Inorganic Lead Exposure in Battery and Paint Factory: Effect on Human Sperm Structure and Functional Activity

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Abstract: Lead is one of the industrially important heavy metals that causes male reproductive impairment among battery and paint factory workers, but information on the structure-function integrity of human spermatozoa is still limited. Therefore, it was necessary to investigate the effect of lead on sperm structure and functional activity in these workers. Oligozoospermia with concomitant lowering of sperm protein and nucleic acid content and the percentage of sperm DNA hyploidy ($P < 0.001$) suggested the diminution of sperm cell production after occupational lead exposure. Low sperm vitality and hypoosmotic swelling percentage along with high malondialdehyde content and altered seminal plasma ascorbate level ($P < 0.001$) indicating damage of sperm cell surface, might be due to high membrane lipid peroxidation and failure of non-enzymatic antioxidant protection after lead exposure. Alteration of sperm membrane surface was also evidenced from scanning electron microscopy and further authenticated by atomic and lateral force microscopy. Lowering of sperm velocity, gross and forward progressive motility with high stationary motile spermatozoa ($P < 0.001$) suggested retarded sperm activity among the exposed workers, which was supported by high seminal plasma fructose level and reduced activity of sperm ATPase ($P < 0.001$). Increased incidence of teratozoospermia was also associated with high blood and semen lead level (PbB, PbS) ($P < 0.001$). Therefore, the results suggested that lead not only affects the sperm count, but also damages the sperm structure and membrane integrity, motility and functional activity among the battery and paint factory workers.

Key words: sperm morphology, sperm motility, sperm membrane lipid peroxidation, blood/semen lead, lead exposed workers.

(Received 1 November 2005, accepted 10 April 2006)
Introduction

The diminution of semen quality due to occupational exposure to heavy metals is a major problem in the world [1–3]. Lead exposure and moderate lead absorption produced an alteration in fertility with decreased production in spermatozoa among battery factory workers, probably due to the direct toxic effect of lead [4–6]. Reduction in sperm velocity, forward progressive (FP) motility, density and count, low antioxidant profile, increased incident of sperm cell abnormality and membrane lipid peroxidation along with high PbB and PbS level was also prevalent after occupational exposure to lead [7, 8]. The action of seminal antioxidant in spermatozoal lipid peroxidation was essential for the maintenance of native structure, so complete loss of antioxidant action caused spermatozoal denaturation during lipid peroxidation [9]. Though a positive correlation of lead metal with the seminal plasma of oligo-, astheno- and teratozoospermia group was reported by Kasperczyk [10], the interference of inorganic lead to the hypothalamic-pituitary-gonadal axis and sperm characteristics has been the subject of great controversy since lead might have a secondary effect on the endocrine axis [11–14]. Moreover, information on the possible impact of lead on the structure and the function of human spermatozoa is still limited. Therefore, it was necessary to investigate the effect of lead on human spermatozoa of the battery and paint factory workers in these aspects.

Subjects and Methods

Chemicals and reagents

For sperm cell staining, hematoxylin monohydrate, light green SF and Eosin were procured from BDH, England (Gurr Certistain). For morphological, biochemical and metal analysis, the following chemicals were purchased from E-Merck, Germany: glutaraldehyde, formaldehyde, diphenyl amine, 2-4 dinitrophenyl hydrazine, disodium hydrogen phosphate dihydrate, sodium hydrogen orthophosphate, sodium hydroxide pellets, sodium chloride, sodium bicarbonate, sodium citrate dihydrate, sodium potassium tartarate, sodium cacodylate trihydrate, zinc sulfate heptahydrate, copper sulfate pentahydrate, anhydrous ferric chloride, resorcinol, D (−) fructose, ascorbate, trichloroacetic acid, acetic acid glacial, nitric acid, hydrochloric acid, sulfuric acid and hydrogen peroxide. BSA fraction V was obtained from Sigma (St Louis, USA). Thiobarbituric acid, orcinol monohydrate, RNA from torula yeast and DNA from herring sperm were purchased from Himedia, India. Tris, DPX mountant, thiourea, bromine ampoule, Folin Ciocalteau’s reagent, rectified spirit and absolute alcohol were procured from Glaxo, India.

Study design and selection of subjects

The present study was conducted in accordance with the Helsinki Declaration (1983). Prior to the study, ethical clearance was obtained from the Indian Council of Medical Re-
search (ICMR), Government of India. Fifty (n=50) non-occupationally exposed control subjects (group I) of active reproductive age (31-45 years) [15] were randomly selected after a proper medical check up by a physician. Lead-exposed workers of the same age group were selected from battery and paint manufacturing factories in Kolkata, India, and were divided into two groups, depending on the duration of exposure: low exposed group (group II: n=30) with 7 to 10 years exposure for 8 hours/day, and high exposed group (group III: n=50) with the same daily duration of exposure, but for longer periods, i.e. more than 10 to 15 years of lead exposure in the factories. Using interview technique as a tool for data collection, detailed information of the subjects was recorded on a pre-designed proforma (questionnaire), and the consent forms for voluntary donation of blood and semen samples were signed by the subjects.

Collection of biological samples

Semen samples were collected from the subjects in a clean, dry, sterilized, wide mouth, well stopper glass vial by masturbation [15, 16]. 0.5 ml semen was stored at −20 °C in a lead-free storage vial for lead content analysis. 2 ml of venous blood was collected aseptically and 1 ml was stored at −20 °C in a lead-free heparinized vial for metal analysis.

Analysis of sperm count, morphology, motility and cellular integrity

Sperm count, motility and morphology were determined [15]. Sperm velocity [17], viability by dye exclusion supra vital staining technique [18] and hypoosmotic swelling test (HOST) [19] were also measured at 400× and 1000× magnifications (CH20i, Olympus, India). The cell cycle phase distribution of sperm head DNA was studied by flow cytometer (FACS Vantage, Beckton Dickinson, USA) [20], and sperm cell surface was analyzed by scanning electron microscopy (SEM) at 10000× and 15000× magnifications (JSM 5200, Jeol Pvt. Ltd., Japan) [21] and also by atomic and lateral force microscopy (AFM and LFM) at native condition using 75000× and 80000× magnifications (Nanoscope E, Digital Instruments Inc., USA) [22], where LFM image indicated surface roughness at native condition, based on the surface composition as per the maker’s opinion (Digital Instruments Inc., USA).

Biochemical analysis of semen

After liquefaction, semen samples were centrifuged at 800× g for 10 minutes, and sperm was separated from the seminal plasma. Sperm ATPase activity [23], sperm membrane lipid peroxidation [24], total sperm protein [25] and nucleic acid contents [26, 27], as well as the amount of seminal plasma fructose [28] and ascorbate [29], were determined by spectrophotometer (DU64, Beckman Instrument Inc., USA).

PbB and PbS analysis

Blood and semen samples of the subjects were acid digested in the digestion chamber of Ethos D Microwave Labstation with Terminal 20 operating system (Milestone Srl, Italy) and
then absorbance was taken at 283.3 nm using atomic absorption spectrophotometer (GBC AVANTA AAS, software version 1.33, GBC Scientific Equipment Pvt. Ltd., Australia) attached to a GF 3000 graphite furnace [30].

**Statistical analysis**

The data obtained from the controls and exposed groups were compared, and one-way ANOVA, two-tail ‘t’ test and Scheffe’s ‘F’ test were carried out for level of significance using the computer based statistical software SPSS®, version 10.0 for Windows (SPSS Inc., USA).

**Results**

**Analysis of subjects as per the questionnaire data**

The demographic details of the subjects is shown in Table 1, which indicates all the subjects belonged to lower socio-economic status and most of them were addicted to smoking and consumption of alcohol as well as gutkha and panparag. There was no reproductive disorder among the subjects, but few workers were going for treatment, as they were issueless (Table 1). Questionnaire analysis also revealed that the majority of the subjects of group I (18.75%), II (19.35%) and III (16.12%) were 35 years of age.

**Table 1. Demographic details of the subjects**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control (group I ) n=50 (%)</th>
<th>Exposed (group II &amp; III) n=30 &amp; 50 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstinence time</td>
<td>4 days</td>
<td>4 days</td>
</tr>
<tr>
<td>Married for 5 years*</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>One child</td>
<td>15</td>
<td>25</td>
</tr>
<tr>
<td>Two children</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>More than two children</td>
<td>35</td>
<td>30</td>
</tr>
<tr>
<td>Issueless, undergoing treatment</td>
<td>Nil</td>
<td>5</td>
</tr>
<tr>
<td>History of miscarriage</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Lower socio-economic status*</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Smoking bidi for 10 years#</td>
<td>20</td>
<td>80</td>
</tr>
<tr>
<td>Alcohol (country liquor) consumption for 10 years</td>
<td>15</td>
<td>95</td>
</tr>
<tr>
<td>Gutkha/panparag consumed for 7 years#</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>Reproductive disease/disorders</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>

*: Wives not taken any pill and male counterpart never used any contraceptive devices  
*: Monthly income: average Rs. 3000.00 (1 US $ = Rs. 45.00 IC)  
#: Contained mainly tobacco (biddi), lime and tobacco (gutkha), dry areca nut and catechu (panparag), used widely in India  
n = Sample size
Effect of lead on sperm count, total sperm protein and nucleic acids content

There was a significant ($P < 0.001$) reduction in sperm count in the workers exposed to lead fumes and dust in the factories when compared to the non-occupationally exposed control persons (Table 2). The controls (group I) were normozoospermic in nature, and the high lead exposed workers (group III) were oligozoospermic, but the low lead exposed workers (group II) were in between the normozoospermic and oligozoospermic category [15]. There was no sign of polyspermidia after lead exposure. In support of sperm count, total sperm protein and nucleic acids contents were estimated, which showed a significant ($P < 0.001$) decrease in the same comparable groups (Table 2). The flow cytometrical analysis of sperm head nuclear DNA also showed a high percentage of hyploidy ($< n$ DNA) at sub $G_1$ phase as the exposure increased (Table 2).

### Table 2. Effect of lead on sperm count, total sperm protein and nucleic acid contents

<table>
<thead>
<tr>
<th>Group</th>
<th>Sperm count ($10^7$/ml)</th>
<th>Sperm protein (µg/mg cells)</th>
<th>Sperm RNA (µg/mg cells)</th>
<th>Sperm DNA (ng/mg cells)</th>
<th>Sperm DNA hyploidy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (I)</td>
<td>137.47 ± 39.42</td>
<td>27.17 ± 4.91</td>
<td>19.22 ± 4.42</td>
<td>72.01 ± 8.97</td>
<td>11.8 ± 7.2</td>
</tr>
<tr>
<td>(50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low exposed (II)</td>
<td>74.70 ± 15.44</td>
<td>14.61 ± 2.98</td>
<td>7.01 ± 1.37</td>
<td>30.17 ± 8.32</td>
<td>17.9 ± 1.50</td>
</tr>
<tr>
<td>(30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High exposed (II)</td>
<td>28.97 ± 10.62</td>
<td>5.57 ± 2.18</td>
<td>2.04 ± 0.67</td>
<td>4.98 ± 3.44</td>
<td>34.5 ± 4.20</td>
</tr>
<tr>
<td>(50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^a$: $P < 0.001$ (compared with group I), $^b$: $P < 0.001$ (compared with group II), $n$ = sample size, Values given were the mean ± SD of the control and exposed groups

Effect of lead on sperm morphological abnormality

Gross morphological abnormality of spermatozoa was significantly ($P < 0.001$) higher in both the exposed groups with respect to the controls (Fig. 1). The low exposed group (44.54 ± 2.93) and the high exposed group (60.04 ± 7.82) were teratozoospermic in nature, whereas the abnormality of the control group (33.75 ± 4.89) was within the normal range [15]. The present study also revealed that total sperm head, mid piece and tail abnormalities increased significantly ($P < 0.001$) after exposure (Fig. 1).

Sperm cell membrane integrity after occupational lead exposure

Sperm viability and HOST percentage was significantly ($P < 0.001$) reduced in both the exposed groups and in between the two exposed groups (Table 3). Lipid peroxidation of sperm membrane showed significant ($P < 0.001$) high malondialdehyde (MDA) content in group II and III workers in comparison to the control subjects of group I, which indicated loss of sperm membrane integrity after occupational lead exposure (Table 3). This finding was supported by a significant ($P < 0.001$) low level of seminal plasma total ascorbate with concomitant high value of dehydroascorbate (DHAA) concentration in seminal plasma of the same comparable groups (Table 3).
Fig. 1. Variations of human sperm morphological abnormality after lead exposure.

Values given were the mean ± SD of the control and exposed groups, where asterisks indicated significant difference of the exposed groups from the corresponding control group (*P < 0.001).

Table 3. Effects of lead on sperm viability, membrane lipid peroxidation and antioxidant profile of seminal plasma

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Sperm viability staining (%)</th>
<th>Sperm viability by HOST (%)</th>
<th>Sperm membrane lipid peroxidation (n mole MDA / 10⁶ cells)</th>
<th>Seminal plasma total ascorbate (µg/ml)</th>
<th>Seminal plasma DHAA (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (I) (50)</td>
<td>79.72±6.01</td>
<td>63.40±8.17</td>
<td>24.53±7.62</td>
<td>74.80±10.49</td>
<td>22.60±9.24</td>
</tr>
<tr>
<td>Low exposed (II) (30)</td>
<td>60.22±5.03[1]</td>
<td>38.33±5.60[1]</td>
<td>73.80±11.74</td>
<td>46.30±5.95[1]</td>
<td>42.30±6.18[1]</td>
</tr>
</tbody>
</table>

[1]: P < 0.001 (compared with group I), [2]: P < 0.001 (compared with group II), n = sample size.

Values given were the mean ± SD of the control and exposed groups.

SEM, AFM and LFM further authenticated the alteration of sperm surface morphology after occupational lead exposure, where 20 sperm cells per sample (n=15 samples in each group that showed high MDA, low sperm viability and altered seminal plasma ascorbate level) were examined. SEM exhibited a sharp depression and granular texture on the sperm cell surface of the exposed workers compared to the smooth membrane surface of the control persons (Fig. 2), hence confirming the previous membrane integrity study of the spermatozoa after lead exposure (Table 3). AFM (Fig. 3) and LFM (Fig. 4) images of sperm head
surface supported the SEM observation at native condition and thus finally confirmed the deterioration of sperm membrane structure after occupational exposure to lead.

Fig. 2. Representative SEM image of control and lead exposed human spermatozoa.

a) Control human sperm (group I) showed normal oval shaped head (H), mid piece (M) and proximal part of tail (T). Well defined acrosome area (A) and smooth membrane surface was prominent. The average length of sperm head was 3.5 µm and average breadth was 2.5 µm. Mid piece was approximately 1.7 µm long and 1.2 µm wide (×15000).

b) Exposed human sperm (group II) showed depression (↓) on swell mid piece (M). Granularity (↑) appeared on sperm head (H) and mid piece surface. (A) was the acrosome area and (T) was the part of the tail. The average length and breadth of sperm head was 3.8 µm and 2.2 µm, respectively (×10000).

c) Exposed human sperm (group III) showed abnormal elongated swell mid piece (M) with depression (↓). (T) was part of tail. Granular texture (↑) was noticed on amorphous shaped sperm head (H) surface. The average length and breadth of sperm head was 2.9 µm and 2.2 µm, respectively (×15000).
Fig. 3. Representative three-dimensional AFM image of control and lead exposed human sperm head surface.

a) The brighter area (↑) of the oval shaped control human sperm head (group I) indicated smooth membranesurface and the darker (reddish-brown) areas represented the lower portion of same cell (↓) or the depression between two adjacent cells (2,3). SEM could not reveal this pattern due to gold coating on the top of the sample, therefore this image was considered as the true structure of control sperm head surface at native condition (×80000).

b) Exposed human sperm head (group II) was characterized by uneven membrane surface (↑) of dark (reddish-brown) colour, hence the surface smoothness of the exposed cell was not comparable with that of the control one even at native condition (×75000).

Fig. 4. Representative three-dimensional LFM image of control and lead exposed human sperm head surface.

a) The control human sperm head (group I) surface was characterized by hill-like pattern, made by darker (reddish-brown) depressions/pits (↓) [range: 50–75 nm diameter] and brighter elevations/blebs like structures (↑) [range: 87.5–100 nm height]. Higher the elevations, brighter the area indicated increased surface roughness (↑), and lower the depressions, darker the area represented comparatively smoother surface (↓) of the control sperm head at native condition (×80000).

b) The exposed human sperm head (group III) surface was also characterized by different types of depressions/pits (↓) [range: 62–78 nm diameter] and elevations/blebs (↑) [range: 93–110 nm height]. Here also the brighter elevated areas indicated rough surface (↑), and darker (reddish-brown) depressed areas showed comparatively smoother zones (↓) at native condition, which was different from that of the control image, hence different surface morphology of sperm head might be due to occupational exposure to lead (×75000).
**Effect of Lead on Human Sperm**

Functional activity of spermatozoa after occupational exposure to lead

Sperm velocity, gross and forward and progressive (FP) motility were significantly ($P < 0.001$) decreased in lead exposed workers (group II and III) of battery and paint factories (Fig. 5). Significantly ($P < 0.001$) high level of seminal plasma fructose and reduced activity of sperm ATPase were also observed in the same comparable groups after occupational lead exposure (Fig. 5). Further, in the present study, sperm velocity, gross and FP motility decreased proportionately, while SM showed an inverse relationship in respect to duration of lead exposure (Fig. 5). The decrement ratio of sperm velocity, gross and FP motility in group I, II and III were approximately 15: 5: 1, 3: 2: 1 and 2.5: 1.8: 1, respectively, whereas the increment ratio of SM spermatozoa of the same comparable groups was approximately 1: 2: 3. This study also showed that the gross sperm motility and FP of all the three groups was much lower than the respective reference values [15].

![Sperm ATPase activity and Fructose levels](image)

**Fig. 5.** Effect of lead on sperm cell activity of control and exposed subjects. Values given were the mean ± SD of the control and exposed groups, where asterisks indicated significant difference of the exposed groups from the corresponding control group.

* $P < 0.001$.  

 []; Group I, []; Group II, []; Group III.
PbB and PbS level

Lead concentration in whole blood and semen was increased significantly \( (P < 0.001) \) in both the exposed groups and also between the two exposed groups (Table 4). The observed value of PbB in both the exposed groups were higher than the WHO’s permissible limit of 40 \( \mu \text{g/dl} \).

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>PbB (( \mu \text{g/dl} ))</th>
<th>PbS (( \mu \text{g/dl} ))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (I) (50)</td>
<td>13.62 ± 2.45</td>
<td>3.99 ± 1.36</td>
</tr>
<tr>
<td>Low exposed (II) (30)</td>
<td>48.29 ± 4.91 experts</td>
<td>10.85 ± 0.75 experts</td>
</tr>
<tr>
<td>High exposed (III) (50)</td>
<td>77.22 ± 1.25 experts</td>
<td>18.30 ± 2.08 experts</td>
</tr>
</tbody>
</table>

\( ^{\text{a1}}: P < 0.001 \) (compared with group I), 
\( ^{\text{b1}}: P < 0.001 \) (compared with group II), \( n = \) sample size, 
Values given were the mean ± SD of the control and exposed groups

Discussion

The present study was the very first study in India to deal with a working population routinely exposed to lead fumes and dust in battery and paint factories. The present study was undertaken with an idea to establish a relation between occupational lead exposure and structure function integrity of human spermatozoa among these factory workers. Several earlier studies reported the direct toxic effect of lead on sperm characteristics, with an increased incident of lead in several body fluids [3, 4, 31, 32]. Chronic lead exposure also caused significant diminution of sperm count among workers [6, 14, 32]. The present study revealed a significant reduction of sperm protein and NA content in the exposed factory workers, which might be related to the diminution of sperm cell production after occupational lead exposure. The whole event was manifested by oligozoospermia when compared with the non-occupationally exposed control subjects of the same socio-economic status [5, 33]. Further, the above finding was authenticated by flow cytometrical cell cycle phase analysis of sperm head DNA, where exposure-dependent decrease in intact DNA content with consequent rise in hyploidy (<n DNA) at sub G1 phase was evidenced among these workers, suggesting lead-induced fragmentation of sperm head DNA [34−36].

The factory workers were exposed to lead dust, fumes and finer particles that caused adverse effects not only on sperm count, but also on sperm morphology, depending on the du-
ration and nature of exposure [2, 10, 13]. Strikingly abnormal gross sperm morphology among the workers was evident in the present study, which was supported by the individual abnormalities of total sperm head, mid piece and tail after exposure, as revealed by light microscopy, and then authenticated by SEM, which further indicated lead induced morphological changes of human spermatozoa after occupational exposure [37–39].

As cellular viability depends on the intact membrane structure [20, 40], diminution of sperm vitality and HOST percentage along with high lipid peroxidation of sperm membrane as well as corresponding non-enzymatic antioxidant (ascorbate) profile in the workers of battery and paint factories suggested the probable loss of sperm membrane integrity after occupational exposure to lead [9, 41, 42]. This observation was supported by the surface structure study of spermatozoa through SEM [38], and then finally confirmed by AFM and LFM at native condition [35, 36, 39]. Several earlier studies provided information regarding sperm morphology and ultrastructural damages, but till date, internet searches revealed no such reports on surface analysis of spermatozoa after occupational exposure to lead, hence the present study reports the first ever SEM, AFM and LFM image of lead exposed human sperm cells.

Fructose was the main energy source for spermatozoal motility through fructolysis, where membrane bound ATPase plays an active role required for the development of progressive forward flagellar motion [5, 43, 44]. Sperm velocity was the average velocity of all spermatozoa in one sample [15], therefore related with gross and graded motile activity (FP, SM etc.) of spermatozoa. In the present study, lowering of sperm velocity and gross and FP motility with concomitant rise of SM were prevalent among the lead-exposed factory workers, which suggested that retarded sperm activity might be due to lead-induced alteration of normal fructolysis. High seminal plasma fructose level with consequent lowering of sperm ATPase activity further supported the above statement, and was also corroborated by several previous observations [7, 8, 35, 38]. Thus, the probable explanation is: in normal physiological conditions, fructose is utilized by ATPase during fructolysis, but lead inhibited this enzyme by replacing Na⁺, hence fructose was not utilized, rather it might be accumulated in the seminal plasma of the affected factory workers [45, 46]. Further, the association of low sperm motility with low antioxidant level and concomitant rise in the rate of MDA production in both the exposed groups of the present study suggested the susceptibility of spermatozoa to lipid peroxidation after occupational lead exposure [47, 48]. Also, a definite role of lead in impaired sperm motility and poor sperm DNA integrity was established in the present study [34–36].

In the present study, deterioration of sperm count, normal morphology, viability, membrane integrity and motile activity were associated with high PbB and PbS level in the workers in respect of duration of exposure [1, 10, 49]. Moreover, moderate lead exposure caused a reduction in sperm characteristics among the factory workers [12], and semen lead was the indicator of the industrial exposure [50].
Therefore, in conclusion it can be hypothesized that lead exposure might be responsible for high blood and semen lead levels in the battery and paint factory workers, and the presence of lead in both these biological fluids indicated that lead might cross the blood-testis-barrier and subsequently produce detrimental effects on human spermatozoa of the working population [5, 45, 51]. The normal cell membrane is the pre-requisite for the proper function of the cell, therefore it can be pointed out that a subtle membrane defect in morphologically abnormal spermatozoa due to lead-induced lipid peroxidation of the membrane in association with low antioxidant protection might be responsible for impaired functional activity of spermatozoa, i.e. impaired motility among these lead-exposed battery and paint factory workers in the present study. Smoking, consumption of alcohol, etc. might be the probable confounders in lead toxicity [52, 53], while the age of the subjects did not show any correlation after exposure, as active reproductive age group subjects were considered throughout the study [15, 16, 54].

Acknowledgements

This work was supported by a DST Grant from West Bengal. ROHC (ICMR), USIC (Jadavpur University) and IUC (Indore University) provided the laboratory facilities. Acknowledgement is due to Dr. B Manna, AD, NICED (ICMR), Kolkata for statistical analysis and Mr. PK Debnath for providing a computer facility. Thanks are due to the sample donors of the study without whose cooperation the study would have been incomplete.

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バッテリーと塗料工場における無機鉛暴露：ヒトの精子の形態と機能に対する影響

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要 旨： 鉛は工業的に重要な重金属の一つで、バッテリーと塗料工場労働者の男子に生殖障害を引き起こすことが知られているが、ヒトの精子の形態と機能に対する影響に関する情報は限られている。従って、これらの労働者における精子の形態と機能に対する鉛の影響を研究することは重要である。精子の蛋白と核酸濃度の低下を伴う乏精子症と精子 DNAの高倍数性の割合から、職業性の鉛暴露によって、精子細胞の産生が低下する事が示唆される。精子の活性性の低下、高濃度のマロンディアリドヒドロケテの脱水、変性の傾向を示唆する。精液アスコルピヒ酸レベルの変化を伴う低浸透圧下で膨化した精子の割合の増加は鉛暴露による過酸化の上昇と非酵素的抗酸化防御の低下によるものと考えられる。

キーワード： 精子の形態、精子の運動性、精子膜脂質の過酸化、血液と精液中の鉛濃度、鉛暴露労働者。
