

Reproductive Health Hazards of Coal mine male Workers in Lakhanpur Open cast mines

Bidut Prava Mohanty and Malaya Ranjan Mahananda *

Department of Environmental Sciences, Sambalpur University, Jyoti-Vihar, Odisha, India, pin-768019

***Correspondence Info:**

Malaya Ranjan Mahananda

Department of Environmental Sciences

Sambalpur University, Jyoti-Vihar, Odisha, India, pin-768019

Phone: +91 9439536915

Fax: +91 663 2431033

E-mail: malaya_env@rediffmail.com

Abstract

The present study shows the impact of coal dust on reproductive health hazards of coal mine male workers in Lakhanpur open cast mines. The study was conducted on the reproductive health status of the male workers working more than ten years in coal mines and residing in same area. Twenty coal mine workers within the age group of 30-45 and a mean age 38.65 ± 0.93 year were included. Thirty five Control subjects with mean age 32.57 ± 0.61 year were selected randomly from the Burla and Jyotivihar area, 100 km away from the study area, those who are not exposed to coal dust. To observe the reproductive hazards Semen profile was undertaken and the total volume (ml in coal exposed group (above 10 years exposure) was found to be $2.08 \pm 0.04^{***}$. Seminal viscosity (cm of semen thread) was found to be $1.64 \pm 0.18^{***}$ and sperm count ($10^6/\text{ml}$) was found to be $40.5 \pm 0.67^{***}$ in coal exposed group (n=20) (above 10 years exposure). $33.38 \pm 0.93^{***}$ of immotile (%) was found in coal exposed group (n=20) above 10 years exposure.

Keywords: Toxicity, Reproductive health hazards, heavy metals.

1. Introduction

Heavy metals are metallic elements which have a high atomic weight and a density much greater (at least 5 times) than water. There are more than 20 heavy metals, but four are of particular concern to human health: lead (Pb), cadmium (Cd), mercury (Hg), and inorganic arsenic (As). They are highly toxic and can cause damaging effects even at very low concentrations. They tend to accumulate in the food chain and in the body and can be stored in soft (e.g., kidney) and hard tissues (e.g., bone). The body has need for approximately 70 friendly trace element heavy metals, but there are another 12 poisonous heavy metals, such as Lead, Mercury, Aluminum, Arsenic, Cadmium, Nickel, etc., that act as poisonous interference to the enzyme systems and metabolism of the body. As such, they are stable elements (meaning they cannot be metabolized by the body) and *bio-accumulative* (passed up the food chain to humans).

The actual causes of increased infertility remain controversial [1]-[3], but research suggests that many substances to which men are exposed and then can affect their fertility may be work-related [4]. The international technological shift in industrial and agricultural development in the 20th century involved handling and exposure to various substances that are harmful to humans, some of which affecting the male reproductive system, such as pesticides, metals, estrogen-like substances, chlorinated compounds, etc. [5][6]. With the above background the aim of the present investigation was carried out to determine the effect of occupational exposure of coal dust on semen quality of the subjects/workers working in open cast mine at Lakhanpur coal mine Project Odisha India.

2. Material and Methods

2.1 Study Site

This prospective study was conducted on the reproductive health status of the male workers working more than ten years in coal mines and residing in same area. The study was undertaken in Lakhanpur Open cast Project, which is the biggest (area wise) coal mines out of seven coal mines in western Odisha. The control subjects were chosen from Jyotivihar, Burla area who are not exposed to the coal dust. Field study was undertaken in the coal mines area, sample collected and processed and then all investigations were carried out at our Departmental Laboratory.

2.2 Subjects

Twenty coal mine workers within the age group of 30-45 and a mean age 38.65 ± 0.93 year were included after informed consent signed. The aim and objectives of the study was explained to each volunteers. Those who agreed to the objectives of the study they were enrolled to the project and

signed written consent were obtained. Each subject was interviewed and a standard proforma was filled up about their general information, working habit, life style factors, medical and reproductive history. Men with any chronic disease, under long term medication, regular alcohol drinking and obesity were excluded from the study. Subjects above the age of 50 year were not included in the study.

Thirty five Control subjects with mean age 32.57 ± 0.61 year were selected randomly from the Burla and Jyotivihar area, 100 km away from the study area, those who are not exposed to coal dust. A signed consent was obtained from each subject and standard proforma was filled up by personnel interview. Subject with proven fertility and without chronic diseases were included in this study. All the semen parameters of the control subjects were within normal range according to WHO criteria. Volunteer's attening over 50 year age, under medication and vasectomized males were excluded in this study.

2.3 Collection of Samples

2.3.1 Semen Sample collection and analysis

After an abstinence of 3 days, semen samples were collected from the volunteers in wide mouthed sterile containers by masturbation. The time and date of sample collection, days of abstinence was recorded in the proforma. The sample was allowed to liquefy at room temperature prior to analysis. Standard measurements of sperm concentration, total sperm count, vitality, motility, viscosity, pH was done immediately after liquefaction according to WHO recommended guidelines. A semen smear was done and allowed to air dry for further morphology analysis. The rest of the semen samples were centrifuged, aliquoted and stored at -20°C for further metal analysis and oxidative stress parameters. All the semen analysis was performed according to the WHO guidelines [7].

For assessing the sperm motility the microscope field was examined systematically and motility of each spermatozoa classified as rapid progression, slow progression, non progressive and immotile.

For studying the sperm morphology a drop of liquefied semen was smeared on a clean glass slide. The smear was then air dried and fixed in ethanol. The slides were then stained in 2% Giemsa for 30min. Dried stained mounted slide were scanned under microscope at 100X objective to asses for morphological abnormalities of sperm head, mid piece and tail [8]. A total of 200 sperms per sample were classified according to their morphology such as small, pin, tapered, large amorphous, double head, dilated or bent mid piece, coiled or short thick tail.

3. Result

3.1 Analysis of subject as per questionnaire

Table 1 showed the distribution of subjects according to the questionnaire. Majority of the subjects working in the coal mines were between the ages of 30 to 38 year i.e within the active reproductive age. All the volunteers are married and fathered the child. Socioeconomic status of

the workers is not very good. The workers are exposed to coal throughout the day as they are living that area and having food from nearby market and the drinking water they are using from pond or bore well of that area. The duration of exposure ranges between 10-25 years of these study subjects. There were no reproductive diseases/disorders among the workers as well as control population, but addiction of smoking, ghutka, tobacco, alcohol (mainly country liquor) etc were predominant among the workers in comparison with the matched non occupationally exposed control subjects.

3.2 General information about subjects

Twenty coal mine workers and 35 controls those who are not exposed to the coal dust are recruited for the study. There was no significant differences between the studied groups as regards age, weight, height, BMI (p values >0.05). The mean values are presented in Table 2.

3.3 Effect of coal on semen parameters of the workers working in the mines

Table 3 showing the semen parameters of both control and coal exposed workers. There is no significant difference in liquefaction time of control and coal exposed subjects. The mean seminal viscosity (mm of semen thread) of coal exposed group significantly declines from the control subject (2.14 ± 0.08 vs 1.64 ± 0.18). Similarly, the mean seminal volume significantly ($p < 0.001$) declines in coal exposed subjects. The mean pH value of both control and exposed subjects are within the normal range, but the mean value of exposed subjects significantly increased. Agglutination observed both in control and exposed subjects but found to be more in the coal exposed subjects. The mean sperm concentration, % of viability significantly decreased ($p < 0.001$) in the coal exposed subjects in comparison to the control. In motility, the mean value of rapid progression (%) and Immotile (%) significantly drops in comparison to control. The mean value of slow progression (%), whereas the non progressive (%) sperm increased in comparison to control subjects.

3.4 Effect of Coal on sperm morphology after occupational exposure

A very high percentage of morphologically abnormal sperm found in coal exposed group. The morphological abnormal sperm number increases with duration of the exposure to the coal. The mean number of normal spermatozoa significantly decreases in comparison to the control. Mean number of defective spermatozoa is significantly increased in the coal exposed subjects and 6.11 times more in comparison to the control. The mean number of head defect, mid piece defect, tail defects and total number of defects are significantly higher ($p < 0.001$) in the coal exposed subjects in comparison to control subjects. The teratozoospermic index significantly decreased ($p < 0.05$) in coal exposed subjects and sperm deformity index increased significantly ($p < 0.01$) in the coal exposed subjects. The data is presented as mean \pm SEM in Table 4.

Table 1: Demographic profile (%) of the subjects as per questionnaire data

Parameters	Control group (n = 35)	Coal exposed group (n=20) (above 10 years exposure)
Married and fathered child	100%	95%
Married not fathered any child	nil	nil
Unmarried	0%	5%
Reproductive diseases/disorder	Nil	Nil
Smoker	20%	19.0%
Tobacco/ghutka	Nil	38.46
Alcohol	12%	17%
Overduty	Nil	Nil
Sleeping habit	6-8h daily	6-8h daily
Under treatment	Nil	Nil
Exposure to coal dust	Nil	Direct

Table 2: General Information of control and coal exposed subjects

Parameters	Control group (n = 35)	Coal exposed group (n=20) (above 10 years exposure)
Age (year)	32.57± 0.61	38.65±0.93 ^{NS}
Weight(kg)	59.8±1.72	64.55±1.61 ^{NS}
Height(cm)	167.4±1.47	166.15±0.8 ^{NS}
BMI (kg/m ²)	21.35±0.51	21.97±0.37 ^{NS}

Data presented as Mean ± SEM. Data compared by students t-test. Control vs Expts. NS-Non significant in comparison to control. n = no of subjects.

Table 3: Semen profile of exposure group after occupational exposure to coal dust

Parameters	Control (n=35)	Coal exposed group (n=20) (above 10 years exposure)
Age (yr)	32.57±0.61	38.65±0.93
Liquification Time (min)	25.85±0.83	27.75±1.05
Volume (ml)	3.93±0.09	2.08±0.04
Seminal viscosity (cm of semen thread)	2.89±0.05	1.64±0.18
pH	7.5±0.02	7.62±0.03
Agglutination	11.2%	10.76%
Viability (%)	92.05±0.58	66.2±0.93
Sperm count (10 ⁶ /ml)	91.64±1.62	40.5±0.67
Motility		
Rapid progression (%)	62.34±1.28	31.65±2.10
Slow progression (%)	21.14±0.84	22.65±1.9
Non-Progression (%)	8.54±0.62	10.5±0.6
Immotile (%)	7.75±0.52	33.8±0.93

Data presented as Mean ± SEM. Data compared by students t-test. Control vs Expts. NS-non significant, *** indicate p<0.001 in comparison to control. N=no of subjects.

Table 4: Semen morphology of the subjects after occupational exposure to coal dust

Parameters	Control (n=35)	Coal exposed group (n=20) (above 10 years exposure)
No of spermatozoa counted	200	200
No. of normal spermatozoa	182.88±1.03	94.25±2.4 ^{***}
% of normal spermatozoa	91.44±0.51	47.12±1.2 ^{***}
No. of spermatozoa with defects	17.11±1.03	104.25±2.59 ^{***}
No. of head defects	10.88±0.72	63.95±2.79 ^{***}
No. of mid-piece defects	5.34±0.39	24.1±1.79 ^{***}
No. of tail defects	2.77±0.36	25.55±4.99 ^{***}
Total no. of defects	19±1.08	110±2.92 ^{***}
Teratozoospermic index	1.12±0.02	1.052±0.01 [*]
Sperm deformity index	0.09±0.03	0.55±0.01 ^{**}

Data presented as Mean ± SEM. Data compared p<0.05, ** indicate p<0.01, *** indicate p<0.001 in comparison to control. n = no of subjects.

4. Discussion

This study was designed to examine the hypotheses that coal dust may affect the semen quality and hypothalamic pituitary axis of the workers exposed for a longer period to coal dust. The present study demonstrated that all the control subjects and coal exposed (95%) subjects are married and fathered the child. All the study subjects are in active reproductive age and not under any medication. There are no difference between age, height, weight and BMI of control and coal exposed subjects.

The study finds a significant decline in semen volume, seminal viscosity without any significant difference between the ages in the subjects working more than 10 years in coal mines. The liquification time did not show any change between the control and coalmine workers. The pH, percent of viable sperm, sperm count decreases significantly in coal exposed subjects. The percentage of rapid progression, non progressive and immotile sperm declines significantly in coal exposed subjects except the slow progressive sperm. Chemicals could adversely affect male reproductive system by, either disrupting the gonadal endocrine axis [8][9]. In recent years it is evident from different studies that the sperm count is decreasing at the rate of 2% per annum for the last 20 years [10][11]. several factors like global warming and changes in life style, rising incidence of sexually transmitted diseases, increase use of drugs, exposure to occupationally hazardous chemicals, environmental pollutants, radiation all appear to have a deteriorative effect on male reproductive health. In this investigation, percentage of normal spermatozoa decreased significantly in coal exposed group. Number of spermatozoa with defects increased highly in coal exposed group as compared to control. Number of defects like head defect, tail defect, mid piece defect and total defect increased. The coal mine workers are exposed to coal dust in their work place and living near to the mines area, drinking the waters from the water bodies there and eating the vegetables from nearby market. According to the review of Ernst and Bonde (1992)[24] low level exposure to chromium through nasal route from welding fumes may not be a major hazard affecting spermatogenesis. However, epidemiological studies from China [13] and India, Danadevi *et al*[14] reported poor semen quality in men who were exposed to hexavalent chromium in the chrome-plating and welding industries. However, occupational exposure to hexavalent chromium varies with intensity and the route, depending upon the nature of work. Decrease in sperm concentrations and motility as well as morphological alterations are frequent findings in semen analysis of the workers of different industries [15][16]. Occupational exposure to high temperature by welders, dyes, drivers, blast furnace workers impairs testicular function and leads to partial or complete spermatogenic arrest depending on period of exposure [10] [17]. Exposure for short periods causes reversible partial spermatogenic arrest leading to oligozoospermia whereas

continues long term exposure causes complete and irreversible spermatogenic arrest and results in azoospermia [17].

The level of heavy metal content like cadmium and lead in blood and semen of both control and coal mine workers are studied. The levels of both the heavy metals were significantly higher in coal exposed subjects. The percentage of smokers in control and coal mine workers are almost same (20% and 19% respectively), but the concentration of lead and cadmium significantly increased in coal mine workers, could be due to exposure to coal dust continuously, living 24 hr in same environment, drinking the water which is contaminated with coal dust. These heavy metals could be the factor for the decline in semen parameters and abnormal morphology like head defect, tail defect, mid piece defect in coal mine workers. Xuezi *et al* [18] in 1992 showed that lead exposure caused prolonged liquefaction, low volume; reduction in sperm count and velocity as well as retarded sperm activity along with high blood leads levels in the male workers. Moreover, semen lead was the indicator of industrial exposure [19] and moderate lead exposure caused reduction in sperm characteristics among the factory workers [20]. Exposure to metals (mainly lead and cadmium) has been long associated with low sperm motility and density, increased morphological anomalies and male infertility [21] [22]. Males employed in metal industries had a decreased fertility when compared with other workers as shown by a delayed pregnancy and reduced semen quality [23][24].

5. Conclusion

It can be hypothesized that the coal dust exposure might be responsible for high level of lead and cadmium in blood and semen of coal mine workers after a long term (10-25 years) exposure. The presence of lead and cadmium in both these biological fluids suggested they might cross blood-testis-barrier [25] at the present exposure level and subsequently produced detrimental effects on sperm structure, sperm parameters. The themes approached in the field of workers' health are important for any country's economic system. The issue of male infertility caused by occupational exposure is pertinent worldwide. Protection of workers against exposure may also allow preventing other alterations and injuries to the human body. In addition to the reproductive system, endocrine disruptors can interfere in other areas such as the immune and endocrine-metabolic systems and can even cause diseases like cancer. Progress is needed in the knowledge of possible effects of exposure on male fertility. Such progress will allow the development of preventive measure within the field of workers' health. Studies in this field should be encouraged, since they are scarce in light of the almost unlimited range of substances to which workers are potentially exposed.

Acknowledgement

The Authors are thankful to Indian Council of Medical Research (ICMR, New Delhi) for funding to carry out this research work.

References

- [1] Swan HS, Elkin EP, Fenster L. Have sperm densities declined? A reanalysis of global trend data. *Environ Health Perspect.* 1997; 105:1228-32.
- [2] Golden AL, Moline JM, Bar-Chama N. Male reproduction and environmental and occupational exposures: a review of epidemiologic methods. *Salud Pública Méx.* 1999; 41:93-105.
- [3] Multigner L, Oliva A. Secular variations in sperm quality: fact or science fiction? *Cad Saúde Pública.* 2002; 18:403-12.
- [4] Petrelli G, Musti M, Figa-Talamanca I. Exposure to pesticides in greenhouses and male fertility. *G Ital Med Lav Ergon.* 2000; 22:291-5.
- [5] Cox C. Masculinity at risk. *Journal of Pesticide Reform.* 1996; 16:2-7.
- [6] Cooper RL, Kavlock RJ. Endocrine disruptors and reproductive development: a weight-of evidence overview. *J Endocrinol.* 1997; 152:159-66.
- [7] WHO. Arsenic and Arsenic Compounds. *Environmental Health Criteria*, 2001 vol. 224. Geneva: World Health Organization.
- [8] Skakkebaek NE, Rajpert-De Meyts E, Main KM. Testicular dysgenesis syndrome: an increasingly common developmental disorder with environmental aspects. *Hum Reprod.* 2001; 16: 972-8.
- [9] Sharpe RM, Irvine DS. How strong is the evidence of a link between environmental chemicals and adverse effects on human reproductive health? *BMJ.* 2004; 328: 447-451.
- [10] Carlsen E, Giwercman A, Kieding N, Skakkebaek NE. Evidence of decreasing quality of semen during past 50 years. *Br. Med J.* 1992; 305: 609-615.
- [11] Sharpe RM. Declining sperm counts in men-is there an endocrine cause? *J Endocrinol.* 1993; 136(3): 357-360.
- [12] Ernst E, Bonde JP. Sex hormones and epididymal sperm parameters in rats following sub-chronic treatment with hexavalent chromium. *Hum Exp Toxicol.* 1992; 11: 255.
- [13] Li H, Chen Q, Li S, YAO W, Li L, Shi X, Wang L, Castranova V, Vallyathan V, Ernst E and Chen C. Effect of Cr (VI) exposure on sperm quality: Human and animal studies. *Ann Occup Hyg.* 2001; 45: 505-511.
- [14] Danadevi K, Rozati R, Reddy PP, Grover P. Semen quality of Indian welders occupationally exposed to nickel and chromium. *Reprod Toxicol.* 2003; 17: 451-456.
- [15] Said TM, Agarwal A, Sharma RK, Mascha E, Sikka SC and Thomas AJ. Human sperm superoxide anion generation and correlation with semen quality in patients with male infertility. *Fertil Steril.* 2004; 82: 871-877.
- [16] Agrawal A and Prabakaran SA. Oxidative stress and anti oxidants in male infertility: a difficult balance. *Iranian J Reprod Med.* 2005; 3: 1-18.
- [17] Dada R, Gupta NP, Kucheria K. Spermatogenic Arrest in Men with Testicular Hyperthermia. *Teratogenesis Carcinogenesis and Mutagenesis.* 2003;1(1), 235-243.
- [18] Xuezhij J, Youkin L, Yilan W. Studies of lead exposure on reproductive system: a review work in China. *Biomed Environ Sci.* 1992; 5:266-275.
- [19] Saaranen M, Suistomaa U, Kantola M, Saarikoski S, Vanha-Perttula T. Lead, magnesium, selenium and zinc in human seminal fluid: comparison with semen parameters and fertility. *Hum Reprod.* 1987; 2:475-479.
- [20] Cullen MR, Kayne RD, Robins JM. Endocrine and reproductive dysfunction in men associated with occupational inorganic lead intoxication. *Arch Environ Health.* 1984; 39:431-40.
- [21] Wagner U, Schlebusch H, Van der Ven H, Accumulation of pollutants in the genital tract of sterility patients. *J Clin Chem Clin Biochem.* 1990;28: 683-88.
- [22] Umeyama T, Ishikawa H, Takeshima H, A. comparative study of seminal plasma trace elements in fertile and infertile men. *Fertil Steril.* 1986; 46: 494-499.
- [23] Robins TG, Bornman MS, Ehrlich RI. Semen quality and fertility of men employed in a South African lead acid battery plant. *Am J Ind Med* 1997; 32: 369-76.
- [24] Bonde P. The risk of male subfecundity attributable to welding of metals. Studies of semen quality, infertility, fertility, adverse pregnancy outcome and childhood malignancy. *Int J Androl.* 1993; 16: Suppl 1, 1-29.
- [25] Marchlewicz J. Effectiveness of blood testes barrier and blood epididymal barrier for lead. *Ann Acad Med Stetin.* 1994; 40:37-51.
- [26] Biswas NM, Ghosh P. Effect of lead on male gonadal activity in albino rats. *KUMJ.* 2004; 2:43- 46.
- [27] Camoratto AM. Inhibition of rat pituitary growth hormone release by subclinical levels of lead. *Toxicologist.* 1990; 10:641.