ORIGINAL ARTICLE

Occupational Exposure to Low Concentrations of Lead Dust and Oxidative Stress in Mine Workers

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Abstract

Background: Several epidemiological studies have reported associations between high levels of lead exposure and oxidative stress (OS). However, research on the effects of low-level lead exposure remains limited. This study aims to assess the relationship between OS parameters and exposure to low concentrations of lead dust in mine workers.

Methods: This cross-sectional study evaluated 73 lead-exposed workers and 70 age- and sex-matched non-exposed individuals. Demographic data and occupational and medical history were collected through questionnaires. Workers' exposure to lead dust was assessed by air monitoring, and blood lead levels (BLLs) were calculated based on inhalation exposure. Blood samples were collected to determine OS parameters. Data were analyzed using SPSS version 21.0.

Results: The mean exposure of workers to lead dust was 24 µg/m³ (range: 1.5 to 185 µg/m³), which complied with the OSHA-PEL and ACGIH TLV-TWA standards for lead dust. The BLL in the exposed workers was found to be 45.47 µg/dL. A significant association was observed between the SOD/MDA ratio and exposure to lead dust. Additionally, a borderline negative association between lead exposure and superoxide dismutase (SOD) activity was found. A significant relationship was noted between workers' BMI and OS biomarkers.

Conclusion: This study's findings suggest that chronic exposure to lead dust may affect OS biomarkers, even at concentrations below the current OSHA-PEL and ACGIH TLV-TWA.

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Introduction

In occupational settings, workers are exposed to a variety of hazards, including chemical, physical, and biological agents.[1-6] Lead (Pb) has long been recognized as one of the most significant health hazards. Lead is widely used in various industries, and workers are primarily exposed to it through inhalation. Occupational exposure to lead is a common cause of toxicity in the blood, liver, nervous system, and kidneys in adults[7]. A growing body of evidence highlights the role of oxidative stress (OS) in lead toxicity. In cases of chronic exposure, lead-induced OS can contribute to the development of hypertension and cardiovascular diseases,[8] and it plays a key role in the underlying mechanisms of lead-induced hematotoxicity[7] and neurotoxicity.[9]

Oxidative stress (OS) is defined as an imbalance between the generation of oxidants, mainly free radicals such as reactive oxygen species (ROS), and the body's antioxidant defenses.[10] Key antioxidants,

including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase, play crucial roles in protecting the body against oxidative damage. The accumulation of ROS leads to a disruption in this balance, resulting in OS.[10] Several epidemiological studies have reported associations between high levels of lead exposure and biomarkers of OS.[11] However, studies examining the effects of low-level lead exposure are limited.[12, 13]

In the body, lead can induce oxidative damage by triggering lipid peroxidation (LPO) and impairing antioxidant defense systems, depending on the level of exposure.[11, 13] The reliance of antioxidant enzyme functions on essential trace elements makes these enzymes particularly vulnerable to lead toxicity. Previous studies have demonstrated significantly lower activities of superoxide dismutase (SOD) in lead-exposed workers compared to non-exposed groups.[11, 14] This reduced activity of SOD and other antioxidant enzymes may contribute to increased membrane LPO.[11] Direct binding of lead to the cell membrane further enhances its sensitivity to LPO. Malondialdehyde (MDA), the end product of LPO, is widely recognized as a reliable biomarker for assessing oxidative stress. Strong correlations have been reported between blood lead levels (BLL) and MDA levels in lead-exposed workers.[14] MDA is the most commonly used biomarker for evaluating LPO in studies of occupational lead exposure[15] and is considered the most sensitive indicator of oxidative stress in lead-exposed populations.[16]

A substantial body of evidence supports the notion that chronic lead exposure, even at concentrations insufficient to produce classic symptoms of lead poisoning, can lead to adverse health effects through increased OS levels. Studies on the impact of low-level lead exposure on OS biomarkers in humans remain scarce, with most published research based on experimental and animal studies.[17] At high exposure levels, OS is well-established as contributing to lead-related diseases. However, this relevance is controversial at low exposure levels, as most mechanistic studies have been conducted at higher concentrations.[13] The present study aimed to evaluate OS parameters in workers exposed to low levels of lead dust during extended work schedules in a lead-zinc mine.

Methods

Subjects and Study Design

This cross-sectional study included 73 workers from a lead-zinc mine and 70 age- and sex-matched non-exposed subjects. A brief questionnaire was administered to collect information on demographic variables, smoking habits, and occupational and medical history. Only workers in a good state of health were included in the study. Additionally, the medical records of the workers were reviewed, and individuals with any disease/disorder, a history of alcoholism, or exposure to chemicals other than lead were excluded. Workers with a time-weighted average (TWA) exposure exceeding the current TLV-TWA were also excluded.

All participants provided informed consent, and the university ethics committee approved the study protocol (IR.SUMS.REC. 1396.S1008). The study followed the Declaration of Helsinki (1964) and its revisions, including the 2000 update.

Assessment of Inhalational Lead Exposure

Workers were divided into similar exposure groups based on process, job, task, and environmental agents. In each group, one worker was randomly selected for air monitoring. A total of 28 personal air samples were collected following the NIOSH 7082 method.[18] Since lead workers were employed on extended work shifts (23 consecutive working days, 8 hours per day, followed by 7 successive rest days), the TLV-TWA for lead required adjustment. The model developed by the Institut de Recherche en Santé et en Sécurité du Travail (IRSST) was utilized for this adjustment.[19] The adjusted TLV-TWA (aTLV-TWA) was calculated to be 47 µg/m³. The procedures used to evaluate individual exposure to lead dust are described in detail in another paper published by our research group.[7]

Estimating Blood Lead Level

We used the equation developed by Richter et al.[20] to estimate BLL based on the workers' inhalational exposure as follows:

$$\text{Log BLL} = 1.430 + 0.165 \times \log \text{ALL}$$

This equation expresses BLL in µg/dL, and air lead levels (ALL) are described in µg/m³.

OS Assessment

Blood samples were collected from the workers at their workplaces before starting their shifts. Samples were drawn by trained personnel and kept in gel-containing tubes without anticoagulant. After centrifugation at 3000 rpm for 5 minutes at room temperature, the sera were separated and transferred into 1.5 mL microcentrifuge tubes, which were then stored at -70°C until analysis. Serum levels of lipid peroxidation (LPO) were measured using the thiobarbituric acid (TBA) reaction with the ZellBio GmbH Malondialdehyde (MDA) Assay Kit (Germany, CAT No. ZB-MDA96A).[21] The activity of superoxide dismutase (SOD) in serum samples was assessed using the ZellBio GmbH Superoxide Dismutase (SOD) Assay Kit (Germany, CAT No. ZB-SOD96A) according to the manufacturer's instructions. The ratio of oxidants, represented by MDA levels, to antioxidants, indicated by SOD concentrations, was used as an OS index.

Statistical Analysis

Data were analyzed using SPSS software, version 21.0. Mean, standard deviation (SD), and relative frequency (percent) were used to calculate variables. Independent samples t-tests and chi-squared tests were employed to compare the variables between groups. Linear regression analysis was conducted to evaluate the strength of the relationship between lead exposure and oxidative stress (OS) biomarkers and to control for the effects of potential confounders on changes in OS parameters. A p-value of <0.05 was considered statistically significant.

Results

The demographic characteristics of the subjects are presented in Table 1. No significant differences were observed between the groups in terms of age and smoking status. However, statistically significant differences were noted in body mass index (BMI), length of employment, and working hours per week. The workers' mean TWA exposure to lead dust was approximately 24 µg/m³ (1.5 to 185 µg/m³). Five workers with exposure levels exceeding the OEL-TWA of 50 µg/m³ set by OSHA and ACGIH were excluded from the study. No detectable levels of lead were found in air samples collected from the workplace environment of the non-exposed group. Based on the equation by Richter et al., the estimated BLL in the exposed workers was 45.47 µg/dL.

Table 2 compares the levels of OS biomarkers between the exposed and non-exposed groups. Serum MDA levels were significantly higher, while SOD activity was significantly lower in the exposed group compared to the non-exposed group. Additionally, the MDA/SOD ratio was significantly elevated in the exposed group.

Table 3 presents the adjusted associations between lead dust exposure and OS biomarkers. After adjusting for potential confounders, including BMI, length of employment, working hours per week, and smoking status, lead exposure demonstrated a borderline significant negative association with serum SOD activity (P=0.06) and a significant positive association with the MDA/SOD ratio (P=0.02). Specifically, lead exposure was associated with a 3.27-unit decrease in SOD activity and a 0.14-unit increase in the MDA/SOD ratio.

Table 4 illustrates the relationships between BMI, age, and OS biomarkers. Regression analysis revealed a significant association between workers' BMI and OS biomarker levels. In contrast, no significant relationship was observed between age and OS biomarkers.

Table 1: Demographic characteristics of the studied participants

Variable	Exposed group (n=73) (mean±SD)	Non-exposed group (n=70) (mean±SD)	P value
Age (year)	35.45±6.30	36.88±6.96	0.20*
BMI (kg/m²)	24.14±3.82	26.01±2.94	0.01*
Length of employment (year)	5.54±3.48	9.58±6.96	0.001*
Working hours per week	62.98±16.38	46.11±3.89	0.001*
Smokers (%)	16 (21.90)	6 (8.60)	0.052†
Married (%)	58 (79.50)	52 (74.30)	0.464†

*Independent sample t-test; †Chi-square test; SD: Standard deviation; BMI: Body mass index

Table 2: Comparison of oxidative stress biomarkers between exposed and non-exposed groups

Parameter	Exposed group (n=73) (mean±SD)	Non-exposed group (n=70) (mean±SD)	P value
MDA (µmol/l)	3.05±0.40	2.43±0.44	0.001
SOD (U/mL)	29.68±4.19	33.78±5.59	0.001
MDA/SOD	0.11±0.02	0.07±0.02	0.001

*Independent sample t-test

Table 3: Adjusted association between exposure to lead dust and OS parameters*

Parameter	B	SE	P value
MDA (µmol/l)	1.73	1.29	0.18
SOD (U/mL)	-3.27	2.28	0.06
MDA/SOD	0.14	0.06	0.02

*Linear regression analysis; SE: Standard error. MDA: Malondialdehyde; SOD: Superoxide dismutase

Table 4: Association between body mass index (BMI) and age with the oxidative stress parameters*

Parameters		B	SE	P value
BMI	MDA (µmole/l)	-0.028	0.013	0.040
	SOD (U/mL)	0.358	0.127	0.006
Age	MDA (µmole/l)	0.004	0.007	0.561
	SOD (U/mL)	-0.009	0.066	0.89

*Linear regression analysis; SE: Standard error; BMI: Body mass index

Discussion

In this study, we evaluated the effects of low-level exposure to lead dust on OS parameters in 73 lead-exposed workers and 70 age- and sex-matched non-exposed controls. The exposed workers were relatively young, and their exposure to lead dust was well below the adjusted TLV-TWA (aTLV-TWA) of 47 µg/m³. However, the mean BLL of 45.47 µg/dl was higher than the current biological exposure index (BEI) for lead, which is 20 µg/dl.[22] Recent studies suggest that there is no safe level of lead exposure,[22] and even exposure levels below the recommended TLV-TWA can lead to OS.

While the mean TWA exposure in our study was approximately half of the current TLV-TWA, the mean BLL was about twice as high as the recommended BEI for lead. This discrepancy can be attributed to the fact that lead has a strong affinity for body tissues, especially bones, as a heavy metal, resulting in a long half-life (approximately 20 to 30 years).[23] Consequently, repeated exposure to lead dust via inhalation can lead to lead accumulation in the body. Our study measured workers' exposure only once during a short period, which may only partially represent their real exposure over their average 5.54-year exposure duration. Other factors that can influence the body burden of lead include particle size, nutrition, and fasting.[24] Previous research has shown that iron deficiency is associated with elevated BLL, suggesting that inadequate iron levels may increase lead absorption.[25] Similarly, the mineral content of meals, particularly calcium and phosphate, can reduce lead absorption.[26]

The accumulation of lead in the body allows for exerting toxic effects. The role of oxidative stress (OS) in the pathophysiology of lead toxicity is well-documented.[21, 27] Consistent with previous studies,[14, 22] we observed significantly higher serum MDA levels, lower SOD activity, and a higher MDA/SOD ratio in the exposed workers compared to the non-exposed subjects (Table 2). However, after adjusting for potential confounders, only the MDA/SOD ratio remained significantly different between the groups (P=0.02), while serum SOD activity showed a borderline significant negative association with lead exposure (P=0.06) (Table 3). This finding is consistent with the results of Yin et al.[19]. Specifically, exposure to lead led to a 3.27-unit decrease in SOD activity (Table 3).

As a well-known product of LPO, MDA serves as a valuable screening tool for oxidative damage to lipids.[10, 28] The direct attachment of lead to cell membranes, particularly in cells with a higher affinity for lead, may increase their sensitivity to the LPO process. Since MDA levels are proportional to the number of double bonds in fatty acids, cells with more double bonds are more susceptible to damage than those with fewer double bonds. As the final product of OS, MDA is a reliable biomarker for lead-induced membrane LPO. Lead exposure induces an overproduction of free radicals and depletes antioxidant defenses, leading to direct reactions of these radicals with biological macromolecules. This increases peroxides, such as MDA, making it the most sensitive OS biomarker. Several studies have shown that LPO levels are directly proportional to lead concentrations, with most studies reporting increased MDA levels in workers with high lead exposure.[21, 29-31] Conversely, some studies have demonstrated significantly higher plasma MDA levels in workers exposed to low levels of lead compared to a non-exposed group.[28, 32, 33] However, in this study, we did not find a significant increase in MDA levels among lead-exposed workers. This finding is consistent with the results reported in other studies.[34]

The electron-sharing affinity of lead plays a crucial role in the covalent attachment between this heavy metal and the -SH groups of proteins. Many enzymes that protect the body from OS contain -SH groups at their active sites, and these enzymes become inactive due to the direct binding of lead.[35] SOD, an antioxidant enzyme that removes peroxides, is a potential target for lead toxicity.[30] As the first detoxification enzyme and one of the most potent antioxidants, a decrease in SOD activity can initiate a chain reaction of free radicals and LPO. The overproduction of free radicals results in peroxidation, producing MDA, which causes crosslinking and polymerization of biological macromolecules.[12] Therefore, evaluating SOD concentration could serve as a complementary test for lead-induced membrane LPO.

Previous studies have shown that lead exposure can cause both an increase and a decrease in serum SOD activity in a dose-dependent manner.[21] Increased SOD activity is typically observed at high levels of lead exposure. In contrast, at lower exposure levels, like those in the present study, SOD activity remained either unchanged or decreased compared to control groups.[14, 22, 32] For instance, Hormozi et al. reported a significant decrease in serum SOD activity in glazers exposed to lead for about eight years in the tile industry. Conversely, Shraideh et al. observed a significant increase in serum SOD activity and MDA levels in Jordanian automobile workers exposed to lead. The findings of the present study align with the notion that lead can induce and elevate OS even at low doses.[15] The reduced serum SOD activity in the exposed group, compared with the non-exposed group, may be attributed to the interaction between lead and copper molecules. Since SOD is a metal-containing enzyme (zinc and copper), lead exposure can result in copper deficiency, which in turn reduces SOD activity.[35]

The ratio of oxidants, as measured by MDA concentration, to antioxidants, represented by SOD levels, was used as an index of oxidative stress (OS). In the present study, the MDA/SOD ratio in the exposed group was more than two-fold higher than in the non-exposed group (0.16±0.02 vs. 0.07±0.02), and the difference was statistically significant (Table 2). After adjusting for potential confounders, lead exposure resulted in a 0.14-unit increase in the MDA/SOD ratio (Table 3). This finding is consistent with the results of Shraideh et al., who reported a 2- to 4-fold increase in the MDA/SOD ratio in lead-exposed workers compared to a control group.[36]

Regression analysis showed a significant association between workers' BMI and OS biomarkers (Table 4). In contrast, no statistically significant association was observed for age. Several studies have reported associations between OS, age, and BMI.[34, 37, 38] Obesity is a state of chronic OS, and obese individuals tend to have significantly lower antioxidant levels.[34] OS progresses with increasing stages of obesity, and individuals with higher age and BMI face a greater risk of OS.[39, 40] While OS biomarkers are generally more strongly associated with age than BMI,[37] no such association was found in this study. This may be due to the relatively young age of the exposed workers (35.45±6.30 years) and their relatively short exposure duration (5.54±3.48 years). However, consistent with other studies,[34, 37, 38] significant associations were observed between BMI and levels of MDA and SOD (Table 4). In obese and overweight individuals, excessive fat accumulation poses health risks, potentially leading to LPO and decreased activity of cytoprotective enzymes, even in the absence of smoking or metabolic, renal, or hepatic disorders.[39]

The present study has some limitations. We assessed only two biomarkers of OS, namely MDA and SOD. Measuring additional biomarkers would have provided a more comprehensive indication of the OS status in lead-exposed workers. Additionally, due to the study's cross-sectional nature, a cause-and-effect relationship could not be established.

Conclusion

Workers at the studied lead-zinc mine had low inhalational exposures to lead dust; however, their BLL exceeded the BEI. The findings suggest that even low-level occupational exposure may affect OS biomarkers, indicating that the current OEL-TWA for lead may not be sufficiently protective for workers. Furthermore, higher BMI could be a risk factor for elevated OS biomarker levels. It is recommended that future longitudinal studies, with sufficient follow-up periods and detailed data on long-term occupational lead exposure, be conducted to substantiate these preliminary observations further.

Authors' Contribution

Fateme Kooshki and Fatemeh Rahimian, Fereshteh Aliasghari: Methodology, Investigation, Data curation, Writing – Original Draft; Masoud Neghab: Supervision, Methodology, Writing – Review and Editing; Esmaeel Soleimani: Writing – Review and Editing, Project Administration, Methodology, Funding Acquisition, Conceptualization.

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