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ORIGINAL PAPER

THE INFLUENCE OF CHRONIC OCCUPATIONAL EXPOSURE TO LEAD ON PERIPHERAL NERVES CONDUCTION

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Highlights

- Chronic lead exposure impairs peripheral nerve conduction.
- Oxidative stress plays a key role in lead-induced neurotoxicity.
- Heme biosynthesis disruption contributes to PNS dysfunction.

Abstract

Background: The impact of lead on the peripheral nervous system (PNS) remains inadequately explored. Despite the absence of classic lead neuropathy resulting from uncontrolled exposure to high metal doses in workers under biomonitoring, the focus shifts to chronic exposure at lower doses. This study aims to evaluate the influence of occupational lead exposure on peripheral nerve conduction in chronically exposed individuals. **Material and Methods:** The study comprised 58 workers, divided based on an 8-year mean serum lead concentration into lower exposure (blood lead [PbB] level ≤40 μg/dl) and higher exposure (PbB level >40 μg/dl) subgroups. Conduction tests and laboratory assessments, including oxidative stress parameters, were conducted. **Results:** In the higher lead exposure group, average amplitudes of motor fiber-evoked potentials and sensory fiber conduction velocity were 8% and 3% lower, respectively, compared to the lower exposure group. Correlation analysis revealed positive correlations between sensory fiber-evoked potential amplitudes and blood serum sulfhydryl groups, and catalase activity. Motor fiber-evoked potential amplitudes negatively correlated with lipofuscin concentration. Lipofuscin concentration also negatively correlated with sensory fiber-evoked potential amplitudes. Zinc protoporphyrin concentration negatively correlated with sensory fiber-evoked potential amplitudes and conduction velocity. **Conclusions:** Chronic lead exposure disrupts peripheral nerve conduction, evident in reduced motor fiber-evoked potential amplitudes and slower sensory fiber conduction velocity. The toxic impact on the PNS may be attributed to oxidative stress and heme biosynthesis disorders. Med Pr Work Health Saf. 2025;76(2)

Key words: occupational exposure, lead, polyneuropathy, nerve conduction, oxidative stress, chronically exposed

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INTRODUCTION

Lead is a toxic element that enters the body mainly through food and inhalation. Environmental or workplace exposure causes lead to accumulate in the hematopoietic system, bones, skin, nervous system, and organs like the liver and kidneys. This leads to functional disorders in almost all bodily systems, including the peripheral nervous system (PNS). The symptoms vary depending on age, exposure duration and intensity, blood lead (PbB) concentration, and factors like alcohol consumption, nutrition, and levels of vitamins and minerals such as calcium.

Lead affects the body through several mechanisms, including oxidative stress and protein function disorders. It disrupts the activity of enzymes like delta-aminolevulinic acid dehydrogenase, leading to metabolic and physiological changes. Higher lead concentrations increase reactive oxygen species, which interfere with the antioxidant system, affecting glutathione metabolism and enzymes

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such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) [1]. Reactive oxygen species cause protein oxidation, lipid peroxidation, and deoxyribonucleic acid (DNA) damage [2]. Lead's high affinity for sulfhydryl groups (SH) and its similarity to essential elements like calcium, magnesium, and zinc allow it to compete for binding sites in proteins and enzymes, disrupting their biological functions [3].

Nervous tissue, particularly in the central nervous system (CNS) and PNS, is highly sensitive to oxidative stress. Neurons have more unsaturated fatty acids in their membranes, making them susceptible to free radical attacks. The myelin sheath, which insulates nerve fibers, contains 76% lipids, while other cell membranes contain about 50% [4]. Unsaturated fatty acids are prone to oxidation, and neurons have a high surface-to-volume ratio, with mitochondria close to cell membranes, making them vulnerable to oxidative damage [5]. Neurons also rely on a delicate balance between the production and neutralization of free radicals for proper function. Disruptions to this balance, as shown in experimental studies, affect ionic balance and membrane potential, leading to neuronal dysfunction [6]. Histochemical studies have revealed uneven distributions of glutathione and oxidative enzymes in peripheral nerves, contributing to selective neurotoxicity [7]. Additionally, antioxidant enzyme activity is lower in PNS cells than in CNS cells, making the PNS more vulnerable to oxidative damage. This suggests that lead polyneuropathy is largely due to lead-induced oxidative stress [8,9].

Lead polyneuropathy has traditionally been seen as a motor disorder, characterized by weakness in muscles like wrist extensors and shoulder muscles. Sensory symptoms are rare, unlike in other heavy metal poisonings like arsenic, thallium, or mercury, where sensory issues dominate. Lead polyneuropathy symptoms often occur alongside other systemic complaints like abdominal pain, constipation, or anemia [10].

While much is known about lead's effects on the human body, its impact on the PNS is less understood. In people chronically exposed to lead, various patterns of PNS involvement are observed. Neuropathy symptoms appear in a significant percentage of those exposed, although the onset is often delayed due to lead's slow release from the bones. This delay complicates establishing a clear link between neuropathy and past lead exposure [11].

In the past, peripheral nerve damage was associated with acute or subacute high-concentration lead exposure. With modern monitoring and employee health checks, such cases are now rare. Instead, the focus has shifted to the effects of chronic, low-dose lead exposure, particularly on the PNS. Assessing this impact is the aim of this paper.

MATERIAL AND METHODS

Material

A group of 58 manual workers of Zinc Smelter "Miasteczko Śląskie" took part in the study. During the medical examination, as part of periodical examinations, the following epidemiological data were collected: age, time of work in exposure conditions, smoking, current and past diseases. Weight and height, as well as blood pressure, were also measured. The study group consisted of all eligible employees who provided informed consent to participate in the study and who had been continuously employed at the Zinc Smelter "Miasteczko Śląskie" during the entire study period, with regular exposure to lead.

In order to determine the degree of exposure to lead, 2 biomarkers were used: PbB concentration and blood zinc protoporphyrin (ZPP) concentration. These parameters were measured >8 years, or less in the case of a shorter period of employment, every 3 months, on average. Blood lead levels were measured quarterly, resulting in approx. 32 measurements per participant >8-year follow-up period. This regular monitoring allowed for a robust assessment of long-term lead exposure.

The criterion for inclusion in the study was occupational exposure to lead and its concentration exceeding 25 μg/dl in whole blood. The criteria for exclusion (non-inclusion) from the study were the presence of chronic diseases other than hypertension and the use of drugs other than antihypertensives, with the exception of drugs from the group of calcium channel blockers.

Peripheral nerve conduction tests were performed in all qualified employees. The examination was carried out by an experienced electromyographer using the Keypoint Dantec device (Natus Neurology, Middleton, USA). Motor fibers in 5 nerves (median, ulnar, radial, peroneal and tibial) and sensory fibers in 4 nerves (median, ulnar, radial, sural) were evaluated bilaterally. In each nerve, latency, evoked potential amplitude and conduction velocity were determined, and in motor fibers additionally the F-wave. Disposable surface electrodes were used for the examination. Averaged results were used for the purpose of statistical analysis.

The study group was divided into 2 subgroups depending on the average value of lead concentration

in blood serum >8 years; a subgroup with lower exposure (PbB ≤40 μg/dl) and a subgroup with higher exposure (PbB >40 μg/dl).

For the purpose of blood chemistry 15 ml of blood was collected from the basilic vein (using Vacuette tubes, Greiner-Bio, Frickenhausen, Germany) to obtain whole blood, serum and erythrocytes. Depending on the frequency of periodic examinations, blood was collected 2–4 times a year. Averaged results were used for statistical analysis. The study was approved by the Bioethics Committee of the Medical University of Silesia in Katowice with the issue of approval No. KNW/022/KB1/108/14 of September 30, 2014. All participants provided written informed consent prior to enrolment in the study. This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

Methods

Determination of PbB concentration

Blood lead levels were measured using atomic absorption spectrophotometry (ICE 3400, Thermo Fisher Scientific, Waltham, MA, USA), and results were expressed in μg/dl.

Determination of ZPP concentration in blood

Zinc protoporphyrin concentration in blood was determined fluorometrically using an Aviv 206 hematofluorometer (AvivBiomedical, Lokewood, NJ, USA), expressed in μg/g hemoglobin (Hb), with excitation at 415 nm and emission at 596 nm.

Determination of protein concentration

Serum protein concentration was measured using a colorimetric biuret reaction method, with results expressed in g/l.

Peripheral blood morphology

Complete blood counts, including leukocytes, erythrocytes, platelets, Hb, and hematocrit, were measured using the Sysmex K-4500 analyzer (GMI, Ramsey, MN, USA).

Determination of Hb

Hemoglobin was quantified in 10% hemolysate using the cyanmethemoglobin method with Drabkin's reagent, results expressed in g/dl.

Determination of lipid peroxide (LPH) concentration

Concentration of lipid peroxide in serum was determined by oxidizing Fe^{2+} to Fe^{3+} in an acidic environment, followed by Fe^{3+} complexation with xylene orange, as per the method of Arab and Steghens [12], expressed in μmol/l.

Determination of malondialdehyde (MDA) concentration

Malondialdehyde levels in serum and erythrocytes were measured fluorometrically, based on a reaction with 2-thiobarbituric acid using the method of Ohkawa et al. [13], and results were expressed as μmol/l for serum and nmol/g Hb for erythrocytes.

Determination of lipofuscin (LPS) concentration

Lipofuscin levels in serum and erythrocytes were determined using the Tsuchida et al. [14] method, and results were presented as relative units (RF).

Determination of SOD activity

Superoxide dismutase activity in serum and erythrocytes was measured using the Oyanagui method [15], expressed in nitro units (NU). In serum, SOD activity was expressed as NU/ml, while in erythrocytes as NU/mg Hb.

Determination of SH concentration

Serum sulfhydryl group concentrations were determined using 5'5'-dithiobis(2-nitrobenzoic acid) and measured at 412 nm, expressed in μmol/l and μmol/g of protein.

Catalase activity assay

Catalase activity in erythrocytes was determined by a spectrophotometric method based on its reaction with methanol and hydrogen peroxide by the method of Johansson and Borg [16], expressed in kIU/g Hb.

Determination of GPx activity

Glutathione peroxidase activity in erythrocytes was measured using the method of Paglia and Valentine [17] which tracks the oxidation of glutathione and nicotinamide adenine dinucleotide phosphate (NADPH), and expressed in IU/g Hb.

Determination of glutathione reductase (GR) activity

Glutathione reductase activity in erythrocytes was determined using the method of Richterich [18], which measures changes in reduced NADPH concentration, expressed in IU/g Hb.

Determination of glutathione-s-transferase (GST) activity

Determination of GST activity in erythrocytes was measured kinetically using the Habig and Jakoby [19] method. Results were expressed in mIU/g Hb.

Determination of calcium concentration

Serum calcium levels were determined by atomic absorption spectrophotometry (ICE 3400), expressed in mmol/l.

Determination of magnesium concentration

Serum magnesium concentration was measured using atomic absorption spectrophotometry (ICE 3400), expressed in mmol/l.

Determination of iron concentration

Serum iron levels were determined using atomic absorption spectrophotometry (ICE 3400), expressed in μg/dl.

Determination of zinc concentration

Zinc concentration in serum was also determined by atomic absorption spectrophotometry (ICE 3400), expressed in mmol/l.

Statistical analysis

Statistical analyses were performed using Statistica 10.0 PL software (StatSoft Polska, Kraków, Polska). Descriptive statistics, such as means, standard deviations, and medians, were calculated. The Shapiro-Wilk test was used to assess the normality of data distribution, and variance homogeneity was verified using the Levene test. Group comparisons were conducted using the Student's t-test or Mann-Whitney U test for independent samples. Spearman's correlation analysis was applied to evaluate relationships between variables, with a significance level of $p < 0.05$ considered statistically significant.

RESULTS

Both subgroups did not show statistically significant differences in terms of age, years of work, body mass index (BMI), hypertension and smoking. However, they differed in the average value of lead concentration in the blood serum determined over the last 8 years of work, which results from the assumptions of the work and the adopted division of the subjects into groups (Table 1).

In the group of people with higher exposure to lead, the average amplitudes of evoked potentials ob-

Table 1. Epidemiological data and lead exposure parameters in a group of 58 manual workers from the Zinc Smelter "Miasteczko Śląskie", Poland

Variable	Participants (N = 58)		Change [%]	p
	average PbB ≤40 μg/dl (N = 36)	average PbB >40 μg/dl (N = 22)		
Socioeconomic				
age [years] (M±SD)	44.25±9.86	47.50±9.68	7	0.225
seniority [years] (M±SD)	19.06±10.88	20.14±10.46	6	0.711
height [cm] (M±SD)	176.72±6.83	175.95±5.11	0	0.651
weight [kg] (M±SD)	85.28±14.51	87.27±13.79	2	0.607
BMI [kg/m²] (M±SD)	27.24±3.99	28.08±3.49	3	0.417
Medical				
diabetes mellitus [%]	0.0	0.0		
coronary artery disease [%]	0.0	0.0		
hypertension [%]	19.4	27.3		0.497
smoking [%]	30.6	36.4		0.654
blood pressure (M±SD)				
diastolic	125.14±9.37	126.59±836	1	0.554
systolic	80.28±4.77	80.00±6.36	0	0.851
PbB (8-year M) [μg/dl] (M±SD)	32.03±6.95	46.88±5.88	46	**0.000**
ZPP (8-year M) [μg/g Hb] (M±SD)	5.25±2.72	7.79±3.09	48	**0.002**

BMI – body mass index, PbB – blood lead, ZPP – zinc protoporphyrin.
Bolded are statistically significant values.

Table 2. Parameters of nerve conduction in a group of 58 manual workers with regular exposure to lead from the Zinc Smelter "Miasteczko Śląskie", Poland

Variable	Nerve conduction				Change [%]	p
	participants with average PbB ≤40 µg/dl (N = 36)		participants with average PbB >40 µg/dl (N = 22)			
	M	SD	M	SD		
Motor fibers						
latency	3.79	0.68	3.85	0.94	2	0.426
amplitude	9.93	4.15	9.18	3.22	–8	**0.022**
velocity	55.48	7.59	54.66	7.98	–1	0.268
Sensory fibers						
amplitude	12.30	6.73	11.11	5.90	–10	0.053
velocity	51.29	7.53	49.82	9.09	–3	**0.036**

PbB – blood lead.
Bolded are statistically significant values.

tained from the motor fibers of the examined nerves and the average conduction velocity in the sensory fibers of the examined nerves were lower by 8% and 3%, respectively, compared to the group of people with lower exposure. In contrast, ZPP levels were 48% higher in the higher lead exposure group than in the lower lead exposure group (Tables 1–2).

There were no statistically significant differences between the studied groups in terms of the values of other conduction parameters of the examined long nerves, the concentrations of oxidative stress markers, morphotic elements of the blood or the concentrations of the tested macro- and microelements in the blood serum (Table 3).

A statistically significant, positive correlation was observed between the magnitude of the amplitudes of evoked potentials in sensory fibers and the concentration of SH, CAT and Hb concentration, hematocrit value and the number of erythrocytes in the blood serum. The number of erythrocytes correlated positively with the values of conduction velocity in sensory fibers. A positive correlation was also observed in the case of the magnitude of the amplitudes of potentials evoked from motor fibers and the concentration of iron and the number of erythrocytes in the blood serum. The value of the amplitudes of potentials evoked from motor fibers negatively correlated with the age of the subjects, magnesium concentration, LPS concentration, GPx and SOD activity. The amplitudes of potentials evoked from sensory fibers showed a negative correlation with age, years of work, BMI, ZPP, magnesium, LPS concentrations, GPx and SOD activity. A negative correlation was also observed between the values of conduction velocity in the sensory fibers of the examined nerves and the number of leukocytes, the activity of SOD and the concentration of ZPP (Table 4).

DISCUSSION

The correlations between age, years of work, and BMI with nerve conduction parameters do not influence the study's findings, as these factors were similar across groups. The negative correlation between evoked potential amplitudes and age aligns with age-related declines in nerve function. Longer work exposure and higher BMI also correspond with aging.

With improved working conditions and lead monitoring, cases of classic lead polyneuropathy, once characterized by wrist muscle weakness, have become rare [11]. However, significant differences in conduction parameters, such as response amplitudes from motor fibers and sensory fiber conduction velocities, were observed, indicating that chronic lead exposure continues to impair peripheral nerve function. The differential effects on motor and sensory fibers likely arise from structural differences, including fiber diameter, myelin sheath thickness, and antioxidant enzyme activities in different types of nerve tissues.

Previous studies commonly report a reduction in motor conduction velocity and motor evoked potentials in individuals exposed to lead. Sensory conduction and evoked potential amplitudes are less commonly affected. Some cases of lead poisoning show electrophysiological changes resembling amyotrophic lateral sclerosis or even primary muscle damage, highlighting the wide variability in clinical presentation [20]. The lack of

Table 3. Peripheral blood morphology, metal concentration and oxidation stress parameters in a group of 58 manual workers with regular exposure to lead from the Zinc Smelter "Miasteczko Śląskie", Poland

Parameter	Participants (N = 58)		Change [%]	p
	participants with average PbB ≤40 µg/dl (N = 36)	participants with average PbB >40 µg/dl (N = 22)		
Peripheral blood morphology (M±SD)				
L [g/l]	6.91±2.12	7.35±1.91	6	0.435
Er [T/l]	4.90±0.27	4.90±0.31	0	0.960
Hb [g/dl]	15.09±0.81	15.33±1.14	2	0.347
Ht [%]	42.97±1.95	43.60±2.98	1	0.332
PLT [g/l]	223.97±38.08	217.14±43.84	–3	0.534
Metal (M±SD)				
Ca [mmol/l]	2.34±0.25	2.39±0.19	2	0.481
Mg [mmol/l]	0.81±0.14	0.83±0.12	3	0.514
Zn [mmol/l]	14.59±5.85	12.74±3.06	–13	0.223
Fe [µg/dl]	21.81±6.34	18.76±10.00	–14	0.303
Oxidation stress (M±SD)				
LPH [µmol/l]	3.23±1.43	3.24±0.46	0	0.972
MDA [µmol/l]	2.83±1.08	3.08±0.60	9	0.336
LPS [RF]	614.98±241.95	592.91±167.29	–4	0.719
SH				
[µmol/l]	344.53±65.08	358.58±57.80	4	0.431
[µmol/g protein]	4.65±0.95	4.94±0.97	6	0.291
SOD [NU/ml]	19.61±3.42	21.38±4.20	9	0.105
MDA [nmol/g Hb]	198.67±69.51	210.55±68.93	6	0.550
LPS [RF/g Hb]	707.96±382.21	714.80±242.04	1	0.943
GR [IU/g Hb]	6.14±2.40	6.53±2.08	6	0.552
GST [mIU/g Hb]	227.90±160.46	300.81±151.25	32	0.109
GPx [IU/g Hb]	73.59±63.01	61.74±56.02	–16	0.498
CAT [kIU/g Hb]	369.88±236.21	435.49±166.31	18	0.279
SOD [NU/mg Hb]	256.31±131.61	215.69±77.45	–16	0.211

Ca – calcium, Er – erythrocytes, Fe – iron, GPx – glutathione peroxidase, GR – glutathione reductase, GST – glutathione-s-transferase, Hb – hemoglobin, Ht – hematocrit, CAT – catalase, L – leukocytes, LPH – lipid peroxide, LPS – lipofuscin, MDA – malondialdehyde, Mg – magnesium, PLT – platelets, SH – sulfhydryl group, SOD – superoxide dismutase, Zn – zinc.

a single mechanism explaining lead's neurotoxic effects is likely due to the differences between acute poisoning and chronic exposure. Individual sensitivity also plays a crucial role [11].

In a 1965 experiment, Fullerton observed nerve damage in 60% of guinea pigs acutely poisoned with lead, while only 12.5% exhibited paralysis, suggesting that lead-induced nerve damage can be subclinical [21]. This reinforces the need for electrophysiological testing in detecting early nerve damage.

In this study negative correlation between ZPP concentration and sensory nerve conduction, likely due to lead's inhibition of ferrochelatase, an enzyme essential for heme synthesis was observed. Lead toxicity disrupts heme production, elevating ZPP levels and impairing nerve function [22]. While the link between ZPP and sensory fibers is stronger than for motor fibers, this may reflect the greater sensitivity of sensory fibers to toxic factors like hypoxia and nutritional deficiencies [23].

Oxidative stress also emerged as a key factor in nerve damage. The negative correlation between LPS concentration, an oxidative stress marker, and evoked potential amplitudes suggests that chronic oxidative stress impairs nerve function [24]. The positive correlation between

Table 4. Spearman's correlation beetween motor and sensory fibres and socioeconomic and medical variables in a group of 58 manual workers with regular exposure to lead from the Zinc Smelter "Miasteczko Śląskie", Poland

Variable	Spearman's correlation				
	motor fibers			sensory fibers	
	latency	amplitude	velocity	amplitude	velocity
Socioeconomic					
age	0.02	**–0.13**	–0.08	**–0.27**	**–0.15**
seniority	0.06	–0.03	–0.06	**–0.14**	–0.07
BMI	–0.02	–0.04	–0.02	**–0.13**	0.00
Medical					
PbB (8-year M)	–0.05	–0.08	0.00	–0.04	–0.03
ZPP (8-year M)	0.00	–0.02	–0.01	**–0.15**	**–0.09**
L[a]	0.06	–0.04	–0.05	–0.06	**–0.16**
Er[a]	–0.01	**0.09**	0.00	**0.12**	**0.10**
Hb[a]	–0.01	0.06	0.01	**0.13**	0.07
Ht[a]	–0.02	0.07	0.01	**0.12**	0.05
PLT[a]	–0.02	–0.03	0.04	–0.02	0.01
Ca	0.07	0.02	0.00	0.10	0.00
Mg	0.08	**–0.10**	–0.09	**–0.14**	–0.06
Zn	–0.04	0.03	0.01	0.03	0.02
Fe	0.06	**0.11**	0.06	0.01	0.01
MDA	0.01	–0.07	–0.05	–0.06	–0.03
LPS	–0.08	0.01	0.08	–0.02	0.05
SH					
[µmol/l]	–0.05	0.07	0.00	**0.11**	0.02
[µmol/g protein]	–0.09	0.02	0.00	**0.10**	0.05
SOD	0.04	0.02	–0.03	–0.05	–0.03
MDA	–0.06	–0.02	0.04	0.00	0.02
LPS	**0.12**	**–0.09**	–0.02	**–0.14**	–0.06
GR	**0.13**	0.02	–0.04	0.09	–0.03
GST	–0.01	0.03	–0.02	0.04	–0.01
GPx	0.09	**–0.11**	–0.04	**–0.23**	–0.02
CAT	0.08	0.07	–0.02	**0.11**	–0.02
SOD	**0.12**	**–0.11**	–0.07	**–0.26**	**–0.11**

BMI – body mass index, Ca – calcium, Er – erythrocytes, Fe – iron, GPx – glutathione peroxidase, GR – glutathione reductase, GST – glutathione-s-transferase, Hb – hemoglobin, CAT – catalase, L – leukocytes, LPS – lipofuscin, MDA – malondialdehyde, Mg – magnesium, PbB – blood lead, PLT – platelets, SH – sulfhydryl group, SOD – superoxide dismutase, ZPP – zinc protoporphyrin, Zn – zinc.
Bolded are statistically significant values.
[a] The result of a survey conducted in 2016.

CAT activity, thiol groups, and sensory fiber function further supports the role of the antioxidant defense system in protecting nerves. Conversely, the negative correlation between SOD and GPx activity with evoked potentials suggests that oxidative stress induces the upregulation of these antioxidant enzymes [25].

The relationship between oxidative stress and nerve dysfunction is further supported by studies of ethanol- and metal-induced neuropathies [6]. In such cases, free radical damage to cellular structures, including membrane lipids and proteins, leads to impaired nerve conduction. Reduced glutathione levels, a key antioxidant,

have been linked to deteriorating nerve function in both diabetic and experimentally induced neuropathy models [26]. Activation of protein kinase enzymes may further deplete intracellular antioxidants, exacerbating nerve damage [27].

The imbalance between oxidative stress and antioxidant defenses leads to myelin damage, resulting in slower conduction, axonal loss, and eventually complete nerve dysfunction [28]. Experimental studies have confirmed that reduced glutathione levels, induced by ethanol or other toxins, significantly impair nerve conduction. Once glutathione levels fall <60% of normal, nerve cell excitability is lost. Lead's impact on calcium channels may further exacerbate nerve dysfunction, as it increases intracellular calcium concentrations, triggering neurotransmitter release and synaptic disruption [29].

While no significant correlation was found between serum calcium levels and nerve function, this may reflect the inability of serum calcium measurements to capture the localized effects of lead on neuronal calcium channels. The observed negative correlation between magnesium levels and evoked potentials may be explained by magnesium supplementation among individuals with high lead exposure, as magnesium is known to protect against lead toxicity by stabilizing DNA, proteins, and mitochondria. Magnesium is also essential for proper calcium channel function and may mitigate some of the neurotoxic effects of lead [30].

This paper highlighted that recent studies suggest that adverse health effects, particularly related to cardiovascular and neurological functions, can occur even at PbB levels below the current regulatory limit. This study emphasizes the importance of ongoing review and potential reconsideration of occupational exposure limits to ensure they reflect the latest scientific evidence and provide optimal health protection for exposed workers.

In conclusion, the observed relationships between oxidative stress markers, heme biosynthesis disruption, and nerve conduction parameters suggest that chronic lead exposure continues to impair peripheral nerve function, even in the absence of classic polyneuropathy. Further research is needed to explore potential protective strategies, such as antioxidant therapy, in populations at risk of chronic lead exposure.

CONCLUSIONS

Chronic exposure to lead disturbs conduction in peripheral nerves, which is manifested by a decrease in the amplitude of potentials evoked from motor fibers and slower conduction speed in sensory fibers. Oxidative stress and heme biosynthesis disorders may be responsible for the toxic effect of lead on the PNS.

AUTHOR CONTRIBUTIONS

Research concept: Michał Dobrakowski, Małgorzata Cisowska
Research methodology: Małgorzata Cisowska
Collecting material: Kinga Filipecka
Statistical analysis: Sławomir Kasperczyk
Interpretation of results: Małgorzata Cisowska
References: Anna Machoń-Grecka

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