

**Lung function decline: A cohort study in a population exposed to coal dust**Minakshi Bhattacharjee<sup>1</sup>, Unni.B.G<sup>1</sup>, S. Das<sup>1</sup>, M. Deka<sup>2</sup>, Wann.S.B<sup>1</sup>

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**ABSTRACT**

Chronic Obstructive Pulmonary Disease (COPD), is characterized by coughing, shortness of breath, sputum production, rapid breathing, wheezing, and weight loss due to the energy required for labored breathing. COPD includes chronic bronchitis and emphysema chronic bronchitis - inflammation of the lining of the bronchial tubes emphysema - permanent destruction of the alveoli. Chronic respiratory diseases have a pre-eminent role in the health conditions of people residing near coalmine areas with implications for morbidity and excess mortality from specific causes. Atmospheric pollution from anthropogenic sources such as coal mining, industrial sources is a serious worldwide concern as it is associated with adverse health effects. The aim of this study was to find a correlation between coal dust and reductions in lung function. An environmental health survey was conducted in and around the surrounding areas of opencast coal mine, Tirap to determine the prevalence of various respiratory symptoms and to assess pulmonary function among the villagers. Although cigarette smoking is the main environmental risk factor, only about 15% of smokers develop clinically significant disease suggesting other influences on disease expression. We screened 412 individuals during surveys at the coal mine site, Assam to find out possible occurrences of COPD and associated environmental risk factors, mainly coal dust exposure. Lung function test was done with a portable spirometer. COPD was diagnosed on the basis ATS guidelines. The subjects were categorized into COPD (Smokers and Non-Smokers) and Non-COPD (Smokers and Non-Smokers). Air quality was monitored using Respirable Dust Sampler and analyzed for Respirable Suspended Particulate Matter (RSPM), SO<sub>2</sub> and NO<sub>2</sub>. Coal dust exposure was a potential factor in development of COPD.

**Key words:** COPD, lung function decline, RSPM, Coal Dust, air pollution.

**1. Introduction**

A widely accepted definition from (Global Initiative for Obstructive Lung Disease) GOLD defines (Chronic Obstructive Pulmonary disease) COPD as “a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases” (Pauwels et al, 2001). Chronic Obstructive Pulmonary Disease (COPD) is one of the leading causes of mortality. By 2020 it is expected to rise to the third position as a cause of death and at fifth position as the cause of disability adjusted life years (DALYs) as per projections made in Global Burden of Disease study (GBDS)(Jindal et al,2006). A summary of field studies from some states of India on prevalence of COPD (published in last three decades) shows prevalence in the range of 5-12.5% in males and 3.2-4.5% in females (Jindal et al,2006).Hardly, any information on the disease is available from the rest of India.

COPD is the consequence of an abnormal inflammatory response to inhalation of noxious agents such as cigarette smoking, occupational exposure and environmental factors. However, individual factors also play an important role (e.g., enzymatic deficiencies, immunological trouble) and in fact only a portion (10-20%) of heavy smokers develop a clinically detectable disease (Hogg et al,2004; Piqueras et al, 2001) . The relative prevalence and severity of mining related occupational lung diseases are a function of the commodities mined, airborne hazard exposure levels, and co-existing illnesses or environmental conditions and lifestyle. The contamination of atmosphere from anthropogenic sources such as coal mining, industrial sources as well as local conditions generated either in home or workplace make a significant contribution as environmental factors, to development of chronic airflow obstruction. The coal-based industries in India are considered to be one of the chief industrial emitters in India. North East part of India has coals with different physicochemical properties compared to other Indian coals (Khare et al, 2011). They have high sulphur and volatile matter with low ash and moisture contents. The present study was undertaken to assess the status of lung function in individuals residing very near to the open-cast coal mine at Ledo, Assam and also to find out whether coal dust exposure has any effect on lung function status.

## **2. Materials & Methods**

### **2.1 Survey, and Lung Function Test at the study site**

Survey was conducted in the vicinity of the open-cast coal mine area at Ledo, near Tirap in Assam (Lat. 27°13'-27°23'N and Long. 95°35'-96°00E); with the help of doctors at the local Primary Health Centre during the period. January 2009 to December 2010. The coal mine has been there since 1870 (Akala et al,1995) and most of the people living in the vicinity have been depending on it for their livelihood. These people are likely to be affected by the coal dust exposure resulting into several lung diseases. Our statistical population was these people who have been living there for 11 to 35 years within this 1.5km of the mine area. However, duration of living of different families was different and thus the exposure to the pollutants was also different. We drew 412 people randomly as the sample for our study irrespective of age, sex, and livelihood. Prior to the study it was not possible to detect people having COPD and Non-COPD , hence the fractions of the sample were not of equal size. Moreover, our sample also depended on the willingness of the people for demographic data, spirometry for analyses. Lung function test (spirometry) was conducted with Spirometer (Model Schiller, USA) for each subject and the data FEV1/FVC (Forced Expiratory Volume in one Second/Forced Vital Capacity) were recorded. At the baseline, pre-bronchodialator spirometry was done for all the subjects. Then we tested for bronchodialator reversibility where each individual was first administered with 4 puffs of Metered Dose Inhaler (100µg/puff) of Salbutamol. They were allowed to rest for 15 mins. and post-bronchodialator spirometry was performed. Irreversible airflow limitation was confirmed when FEV1 was less than 12%.These persons were included in symptomatic COPD group. Chronic Airflow Obstruction was diagnosed on the basis of medical history of exposure to coal dust, physical examination and spirometry data, according to ATS guidelines(ATS,1995). Later on, we categorized the sample into different fractions of male-female, age, smoking status etc. Data were recorded in Questionnaire formats for each individual willing to participate in the survey. Questions regarding smoking habits, age, gender, occupation, environment etc., were included in the questionnaire. The study was carried out at CSIR(NEIST), Jorhat after ethical clearance from Institutional Ethics Committee NEIST, Jorhat.The authors declare that they have no conflicts of interest.

## **2.2 Air Analysis**

Air quality was assessed simultaneously during each survey at different locations near the site during the period January 2009 to December 2010. Sampling of air was done with Respirable Dust Sampler (Envirotech Model APM 460 BL) and data were calculated for Respirable suspended particulate matter (RSPM). Determination of SO<sub>2</sub> and NO<sub>2</sub> in ambient air was done by modified West and Gaeke Method and Sodium Arsenite Method respectively, (as per manufacturer's instructions by Central Pollution Control Board). The data were categorized into different periods as December-March, (winter) April-July, (summer) and August-November (autumn) to observe for seasonal variation in the parameters throughout the years.

## **2.3 Statistical Analyses**

Data were tabulated and classified as per the study variables mentioned in 'Material & Methods'. Significance of variations in RSPM, SO<sub>2</sub> and NO<sub>2</sub> among seasonal periods i.e. Apr-July, Aug.-Nov., and Dec.-March was tested by applying Kruskal-Wallis test (K), a distribution free test. Chi-square test with Yates correction was applied to test significant difference in the number between smoker and non-smokers, males and females amongst symptomatic and asymptomatic subjects. Unpaired 't' test was applied to test for significant difference in continuous variables and X<sup>2</sup> test was used to test the significance in categorical variables.

## **3. Results**

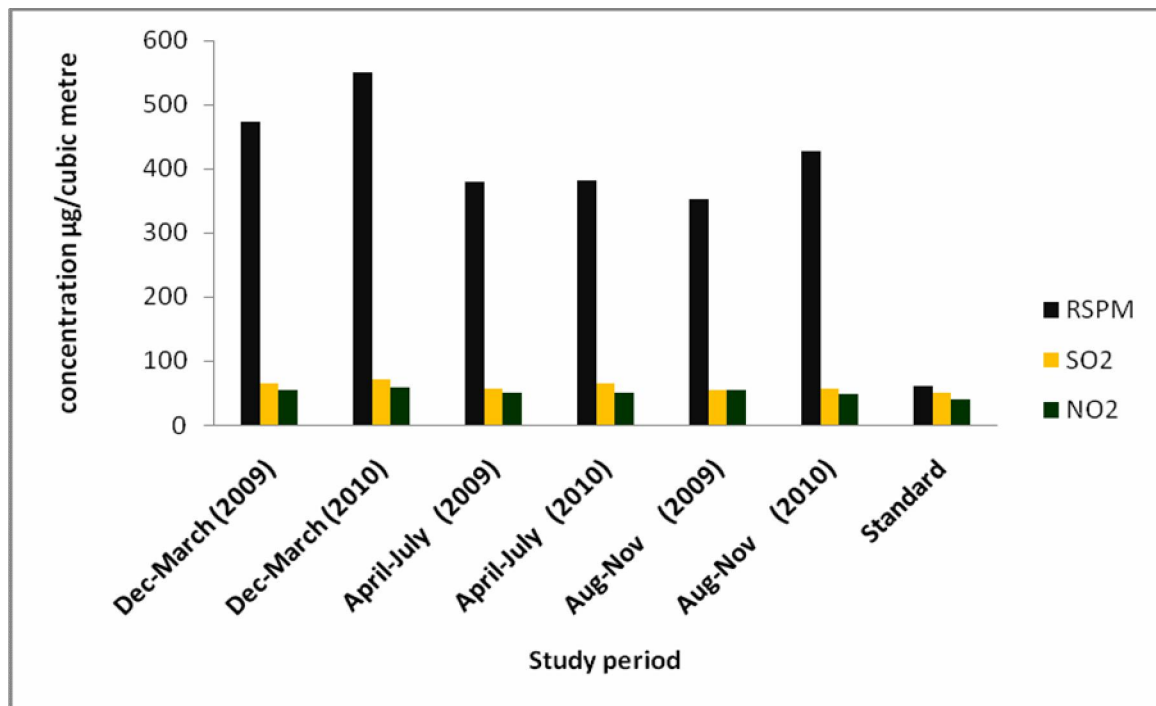
The values for Respirable suspended particulate matter (RSPM), SO<sub>2</sub> and NO<sub>2</sub> were found to be higher in the coal mine site; as shown in Fig.1.compared to standards (Central Pollution Control Board). Even the annual average values for RSPM SO<sub>2</sub>, NO<sub>2</sub> were higher than standards during both the years 2009 and 2010. The highest values for RSPM, SO<sub>2</sub> and NO<sub>2</sub> were observed during the period (Dec-March), followed by (Apr-July) and (August – Nov). Significant seasonal variation was observed for RSPM (\*K=13.02,df=2,p<0.01), SO<sub>2</sub> (\*K=23.1,df=2,p<0.01), during 2009 but there was no significant variation in levels of NO<sub>2</sub> (\*K=5.11,df=2,p>0.01).During 2010 also ,the RSPM (\*K=6.93,df=2,p<0.01), SO<sub>2</sub> (\*K=10.59,df=2,p<0.01) and NO<sub>2</sub> (\*K=12.87,df=2,p<0.01) varied significantly.[ \*K = Kruskal Wallis analysis of variance].

### **3.1 Lung Function, Smoking Status and sorting out of the subjects**

The symptomatic smokers (n = 92) and non-smokers in the (n = 194) coal mine showed obstructive pattern of lung function in both males and females. In case of asymptomatic non-smokers and asymptomatic smokers in both the areas, no lung function decline was observed in either males or females. Detailed observations on demographic variables are shown in Table 1.

In the coal mine site, amongst COPD non-smokers (n=194) and Non-COPD non-smokers (n=84), significantly, more number of symptomatic males [ $\chi^2 = 7.08$ ,  $p = 0.007$ , OR = 0.39 (0.18 – 0.83)] and symptomatic females [ $\chi^2 = 7.08$ ,  $p = 0.007$ , OR = 0.39 (0.18 – 0.83)] was recorded. Amongst COPD smokers (n = 92) and Non-COPD smokers (n = 42) the difference was not significant. No significant difference was observed in smoking pack years between COPDs and Non-COPDs ( $t = 0.21$ ,  $df = 132$ ,  $p = 0.8$ ). A significant difference was observed

in lung function amongst COPD smokers versus Non-COPD smokers [ $t = 27.17$ ,  $df = 132$ ,  $p = 0.006$ ]; and COPD Non-Smokers versus Non-COPD non smokers [ $t = 34.53$ ,  $df = 276$ ,  $p = 0.003$ ]. The coal dust exposure years also significantly differed amongst COPD smokers versus Non-COPD smokers [ $t = 7.03$ ,  $df = 132$ ,  $p = 0.009$ ]; and COPD Non-Smokers versus Non-COPD non smokers [ $t = 9.71$ ,  $df = 276$ ,  $p = 0.02$ ]. The bronchodilator data showed  $FEV_1 < 12\%$  in the COPDs, confirming airflow obstruction. Thus, the subjects were sorted as COPDs and Non-COPDs based on their  $FEV_1/FVC$  ratio and post bronchodilator spirometry. The age was not significantly different amongst COPD smokers versus Non-COPD smokers [ $t = 0.28$ ,  $df = 132$ ,  $p = 0.77$ ]; and COPD Non-Smokers versus Non-COPD non smokers [ $t = 0.796$ ,  $df = 276$ ,  $p = 0.42$ ].



**Figure 1:** Levels of RSPM, SO2 and NO2 in the open cast coal mine site during 2009-2010.

Table 1: Demographic and Lung Function observations in the subjects participating in our study at Open cast coalmine site (Ledo, Assam)

Variables	STUDY POPULATION (COAL MINE AREA)			
	COPD SMOKERS (N = 92)	COPD NON-SMOKERS (N=194)	NON-COPD NON-SMOKERS (N= 84)	NON-COPD SMOKERS (N= 42)
aMale	85	140	73	38
bFemale	7	54	11	4
Sex Ratio (F/M)	0.08	0.38	0.15	0.10

cSmoking (Pack Years ± S.D)	15.91± 7.62			16.21 ± 7.31
dCoal Dust Exposure (Years ± S.D)	30.40 ± 13.76	30.03 ± 13.16	15.17 ± 7.2	15.16 ± 3.83
eMean Age (± S.D)	40.5 ± 10.58	35.53 ± 13.14	34.17 ± 12.84	41.0 ± 11.99
fFEV1/FVC (% predicted) (± S.D)	53.15 ± 8.93	56.68 ± 9.56	112.75 ± 16.97	107.78 ± 15.11
gPost Bronchodilator FEV1 (liters)	0.87 ± 0.09	0.85 ± 0.13		

a COPD Smokers vs Non COPD Smokers :X<sup>2</sup> =0.14 , p =0.70 , OR = 0.29 (1.28-5.27)

a COPD Non Smokers vs Non COPD Non Smokers\* : X<sup>2</sup> =7.08, p =0.007, OR =0.39 (0.18 – 0.83)

b COPD Smokers vs Non COPD Smokers: X<sup>2</sup> =0.14 , p =0.70 , OR =0.29 (1.28-5.27)

b COPD Non Smokers vs Non COPD Non Smokers\* : X<sup>2</sup> =7.08, p =0.007 , OR =0.39 (0.18 – 0.83)

c COPD Smokers vs Non COPD Smokers : t = 0.21, df=132, p = 0.8

d COPD Smokers vs Non COPD Smokers\* :t =7.03 , df=132 , p =0.009

d COPD Non Smokers vs Non COPD Non Smokers\*: t = 9.71, df=276 , p = 0.02

e COPD Smokers vs Non COPD Smokers :t =0.28, df=132 , p =0.77

e COPD Non Smokers vs Non COPD Non Smokers: t = 0.796, df= 276, p = 0.42

f COPD Smokers vs Non COPD Smokers\* :t =27.17, df=132 , p =0.006

f COPD Non Smokers vs Non COPD Non Smokers\*: t = 34.53, df= 276, p = 0.003

\*Statistically significant

#### 4. Discussion

Air analysis (done in three different periods during 2009 and 2010) showed highest RSPM, SO<sub>2</sub> and NO<sub>2</sub> during Dec-March and lowest during August-Nov. Despite seasonal variation, the levels of all these parameters were higher than standard limits in all seasons in both the

years. The overall annual data reveals that our study site is considerably polluted and the population in the area is exposed to recurrent episodes of acute air pollution. As our study was specifically focussed on the residential areas very near to the coal mine areas of Ledo, India, undoubtedly, respirable mixed coal dust was a significant contributor to the suspended particulate matter. Moreover, we also found that the COPDs were exposed for a significantly longer duration to coal dust than the Non-COPDs ( $p < 0.05$ ) in both smokers and non-smokers. Several longitudinal, epidemiological and associative studies have established that acute episodes of atmospheric pollution causes increased risk of adverse pulmonary events (Dockery et al, 1993; Laden et al, 2006). Significantly, more number of symptomatic i.e. people with COPD was recorded than asymptomatic i.e. people without COPD in our study site ( $p < 0.01$ ). Amongst other known risk factors of COPD, the genetic deficiency of Alpha 1 Antitrypsin (A1AT) attributed to ZZ type is the best documented reasons (Carp et al 1978). Phenotype M is the normal variant phenotypes S and Z are the two most frequent abnormal variants (Hutchinson et al, 1998). Calculated values of PiZZ prevalence are approximately: 1:1000—1:45,000 in Western and Northern Europe, 1:45,000 – 1:10,000 in Central Europe; and 1: 10,000 – 1:90,000 in Eastern Europe and in southernmost and northern areas of the continent. In the White population of USA, Canada, New Zealand, PiZZ phenotype prevalence ranges from 1: 2000 – 1:7000 individuals (Andolfalto et al, 2003).

Our previous findings suggests that A1AT deficiency is not prevalent in our population subset and all the subjects were having the normal MM type of Alpha 1 Antitrypsin gene (Unni et al, 2011). As already documented, the ZZ allele is rarely present in most of the populations, the reasons for occurrence of COPD in our study subset could be attributed to other factors such as coal dust exposure. The data of prevalence of COPD in Asian countries is patchy and the disease burden is high. Studies on COPD in the Indian population is very limited. There are very less data on genetic epidemiologic studies of AAT deficiency in countries like India; and thus it is essential to ascertain whether populations are at risk or not. We did not find any significant variation in age and smoking status amongst the subjects (COPDs and Non-COPDs). There are reports that only 10-20% of the smokers develop COPD suggesting the involvement of other factors (Hogg et al, 2004; Piqueras et al, 2001). Exposure to air pollutants leads to a variety of health effects depending on the type of pollutant, amount of the pollutant exposed to, duration and frequency of exposure, and associated toxicity of the specific pollutant (Lokuge Y. et al. 2010). The health effects caused by air pollutants may range from subtle biochemical and physiological changes to difficulty in breathing, wheezing, coughing and aggravation of existing respiratory condition.

Coal dust exposure seemed to be a potential factor in disease development and progression. Moreover, COPD is a complex polygenic disease and ethnic differences exist (Mehrotra et al, 2010). It is also possible that certain other genes might play a role and these genes need to be studied to find out whether individual susceptibility due to genetic factors or external risk factors such as smoking, occupational exposure etc. are responsible for the disease.

### **Acknowledgement**

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## **List of Abbreviations**

COPD: Chronic Obstructive Pulmonary Disease

RSPM: Respirable Suspended Particle Matter

GOLD: Global Initiative for Obstructive Lung Disease.

DALY(s): Disability Adjusted Life Year(s)

GBDS: Global Burden of Disease Study.

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