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RESEARCH ARTICLE

Exposure To Coal Mine Dust Predisposes Mine Workers to Oxidative Stress and Diabetes Mellitus

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ABSTRACT:
Exposure to coal mine dust has several health implications and diseases of the respiratory and cardiovascular have been reported. This study intended to investigate the effect of exposure to coal mine dust on the levels of plasma glucose (PG) and serum antioxidants in the miners. The levels of fasting PG (FPG), postprandial PG (PPG), superoxide dismutase (SOD) and glutathione peroxidise (GPX), thiobarbituric acid reactive substances (TBARS) of the miners exposed to coal mine dust for 10 and 20 years were compared to those of the controls. The observations of the study reveal that the miners remain unaffected during the 10 year exposure period and their glycemic status was under control. But high PPG levels ($p < 0.001$) were seen in miners exposed to coal mine dust for 20 years and thus prolonged exposure may predispose them to Type 2 diabetes mellitus. The low levels of SOD and GPX and high TBARS indicated that oxidative stress may be the underlying cause for this predisposition to diabetes.

KEYWORDS: Antioxidants, Coal mine dust, Oxidative stress, Plasma glucose, Type 2 diabetes.

INTRODUCTION:
The exposure to coal mine dust increases the risk for various non-communicable diseases such as ischemic heart disease, lower respiratory disease, cardiovascular disease and chronic obstructive pulmonary disease[1,2]. The epidemiological evidences for establishing an association between air pollution and incidence of diseases are abundant. These reports mention the biological effects of particulate matter[3]. However, the reports on the effect of coal mine dust on biochemical parameters are few.

Hospitalization or mortality is frequently associated with exposure to particulate matter (PM) because increased concentration of PM in air causes diseases such as Type 2 diabetes, cardio-vascular issues, respiratory problems and cancer.

Oxidative stress and inflammation are the prime processes which culminate in several health disorders. These processes are triggered by exposure to PM when PM in air gain entry into the body via dermal, oral and nasal routes. Oxidative stress and inflammation influence metabolic pathways and hence causes diabetes, dyslipidemia, fatty liver, etc[1,4]

The emissions from coal-fired power plants are of great concern[5]. Newborns, infants, children and elderly people are largely affected by the PM emissions by coal plants and mines. Premature deaths are also caused by coal pollution[6] Coal consists of several metals such as aluminium, iron, copper, zinc, arsenic, nickel, cadmium, cobalt and lead[7]. Chronic exposure to these metals causes toxic effect. Coal miners are exposed to large quantities of dust and acute or chronic inhalation of coal mine dust results largely in respiratory diseases, gastric cancer and heart attacks. Coal mine dust can be genotoxic and has been proved to cause DNA damage in lymphocytes[8].

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Table 1. Characteristics of the study participants

Characteristics	Miners		Non-miners	
	Male	Female	Male	Female
Sample size (N)	60	60	60	60
Age (years)				
Group I(36 – 45 years)	42.4 ± 2.2	39.5 ± 3.1	41.7 ± 2.1	40.4 ± 3.3
Group II(46 – 55 years)	48.1 ± 3.4	47.3 ± 1.1	50.3 ± 4.6	49.2 ± 2.8

This study was conducted to study the effect of exposure to coal mine dust in miners who work in a coal-fired thermal power plant situated at Neyveli. The impact of exposure on the glucose levels and antioxidants in miners was assessed.

METHODS:

This study is an open, parallel, prospective, non-randomized, concurrent study done in comparison with sex and age matched controls.

Study participants:

The characteristics of the study participants are given in Table 1. Men and women in the age group of 36 - 55 years and who have been working in coal mine for a period of 10 years and 20 years were enrolled as study cases. The miners were grouped as Group I consisting of individuals in the age group of 36 – 45 years and Group II consisting of individuals in the age group of 46 – 55 years. Males and females in the age group of 36-55 years who are residing in Neyveli Township were recruited as controls.

Approval from NLCIL (NLC India Limited) and Chief General Superintendent/Medical of NLCIL General Hospital was obtained for this study before the commencement of data collection. Approval from institutional ethics committee Vels Institute of Science, Technology and Advanced Studies, Chennai (VISTAS-SPS/IEC/V/2017/09) was obtained.

Exclusion criteria:

Miners and controls who smoke and drink, under medication, pregnant and lactating were excluded. Controls who use coal for domestic purpose were also excluded.

Measurement of Biochemical Parameters:

COBAS-C 311 fully automated analyser and ROCHE reagents, calibrators and controls were used. The biochemical parameters that were studied are fasting plasma glucose (FPG), post-prandial plasma glucose (PPPG), glycosylated haemoglobin (HbA_{1C}), superoxide dismutase (SOD), glutathione peroxide (GPx) and serum thiobarbituric acid reactive substance (TBARS).

RESULTS AND DISCUSSION:

Emissions from coal-fired thermal power plants and particulate matter emanating from coal mining areas are responsible for a range of illnesses worldwide. Several clinical studies and case studies have been carried out globally to document the ill-effects of air pollution and pollution caused by coal mining and coal power plants. There are comprehensive studies on adverse health effects of environmental pollution caused by coal in countries like USA, Australia and few European countries. Reports from India are scarce and hence this study was aimed at investigating the effect of exposure to coal mine dust in miners working in a coal-fired power plant at Neyveli, Tamil Nadu, India.

Despite the availability of sufficient evidence for establishing the association between air pollution and the onset of type 2 diabetes mellitus, the reports on effect of coal dust pollution on the plasma glucose levels in humans are very few. A total of 240 participants were included in this study, out of which 120 served as controls and the rest were coal miners. It was hypothesised the the exposure period to coal mine dust may influence the glycemic status of an individual and miners who were exposed to coal mine dust for 10 and 20 years were considered for this study.

The levels of FPG, PPG and HbA_{1C} of the miners were compared to those of the controls. The controls were residents residing in Neyveli and were not directly exposed to coal mine dust. The observations are tabulated in Tables 2 a-d.

Table 2a. Biochemical and antioxidant markers in male population of age 36 – 45 years N = 60

S. No	Parameters	Control Group	Study Group[a,b]	
			10 Years*	20 years*
1	FPG (mg/dL)	97.4 ± 1.24	107.6 ± 2.41	114.5 ± 3.14
2	PPPG (mg/dL)	122.7 ± 1.09	138.7 ± 4.52	168.97 ± 5.64
3	HBA1C (%)	5.80 ± 0.08	6.1 ± 0.11	6.67 ± 0.09
4	SOD (IU/L)	197.52 ± 3.6	187.97 ± 4.17	188.72 ± 6.32
5	GPx (IU/L)	8867.5 ± 123.8	7538.07 ± 106.78	7441.75 ± 201.89
6	TBARS (nmol/ml)	3.02 ± 0. 08	4.16 ± 0.92	5.23 ± 1.2

* number of years of exposure to coal mine dust

[a] – the results are statistically significant when compared to the control group ($p < 0.001$)

[b] – the results are statistically significant when compared between the study groups ($p < 0.001$)

Table 2b. Biochemical and antioxidant markers in male population of age 46 – 55 years N = 60

S. No	Parameters	Control Group	Study Group[a,b]	
			10 Years*	20 years*
1	FPG (mg/dL)	95.37 ± 2.31	98.6 ± 1.78	110.3 ± 3.42
2	PPPG (mg/dL)	126.47 ± 3.68	127.2 ± 4.11	156.4 ± 3.68
3	HBA1C (%)	5.68 ± 0.09	6.2 ± 0.12	7.41 ± 0.09
4	SOD (IU/L)	196.2 ± 3.48	187.65 ± 3.87	180.2 ± 4.56
5	GPx (IU/L)	8527.5 ± 121.5	7328.47 ± 98.3	7201.75 ± 101.6
6	TBARS (nmol/ml)	3.13 ± 0.09	4.42 ± 0.82	5.53 ± 0.56

* number of years of exposure to coal mine dust

[a] – the results are statistically significant when compared to the control group ($p < 0.05$)

[b] – the results are statistically significant when compared between the study groups ($p < 0.001$)

Table 2c. Biochemical and antioxidant markers in female population of age 36 – 45 years N = 60

S.No	Parameters	Control Group	Study Group[a,b]	
			10 Years*	20 years*
1	FPG (mg/dL)	92.6 ± 2.12	100.9 ± 4.35	111.5 ± 4.32
2	PPPG (mg/dL)	124.9 ± 3.78	124.3 ± 4.52	130.8 ± 3.12
3	HBA1C (%)	5.4 ± 0.06	6.1 ± 0.08	6.5 ± 0.07
4	SOD (IU/L)	187.73 ± 4.36	180.97 ± 3.76	178.72 ± 4.13
5	GPx (IU/L)	8396.7 ± 109.8	7253.8 ± 126.5	7141.75 ± 114.8
6	TBARS (nmol/ml)	3.09 ± 0.76	4.21 ± 0.83	5.07 ± 0.94

* number of years of exposure to coal mine dust

[a] – the results are statistically significant when compared to the control group ($p < 0.001$)

[b] – the results are statistically significant when compared between the study groups ($p < 0.0001$)

Table 2d. Biochemical and antioxidant markers in female population of age 46 – 55 years N = 60

S. No	Parameters	Control Group	Study Group[a,b]	
			10 Years*	20 years*
1	FPG (mg/dL)	95.4 ± 3.48	108.7 ± 2.09	116.7 ± 3.12
2	PPPG (mg/dL)	128.7 ± 3.23	127.5 ± 2.40	140.7 ± 3.59
3	HBA1C (%)	5.8 ± 0.06	6.1 ± 0.03	6.7 ± 0.02
4	SOD (IU/L)	191.12 ± 3.67	182.38 ± 4.79	181.7 ± 5.72
5	GPx (IU/L)	8178.75 ± 86.4	7629.0 ± 99.5	7175.75 ± 89.9
6	TBARS (nmol/ml)	3.45 ± 1.09	4.67 ± 0.96	5.82 ± 0.94

* number of years of exposure to coal mine dust

[a] – the results are statistically significant when compared to the control group ($p < 0.001$)

[b] – the results are statistically significant when compared between the study groups ($p < 0.001$)

The FPG levels in the study groups and the control groups were within the normal range. The PPG levels of the male miners who were exposed for 20 years to coal mine dust were found to be significantly high ($p < 0.001$). The male miners who were exposed for 10 years to coal mine dust and the female miners of both 10 and 20 years of exposure period remained unaffected. Their PPG levels were within the normal range, the difference between the miner group and the control group was significant though ($p < 0.001$).

Studies confirm that air pollutants can contribute to decreased insulin sensitivity and increased incidence of hyperglycemia. It is suggested that, air particulate matter can cause irritation to the human system, thereby triggering localized inflammation. The body responds to inflammation via the activation of the immune system and the autonomic nervous system. The hormones swing into action and alter the metabolic pathways, predominantly the glucose homeostasis and lipid metabolism. The constant stress exerted on the body results in altered functions of the pancreas[9]. Exposure to particulate matter even for a short duration of 58 days was found to be associated with low insulin sensitivity and high fasting glucose levels. The effect of air pollution was worse in obese study participants and it affected glucose tolerance[10].

Brook, 2005[11] proposed that pulmonary inflammation and oxidative stress may be the contributing factors for the incidence of Type 2 diabetes mellitus in population exposed to pollution. Hence the level of enzymic antioxidants SOD and GPX and serum TBARS were studied to assess the extent of oxidative stress in miners (Table 2 a-d). The level of SOD and GPX were significantly low in the miners, irrespective of the genders. The serum TBARS were significantly high in all the groups except the control groups. These observations imply that the enzymic antioxidants were utilized to scavenge the reactive oxygen species generated by the oxidative stress caused by coal dust pollution. Exposure to organic pollutants was reported to

be a pathogenic precursor of type 2 diabetes[12].

Coal-fired thermal power plants contribute to serious pollution and emit PM which contain sulphates, nitrates and metal salts. These PM damage various systems of the body by instigating continuous production of reactive oxygen species[13,14]. The results of our study were in agreement with previous reports on coal miners [15-17].

CONCLUSION:

The study was conducted to investigate the impact of exposure to coal mine dust in miners for 10 years and 20 years. The observations of the study revealed that the exposure to coal mine dust for a period of 20 years predisposes the miners to Type 2 diabetes. The contributing factor for this predisposition was found to be oxidative stress which was indicated by the level of serum antioxidants and serum TBARS in the study groups.

CONFLICT OF INTEREST:

None.

REFERENCES:

1. Fan Y, Jian-Jun Huang, Chen-Ming Sun, Nan Qiao, Hai-Xia Zhang, Hui Wang et al. Prevalence of dyslipidaemia and risk factors in Chinese coal miners: a cross-sectional survey study. Lipids in Health and Disease. 2017; 16:161
2. Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C and Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology. 2005; 16:25–32.
3. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation. 2010; 121: 2331–2378.
4. Kim JW, Park S, Lim CW, Lee K and Kim B. The Role of Air Pollutants in Initiating Liver Disease. Toxicology Research. 2014; 30(2): 65-70
5. www.urbanemissions.info
6. Sanchez L and Luan B. The Cost of Coal in Indonesia. www.iisd.org/gsi. 2018.
7. Huang Xl, Li W, Attfield MD, Nadas A, Frenkel K and Finkelman RB. Mapping and Prediction of Coal Workers' Pneumoconiosis with Bioavailable Iron Content in the Bituminous Coals, Environmental Health Perspective. 2005; 113(8): 964-968
8. Atabay MM. The Cytotoxic and Haematological Effect of Coal Dust to Underground Miners. American Journal of Biochemistry. 2011; 1(1): 4-8
9. Carpenter DO. Environmental contaminants as risk factors for developing diabetes. Reviews in Environment Health. 2008; 23: 59–74.
10. Chen Z, Muhammad T. Salam, Claudia Toledo-Corral, RichardM.Watanabe, Anny H. Xiang, Thomas A. Buchanan et al. Ambient Air Pollutants Have Adverse Effects on Insulin and Glucose Homeostasis in Mexican Americans. Diabetes Care. 2016; 39: 547–554
11. Brook RD. You are what you breathe: evidence linking air pollution and blood pressure. Current Hypertension Report. 2005; 7(6): 427–434.
12. Lee DH, In-Kyu Lee, Soo-Hee Jin, Michael Steffes, David R and Jacobs JR. Association Between Serum Concentrations of Persistent Organic Pollutants and Insulin Resistance Among Nondiabetic Adults. Diabetes Care. 2007; 30: 622–628
13. Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, et al. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 2006; 295: 1127–1134.
14. Zhou Q, Huang GH and Chan CW. Development of an intelligent decision support system for air pollution control at coal-fired power units. Expert in System Applications. 2004; 26: 335–356.
15. Kaur A, Gill MS, Gupta K and Manchanda KC. Effect of occupation on lipid peroxidation and antioxidant status in coalfired thermal plant workers. International Journal of Applied and Basic Medical Research. 2013; 3(2): 93–97.
16. Fubini B and Hubbard A. Reactive oxygen species (ROS) and reactive nitrogen species (RNS) generation by silica in inflammation and fibrosis. Free Radical Biology and Medicine. 2003; 34: 1507–1516.
17. Pinho RA, Bonatto F, Andrades M, Frota ML, Jr, Ritter C, Klamt F, et al. Lung oxidative response after acute coal dust exposure. Environment Research. 2004; 96: 290–297