Lead-based paint's impact on certain blood parameters, kidney functions and oxidative stress

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Abstract. Lead is added to the paint to improve its dryness, durability, and resistance to corrosion in addition to adding color. One recognized occupational toxin is lead Increased exposure to lead can have negative health impacts on the body, such as liver, kidney, and nervous system damage. Anemia, hypertension, and a host of other illnesses can also be brought on by it. Many blood indicators, antioxidants, and kidney function of lead-exposed workers were studied in this study. A control group of forty healthy individuals who were not exposed to lead at work and fifty men between the ages of 18 and 50 who work in paint-related professions were chosen. In comparison to the control group, the results indicated an increase in the number of white blood cells and a reduction in the number of red blood cells. We found no difference in the amount of creatinine in the blood of the workers and the control group, except for a rise in the percentage of urea in the blood of the workers compared to the group that was not exposed when conducting renal functions. The results also revealed a significant rise in the levels of both (MDA and CAT).

1 Introduction

Paints are complicated mixes of solvents and metals that might cause health problems in those who are exposed to them [1]. Paint is enriched with lead not only to provide color but also to promote drying, durability, and resistance to corrosion. Many lead compounds, including lead oxide, lead carbonate, sometimes referred to as white lead, and lead chromate, are utilized as pigments in paints [2]. One of the first metals that humans have ever found, lead is present in everything. Lead has been used extensively in a variety of sectors, including paint, ceramics, plastics, and others, due to its special qualities, which include softness, high ductility, low melting point, and corrosion resistance Lead is a well-known occupational toxin with strong. toxicological effects. Lead's extended environmental persistence can be attributed mostly to its non-biodegradable nature [3]. High lead exposure can have detrimental effects on the body, including damage to the kidneys, liver, and nervous system; it can also cause anemia, hypertension, cardiovascular disease, immune system deficiencies, infertility, and developmental issues like learning disabilities, memory loss, and cognitive impairments [4]. The kidneys and liver are where the majority of lead is first deposited, and

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this might result in pathological alterations in these organs. A decrease in the quantity and number of renal glomeruli as well as chronic kidney disease can be brought on by prolonged lead poisoning [5, 6]. Lead also damages the liver's cells and tissues through oxidation [7]. Lead exposure is another major factor affecting blood and therefore human health. Since blood is a common site of lead poisoning, it exhibits significant changes in a number of hematological characteristics that may serve as biomarkers for the condition. Higher than 10 µg/dL blood lead levels have been linked to a number of symptoms, which are either caused by decreased heme production or by a faster rate of blood cell lysis. The significant drop in blood hemoglobin levels, hematocrit values, and normal erythrocyte transformation following lead exposure all attest to this. White blood cell counts often tend to rise, possibly as a result of lead's direct toxicity to leukocyte production in lymphoid organs, as well as other impacts, excessive lead levels prevent platelet aggregation in human blood [8]. Lead is hazardous to the hematologic, renal, and neurological systems in a dose-dependent and varied manner. Chronic lead exposure can cause developmental delays and acute toxic encephalopathy when blood levels are very high (such as greater than 4.80 mol/L). However, most individuals with increased lead levels are either asymptomatic or have a range of nonspecific symptoms such as anorexia, vomiting, or stomach discomfort [9]. In a lot of developing nations lead-based paints are still produced in large quantities [10]. For example, in China, which is the world's biggest producer and user of paints [11]. Even though lead in paint has been subject to a regulatory restriction since 1986, lead-based paints are still commonly accessible in the market and frequently have excessive lead levels [12]. The GAELP wants to eradicate lead-based paint worldwide, however there are still numerous obstacles in the way of this aim, despite international efforts to create regulatory standards. These obstacles, together with a lack of political will in affluent countries to support such programs in poor countries with technical aid, might result in disappointment if left unchecked [13]. Furthermore, lead exposure from numerous sources, such as paints, produces oxidative stress, which causes an imbalance between the formation and elimination of ROS (interactive oxygen types) in tissues and cellular components, causing damage to membranes, DNA, and proteins [14].

2 Method

This study was conducted on 90 male volunteers. The volunteers were divided into two groups. The first group included 50 paint workers, and the second group included 40 non-workers (the control group). The ages of the volunteers ranged from (19-50) years, and the duration of exposure to the workers ranged from (4-11) years, according to a questionnaire distributed to all volunteers, as shown in Table (1). The method of collecting samples was by taking an amount of (5 ml) of blood from all members of the study sample using special tubes for each purpose in accordance with standard laboratory conditions. These tests included measuring lead in some blood indicators including (WBC, HGB, MCV, RBC) according to [15], kidney function measurements (CREATININ, UREA) according to [16], and antioxidants (CAT, MDA) according to [17, 18].

Table 1. Information about the participants.

Age ?	
What is your highest level of education ?	
Weight ?	
Height ?	
Hours of Work ?	
years of Work ?	
Do you have a chronic diseases ?	

Have you smoked in your entire life ?	

2.1 Analytical statistics

SPSS software (V.27, SPSS Inc., Chicago, IL, USA) was used to analyze the data. The continuous variables were represented by the mean ± SD standard division, whereas the numerical variables were given as a number (%). Kolmogorov-Smirnov test was performed to evaluate the data's normal distribution, and independent t-test was employed to compare the two groups. One-way analysis of variance (ANOVA) and Tukey's post hoc test were utilized for multiple comparisons. Pearson correlation analysis of the factors under study. A P-value of less than 0.05 was deemed statistically significant [19].

3 Results and discussion

Table 2. Comparison of Hematology parameters between Pb Exposure workers and controls group.

Variables	Workers n=50	Control n=40	p-value
Age (year)	30.8±8.66	27.25±8.53	0.060 NS
Exposure period	10.9±4.86	0±0	NA
WBC (10^9/L)	8.76±1.78	7.56±0.64	0.0001*
RBC (10^12/L)	4.88±0.6	5.12±0.41	0.026*
MCV (FL)	85.53±4.32	87.18±1.76	0.017*
HGB (g/L)	13.25±1.03	13.63±0.4	0.021*

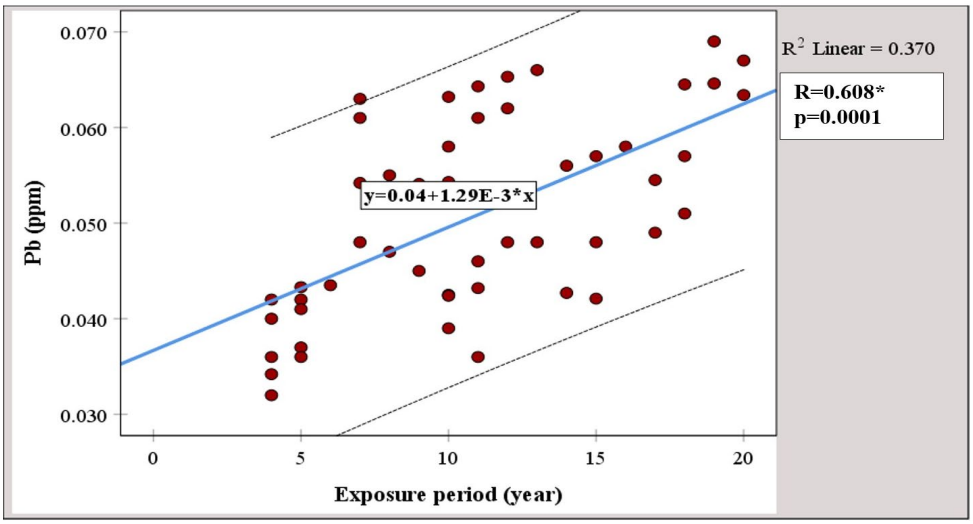

Fig. 1. Positive correlation between Pb (ppm) with exposure period (year) levels in worker groups

The results of measurements of some blood indicators in Table (2) showed that workers who were exposed to lead-based paints had a significant increase ($p<0.05$) in white blood cells compared to the control group. The results also showed a significant decrease ($P<0.05$) in RBC, HGB, and MCV in the control group. Therefore, in this study, we measured the percentage of lead in some blood indicators, and the results of the statistical analysis in Table (2) showed a significant increase in the white blood cells of the workers compared to the control group. The increase in the number of white blood cells in workers can be explained by the fact that the primary function of these cells is to provide the primary defense or immune response against foreign metals or chemicals entering the body. This result is also

consistent with the study conducted by Ercal, et al [20] on paint workers, where they found an increase in the number of white blood cells in the workers' blood and explained that this increase is due to allergic reactions secondary to occupational exposure to lead and paint materials, and this plays an important role in chronic inflammation. The results of the current study are also consistent with the study conducted by El-Gharabawy, et al [21] on a number of paint workers. They found a noticeable increase in the number in (WBC) and explained this increase as a result of exposure to paint materials, which leads to the release of histamine (the production of an inflammatory mediator from mast cells, which leads to an increase in the number of WBC in the blood. From Table (2), we notice a significant decrease in red blood cells and hemoglobin in the blood of paint workers compared to the control group. We suggest that the reason for the decrease is due to a decrease in the bone marrow that produces red blood cells and as a result of a deficiency of RBC, leading to a deficiency of HGB in the workers' blood. The results of the current study do not agree with the results of the study conducted by Amina and Abdennour [22] on paint workers, as they did not notice a decrease in red blood cells and hemoglobin, and they interpreted this result as despite the toxicity of the paint, but this toxicity does not appear until after ten years of exposure, which indicates the presence of paint toxicity and its mechanism This toxicity comes from the blood-forming organs (the bone marrow). Also, the current study, we notice a significant decrease in the MCV of the workers compared to the control group. We explain this decrease due to a lack of iron in the body due to exposure to paints containing lead. This study is consistent with a study conducted by Chwalba et al [23] on workers exposed to lead, who found that lead affects and reduces the volume of red blood cells (MCV).

Table 3. Comparison of kidney function parameters between workers exposed to lead paint and the control group.

Variables	Control n=40	Workers n=50	p-value
Creatinine (mg/dl)	0.71±0.12	0.76±0.16	0.096 NS
Urea (mg/dl)	27.93±2.92	30.95±7.26	0.009*

The kidney function measures shown in Table (3) demonstrated that there were no statistically significant ($p>0.05$) variations in the serum creatinine levels between the control group and workers. In contrast to the control group, there was a notable rise in urea levels among the workers. Therefore, in order to ascertain the impact of lead paint exposure on workers, we examined kidney function in this study. Based on the statistical data, we found that there was no change marked in the creatinine concentration in the workers' serum when compared to the control group (table 3). The current research's findings are in line with a study conducted by Bagepally et al [24] on 97 lead-exposed workers in a battery factory, whereby they observed no differences in the concentration of kryatin between the exposed group and the control group. As we can see from Table (3), which presents the study's findings, the workers' blood urea levels were significantly higher than those of the control group.We propose that tubulointerstitial inflammation among workers as a result of lead exposure from paints is the cause of the rise. The present investigation corroborates the findings of Nakhaee et al, [25], who examined 100 individuals employed in lead-containing paint-related fields and discovered a higher level of urea in the blood of the workers than in the control group. In an identical study, Orisakwi et al, [26] examined 25 individuals employed in the lead paint industry and 25 individuals who were not exposed, serving as the control group. Their findings indicated that there was no rise in creatinine levels in the workers' blood when compared to the control group, which aligns with the current study's findings; however, they did not find an increase in urea levels in the workers' blood, despite a notable increase in lead concentration in the workers' blood, which is inconsistent with the current study's findings.

Table 4. Comparison of antioxidant parameters between workers exposed to lead-containing paint and a control group.

Variables	Control n=40	Workers n=50	p-value
MDA (umol/l)	121.04±9.11	140.29±13.97	0.0001*
CAT (u/ml)	11.57±2.26	109.35±34.03	0.0001*

The antioxidant measures shown in Table (4), show a highly significant (P>0.05) increase in MDA and CAT in lead-exposed workers compared to the control group.

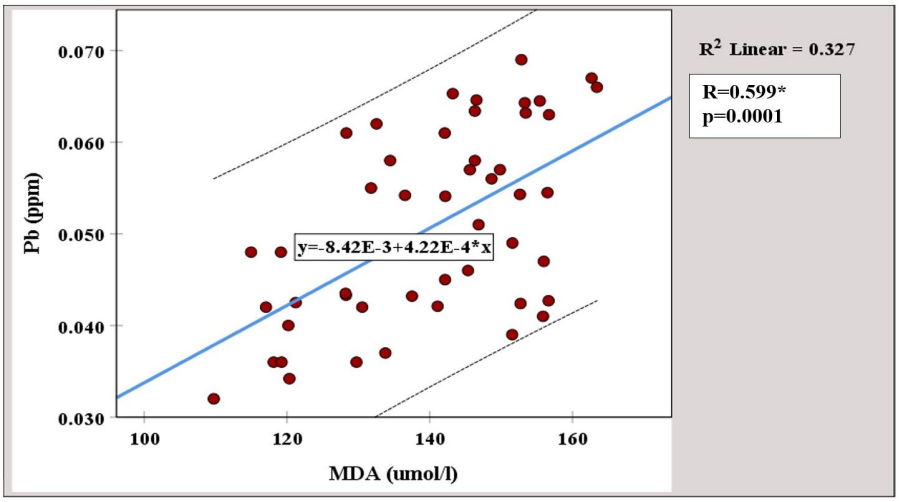

Fig. 2. Positive correlation between Pb (ppm) with MDA (umol/l) levels in worker groups.

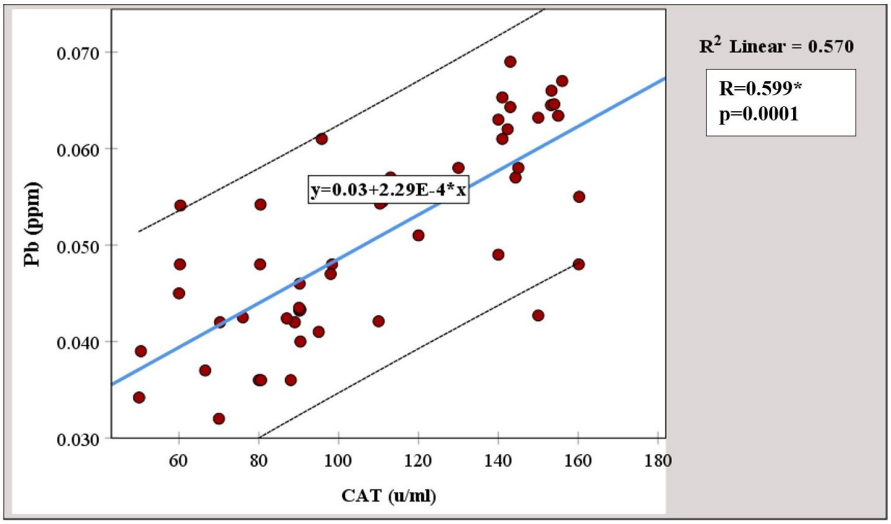

Fig. 3. Positive correlation between Pb (ppm) with CAT (u/ml) levels in worker groups.

The current study's statistical results, as shown in Table (4), demonstrate a significant increase in MDA and CAT among workers exposed to lead-containing paints as compared to the control group. The explanation for the substantial rise, we believe, is that lead in paints causes free radicals to develop, resulting in oxidative damage. Reactive oxygen species are produced by lead, which weakens antioxidant cell defenses. Additionally, it can alter the content of fatty acids and membrane integrity, deplete glutathione, interfere with some

essential metals required for antioxidant enzyme activities, and/or increase cell vulnerability to oxidative assault [27, 28]. The present research's findings are in line with another study by Muhammad et al, [29] that involved 56 individuals with lead paint-related jobs and a comparable number of non-workers who served as the control group. The required laboratory analyses were carried out for many antioxidants, including catalase (CAT), and their findings indicated that exposure to lead from paints caused a significant rise in catalase levels relative to the control group.

Table (4) exhibits a noteworthy rise in MDA, as per the statistical findings. We see this rise as potentially resulting from exposure to lead-based paints, which inhibit the action of antioxidant enzymes and deplete substrates like glutathione. may increase reactive oxygen species (ROS) concentrations [30]. The current research's findings are congruent with those of a similar study done by Madhavi and Devi [31] on 67 workers and 50 healthy adults. When compared to the control group, the workers' MDA % increased, which they attributed to the existence of oxidative stress caused by lead exposure.

4 Conclusion

The current investigation discovered a considerable rise in oxidative stress as well as an increase in the proportion of urea in the blood of lead paint workers. As a result, it is vital to phase out lead-based paints in large quantities due to their detrimental and cumulative effects on worker health, and to take the required precautions to ensure worker safety. Workers must also be made aware of the dangers and toxicity of lead in order for them to take the required measures, such as wearing masks and gloves, washing their hands thoroughly before eating, and not working in confined places.

References

1. C. Cassini, C. Calloni, G. Bortolini, S.C. Garcia, M.A. Dornelles, J.A. Henriques, B. Erdtmann, M. Salvador, International journal of occupational medicine and environmental health, **24,** 308-19 (2011)
2. A. Kumar, & P. Gottesfeld, Science of the Total Environment, **407(1),** 333-337 (2008)
3. G. Flora, D. Gupta, & A. Tiwari, Interdisciplinary toxicology, **5(2),** 47-58 (2012)
4. A. E. Nigra, A. Ruiz-Hernandez, J. Redon, A. Navas-Acien, & M. Tellez-Plaza, Current environmental health reports, **3**, 416-433 (2016)
5. M. Kshirsagar, J. Patil, A. Patil, G. Ghanwat, A. Sontakke, & R. K. Ayachit, Al Ameen J Med Sci, **8(2),** 107-114 (2015)
6. M. C. Buser, S. Z. Ingber, N. Raines, D. A. Fowler & F. Scinicariello, International journal of hygiene and environmental health, **219(3),** 261-267 (2016)
7. B. Dadpour, R. Afshari, S. R. Mousavi, S. Kianoush, M. R. Keramati, V. A. Moradi, ... & M. Balali Mood, Iranian Journal of Toxicology, **10(2),** 1-6 (2016)
8. R. R. Ray, Proceedings of the Zoological Society, Springer India, 69 161-172 (2016)
9. P. M. Lavoie, & B. Bailey, CMAJ, **170(6),** 956-956 (2004)
10. H.M.R. Alhasnawi, Ahmed Abduljabbar Jaloob Aljanaby, Gene Reports, **26,** 101520 (2022) https://doi.org/10.1016/j.genrep.2022.101520.
11. S. Y. Wu, J. Ye, Y. Tian, Y. J. Wang, & X. D. Jian, *The current status of lead paint control in China and suggestions*, Modern Chemical Industry (2017)
12. D. O'Connor, D. Hou, J. Ye, Y. Zhang, Y. S. Ok, Y. Song, ... & L. Tian, Environment international, **121**, 85-101 (2018)

13. Q. Tan, & J. Li, Nature, **547(7662)**, 162-162 (2017)
14. R. C. Patra, A. K. Rautray, & D. Swarup, *Oxidative stress in lead and cadmium toxicity and its amelioration*, Veterinary medicine international (2011)
15. B. M. Muhammad-Baqir, A. A. Fattah, D. R. A. Al-Janabi, & A. A. J. Aljanaby, BIO Web of Conferences, **65**, 05049 (2023)
16. D.R.A. Al-Janabi, and A.A.J. Aljanaby, BIO Web of Conferences, **84**, 03014 (2024)
17. B. Mayer, M. Schumacher, H. Brandstätter, F. S. Wagner & A. Hermetter, Analytical biochemistry, **297(2),** 144-153 (2001)
18. J. A. Buege, Methods in enzymology, **52**, 302-310 (1978)
19. L. M. Sullivan, *Essentials of biostatistics in public health*, Jones & Bartlett Learning (2017)
20. N. Ercal, R. Neal, P. Treeratphan, P. M. Lutz, T. C. Hammond, P. A. Dennery & D. R. Spitz, Archives of environmental contamination and toxicology, **39**, 251-256 (2000)
21. R. M. El-Gharabawy, E. I. El-Maddah, M. M. Oreby, H. S. Salem, & M. O. Ramadan, Journal of immunotoxicology, **10(3)**, 270-278 (2013)
22. M. Amina, & C. Abdennour, Advances in Environmental Biology, **8(24),** 37-42 (2014)
23. A. Chwalba, B. Maksym, M. Dobrakowski, S. Kasperczyk, N. Pawlas, E. Birkner, & A. Kasperczyk, Toxicology and applied pharmacology, **355**, 174-179 (2018)
24. I. P. Odigie, C. O. Ladipo, R. R. Ettarh, & M. C. Izegbu, Nigerian Journal of Physiological Sciences, **19(1)**, 27-32 (2004)
25. B. S. Bagepally, R. Kalahasthi, & T. Barman, Journal of Molecular Pathophysiology, **5**, 1-7 (2016)
26. S. Nakhaee, A. Amirabadizadeh, J. Brent, & O. Mehrpour, Basic & clinical pharmacology & toxicology, **124(5),** 621-628 (2019)
27. O. E. Orisakwe, E. Nwachukwu, H. B. Osadolor, O. J. Afonne, & C. E. Okocha, Toxicology and industrial health, **23(3),** 161-165 (2007)
28. F. Farmand, A. Ehdaie, C. K. Roberts, & R. K. Sindhu, Environmental Research, **98(1),** 33-39 (2005)
29. L. Cabell, C. Ferguson, D. Luginbill, M. Kern, A. Weingart & G. Audesirk, Toxicology and applied pharmacology, **198(1),** 49-60 (2004)
30. I. K. Mohammad, A. A. Mahdi, A. Raviraja, I. Najmul, A. Iqbal & V. Thuppil, Arhiv za higijenu rada i toksikologiju, **59(3),** 161 (2008)
31. M. Hermes-Lima, V. G. Valle, A. E. Vercesi & E. J. Bechara, Biochimica et Biophysica Acta (BBA)-Bioenergetics, **1056(1)**, 57-63 (1991)