant supplementation may offer protection against cotton dust induced oxidative stress. The results need to be confirmed on larger number of workers.

Acknowledgment

The author is grateful to all workers involved in this study for their co-operation.

References

1. Aebi H (1973): Catalases. In: Bergmeyer HU (Ed.) Methods of enzymatic analysis (pp. 673-84). Academic Press, New York.
2. Arthur JR and Boyne R (1985):. Superoxide dismutase and glutathione peroxidase activities

in neutrophils from selenium deficient and copper deficient cattle. Life Sci. 36: 1569-75.

3. Banerjee BD, Seth V and Ahmed RS (2001): Pesticide induced oxidative stress: perspectives and trends. Rev Environ Health. 16: 1-40.
4. Christiani DC, Wang XR, Pan LD, Zhang HX, Sun BX, Dai H, Eisen EA, Wegman DH and Olenchock SA (2001): Longitudinal changes in pulmonary function and respiratory symptoms in cotton textile workers: a 15-yr follow-up study. Am J Respir Crit Care Med .163:847–853.
5. Daniel WW (1987): Biostatistics, a foundation for analysis in the health science. .Wiley series in probability and mathematical statistics, USA..
6. Fang SC, Mehta AJ, Hang JQ, Eisen EA, Dai HL, Zhang HX, Su L and Christiani DC (2013): Cotton dust, endotoxin and cancer mortality among the Shanghai textile workers cohort: a 30-year analysis. Occup environ med. 70 (10):722-9.
7. Ghasemi A, Hedayati M and Biabani H (2007): Protien precipitation methods evaluated for determination of serum nitric oxide end products by the Griess assay. Journal of Medical Science Research. 2:29–32.
8. Gonzalez-Quintela A, Alende R, Gude F, Campos J, Rey J, Meijide LM, Fernandez-Merino C and Vidal C (2007): Serum levels of immunoglobulins (IgG, IgA, IgM) in a general adult population and their relationship with alcohol consumption, smoking and common metabolic abnormalities. Clinical and Experimental Immunology. 151: 42–50.
9. Halliwell B (1996): Antioxidants in health and diseases. Ann Rev Nutr. 16: 33-50.
10. Jones DP (2000): Redox Potential of GSH/GSSG Couple: Assay and Biological Significance. Methods of Enzymology. 348:93-112.
11. Kamat SR, Taskar SP, Iyer ER and Naik M (1979): Discrimination between byssinosis and chronic bronchitis in cotton mill workers by serum immunoglobulin patterns. J Soc Occup Med. 29: 102.
12. Karnik AB, Parikh JR, Suthar AM, Patel MM, Thakore KN, Patel HH, Lakkad BC, Kulkarni PK and Nigam SK (1987): Immunoglobulins, serum proteins and lactate dehydrogenase levels in workers exposed to cotton dust. Indian J Med Res. 85: 222.
13. Marnett, LJ (2002): Oxy radicals, lipid peroxidation and DNA damage. Toxicology. 181–182, 219–222.
14. Massoud A and Taylor G (1964): Byssinosis antibody to cotton antigens in normal subjects and in cotton cardroom workers. Lancet. 284: 607-610.
15. Miller NJ, Rice-Evans C, Davies MJ, Gopinathan V and Milner A (1993): A novel method for measuring antioxidant capacity and its application for monitoring the antioxidant status in premature neonates. Clin Sci. 84:407-412.
16. Paglia DE and Valentine WN (1967): Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J Lab Clin Med. 70: 158-69.
17. Perricone C, De Carolis C and Perricone R (2009): Glutathione: a key player in autoimmunity. Autoimmun Rev. 8: 697–701.
18. Pryor WA, Stanley JP and Blair E (1976): Autoxidation of polyunsaturated fatty acids: II. A Suggested mechanism for the formation of TBA-reactive materials from prostaglandin-like endoperoxides. Lipids. 11:370-379.
19. Rajpurkar A, Dhabuwala C, Yang J and Haikun L (2000): Chronic cigarette smoking induces an oxidant-antioxidant imbalance in the testis. J Environ Pathol Toxicol. Oncol. 19:369-73.
20. Satoh K (1978): Serum lipid peroxide in cerebro-vascular disorder determined by a new colorimetric method. Clin Chim Acta. 90: 37-43.

21. Sayyed AK, Despande KH, Suryakar AN, Ankush RD and Katkam RV (2008): Oxidative stress and serum α1 Antitrypsin in smokers. Indian J Clin Biochem. 23 (4): 375-7.
22. Sultana R, Ferdous KJ, Hossain M, Zahid MS and Islam LN (2012): Immune functions of the garment workers. Int J Occup Environ Med. 3(4):195-200.
23. Tsuji C, Minhaz MU, Shioya S, Fukahori M, Tanigaki T and Nakazawa H (1998): The importance of polymorphonuclear leukocytes in lipopolysaccharide-induced superoxide anion production and lung injury: ex vivo observation in rat lungs. Lung.176:1–13.
24. Valko M, Leibfritz D, Moncol J, Cronin MTD, Mazur M and Telser J (2007): Free radicals and antioxidants in normal physiological functions and human disease. International Journal of Biochemistry & Cell Biology. 39 (1): 44–84.
25. Victor VM and De la Fuente M (2003): Several functions of immune cells in mice changed by oxidative stress caused by endotoxin. Physiol Res. 52:789–796.
26. Wang XR, Eisen EA, Zhang HX, Sun BX, Dai HL, Pan LD, Wegman DH, Olenchock SA and Christiani DC (2003): Respiratory symptoms and cotton dust exposure; results of a 15 year follow up observation. Occup Environ Med. 60:935–941.