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Research Article

Effect of lead on hematological parameters and histopathology of the liver, kidney and spleen of female albino mice

Nguyen Thi Thuong Huyen[1], Truong Nguyen Lan Chi[2], Truong Van Tri[1], Nguyen Thi Thanh Hang[1], Thai Ke Quan[3,*]

ABSTRACT

Objectives: The objective of this study was to examine the influence of lead toxicity on the weight gain, blood cell count, blood biochemical parameters and histopathological perspective of the liver, kidney and spleen of mice. **Methods**: Sixty female mice (7 weeks old) were randomly divided into 5 groups: Group 1 — control 1 (W): received only water without chlorine, group 2 — control 2 (N): received nitrate (from potassium nitrate); group 3-5 (L5, L7, L9): received lead from lead (II) nitrate (50; 70 and 90 mg Pb/kg bw, respectively). All groups (2, 3, 4, 5) were administered nitrate or lead once daily. Half of the mice in each group were sacrificed after 4 weeks and 8 weeks. Blood and serum samples were collected to run the complete blood count and biochemical parameter analysis. At the same time, the liver, kidneys and spleen were collected to analyze the histological structure and lead accumulation. Once daily, they drank water without chlorine ad libitum. Each mouse was weighed every two weeks under overnight fasting conditions to assess its weight gain. **Results:** The results showed that mice exposed to lead concentrations (50, 70 and 90 mg/kg bw) for 4 and 8 weeks experienced reduced weight gain, decreased radius and quantity of red blood cells, and an increase in the number of white blood cells and platelets; the AST, ALT, and BUN indices showed a tendency to decrease, whereas the Creatine index showed an inclination to increase; the liver and kidney tissue structures were damaged, and the spleen tissue exhibited the invasion of white pulp into the red pulp. The concentration of 90 mg/kg bw had the most significant effect among the three concentrations tested. **Conclusions:** This study provides evidence that exposure of mice to a lead concentration of 90 mg/kg bw can have various negative effects on their health, including reduced weight gain, changes in blood cell counts, altered biochemical parameters, and liver and kidney tissue damage. These findings highlight the negative impact of lead exposure on overall health and suggest the need for further investigation and preventative measures.

Key words: albino mice, hematological parameters, histological structure, lead, toxicity.

[1]*Ho Chi Minh City University of Education, Department of Biology, Viet Nam*

[2]*Hung Vuong High School for the Gifted, Binh Duong Province, Viet Nam*

[3]*Saigon University, Ho Chi Minh City, Viet Nam*

Correspondence

Thai Ke Quan, Saigon University, Ho Chi Minh City, Viet Nam

Email: tkquan@sgu.edu.vn

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INTRODUCTION

Lead is a commonly used element in various industries, such as petroleum production, paint, plastic, and auto mechanics. Lead is a stable metal in the environment that can exist in the form of ions and inorganic and organic compounds[1]. Lead exposure mainly occurs through the respiratory and gastrointestinal tracts[2]. Lead has the characteristic of bioaccumulation in living organisms because it is not used in metabolism. In humans, chronic lead poisoning is more common[3]. Lead is a multiorgan toxicant and can cause irreversible damage to the histopathological structure and function of lead-accumulated organs. Some studies have linked lead poisoning to several diseases of the heart[4–6], liver and kidney[5,6], spleen[5], and central and peripheral nervous system[3]. The burden of health problems, disability and healthy life lost on people with lead exposure is measurable and substantial[7,8].

Currently, more researchers have investigated the toxic effects of lead and developed natural ingredients that have the potential to counteract those toxic effects or improve the health consequences of lead exposure in humans (such as vitamins B, C, and E, flavonoids, and herbal antioxidants[3]). Many studies have reported decreases in red blood cell count (RBC), Hb, and MCH in both mouse and rat models[9,10]. The white blood cell count (WBC) increased in lead-exposed mice[9,11]; however, some studies noted a decrease in WBC[12]. The white blood cell proportion varies depending on the study[13–15]. Some studies recorded an increase in peripheral blood platelet counts[9,15,16]. The spleens of lead-exposed mice had enlarged white pulp[13], lymphoid follicular hyperplasia, and increased red blood cell breakdown in red pulp[14]. For the liver, AST and ALT activities are increased[11,17], markedly in the chronic state[12]. MDA levels significantly increased in lead-exposed

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mice[18]. Common structural damage to liver tissue includes shrinking of hepatocytes[19], foamy degeneration[17,20], necrosis[20,21], apoptosis[22], and a slight increase in the invasion of inflammatory cells[23,24]. The kidneys of lead-exposed rats resulted in a gradual decrease in the activity of the SOD and CAT enzymes over time[22,25]. However, the creatinine level was not different from that of the control group[23]. Structural damage to the renal tissue was mostly observed in the renal tubules, cortical interstitial cells, and renal parenchyma: increased lymphatic permeability[4,23], congestion[10], some hyperplasia tubules with cell aggregation[22], necrosis, and hemorrhage[20].

There are few studies evaluating the toxic effects of lead on hematological and biochemical parameters and the histological structure of organs related to those parameters at the same time. Therefore, this study aims to further investigate the effects of different lead concentrations on albino mice when simultaneously considering complete blood count, some biochemical parameters, and the histological structure of the liver, kidneys and spleen. Then, we aimed to find a suitable concentration of lead that can give clear effects on albino mice in the above criteria simultaneously.

METHODS

Chemicals

Lead nitrate (Merck, code. L/1500/50) was used for oral administration solutions.

Animals

Female albino mice (5 weeks old) were purchased from the Pasteur Institute (Ho Chi Minh City, Vietnam). Mice were maintained at a room temperature of 28°C, humidity of 55-70%, and 12-hour light/dark cycle and allowed access to food pellets and water without chlorine ad libitum for 2 weeks. Mices were placed in glass cages (30x19x19 cm) lined by rice husks at the bottom of the cage. All experimental procedures were approved by the Ethical Committee on Animal Experimentation of the University of Science, VNUHCM (ACUCUS, No. 1170B/KHTN-ACUCUS). We have made all efforts to minimize mouse suffering.

Experimental design

The lead concentration that can be acutely toxic to an albino mouse is 2.42 mM (equivalent to 500 mg/kg bw)[26]. The chronic toxicity concentration was chosen to be 1/10 of the acute concentration (50 mg/kg bw). Three concentrations (50, 70 and 90 mg/kg bw) were chosen to examine our experience.

After two weeks of stabilization and acclimation in the laboratory, sixty female mice were randomly divided into 5 groups: two control groups and three experimental groups. Group 1 — control 1 (W): received only water without chlorine, group 2 — control 2 (N): received nitrate (from potassium nitrate) equal to the amount of nitrate present in lead nitrate with a lead concentration of 90 mg/kg bw; group 3-5 (L5, L7, L9): received lead from lead (II) nitrate (code. L/1500/50) (50; 70 and 90 mg Pb/kg b.w), respectively. All groups (2, 3, 4, 5) were administered nitrate or lead once daily (30 minutes before breakfast) by gavage with an 8-needle syringe (1 mL).

Half of the mice in each group were sacrificed after 4 weeks and 8 weeks. Blood and serum samples were collected to run the complete blood count and biochemical parameter analysis. At the same time, the liver, kidneys and spleen were collected to analyze the histological structure and lead accumulation. Once daily, they drank water without chlorine ad libitum. Each mouse was weighed every two weeks under overnight fasting conditions to assess its weight gain.

Hematological Analysis

For hematological investigation, blood was collected from the tail vein of each mouse individually. Mice were fasted for 8-12 hours before blood collection. The number of blood cells (red blood cells (RBCs) and white blood cells (WBCs)) was determined by direct counting in the cell counting chamber. Platelet (PLT) count was determined by the Fonio method. The size of RBCs was measured directly on the Giemsa-stained blood smear (100 RBCs/slide, 3 slides/mouse and 12 mice/group at baseline and after 4 weeks of the experiment = 3600 RBCs/group; but only 6 mice/group after 8 weeks = 1800 RBCs, randomly) by S-EYE application software.

Blood biochemical investigation

For biochemical investigation, mice were fasted for 8-12 hours before blood collection. Blood was collected from the retro-orbital sinus vein by hematocrit capillary tubes after anesthetic administration. The appropriate volume of blood was collected and transferred to a 1.5 mL Eppendorf tube without anticoagulant to obtain serum. After resting for 30 minutes, the blood sample was centrifuged at 2400 rpm and 27°C for 30 minutes to obtain the serum for biochemical investigation. Serum was stored in the freezer (-4°C) until analysis.

These parameters, including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), were analyzed by the enzymatic method: NADH (No. P-5'-P). Creatinine (CRE) levels were measured by the kinetic alkaline picrate method, while blood urea nitrogen (BUN) levels were determined by the urease method.

Tissue Preparations and Histological Analysis

At the end of blood collection, the mice were killed by cervical dislocation. The liver, kidneys and spleen from each mouse were quickly removed and washed in 10% formaldehyde solution with KH_2PO_4 (4 g/1000 mL) and Na_2HPO_4 (6.5 g/1000 mL). Then, they were fixed in 10% formaldehyde solution with KH_2PO_4 (4 g/1000 mL) and Na_2HPO_4 (6.5 g/1000 mL) for hematoxylin and eosin (H&E) staining. Each slide was analyzed for tissue damage by visually examining images using a Nikon microscope (TiU, Nikon).

Staticstical Analysis

The data were processed with Minitab 18 software. The overall differences among groups were compared by one-way ANOVA (followed by Tukey's test) and two-way ANOVA. *p* values less than 0.05 were considered significant. Data are presented as the mean ± 95% CI.

RESULTS

Body weight gain

During the first 4 weeks, the weight gains increased rapidly and not significantly in the control groups (W and N; $p > 0.05$). Meanwhile, in the lead exposure groups (L5, L7 and L9), the weight gains were slowly and plummeted compared to both control groups ($p < 0.01$) but were not significant between the L7 and L9 groups ($p > 0.05$) (**Figure 1**A, B). The weight gains in the lead exposure groups (L5, L7 and L9) continued to decrease sharply compared to the two control groups ($p < 0.01$); however, there was no significant difference between the two control groups ($p > 0.05$) at the 6^{th} and 8^{th} weeks of the study (**Figure 1**C, D). The study found a correlation between lead concentration and weight gain, with weight gain decreasing gradually as lead dose increased across the three concentrations examined. The most significant impact was observed in the L9 group (90 mg/kg bw).

Hematological Analysis

The results in Table 1 indicated that the RBC, WBC and PLT counts remained flat in the W and N groups ($p > 0.05$); meanwhile, they were significantly different in all lead exposure groups (L5, L7 and L9; $p < 0.01$). The RBC count decreased slightly when the lead concentration and time experiment increased ($p < 0.01$), especially plummeting in the L7 group (70 mg Pb/kg bw). In contrast, the WBC and PLT counts increased when the lead concentration and time experiment increased, respectively ($p < 0.01$), especially the WBC count in the L7 group (70 mg Pb/kg bw), while the PLT count in the L9 group (90 mg Pb/kg bw).

When statistically analyzing the correlation between treatment time and lead concentration, the results showed that each experimental lead concentration and the time of treatment affected RBCs, WBCs and PLTs ($p < 0.01$). This proved that the longer the duration of lead exposure was, the more obvious the difference in RBCs, WBCs and PLTs of mice between groups was.

The radius of RBCs in the control groups remained relatively flat at survey timelines (week 0, week 4 and week 8 ($p > 0.05$). Meanwhile, the radius of RBCs in all lead exposure groups (L5, L7 and L9) was reduced compared to that in the control groups (W and N) at week 4 and week 8 ($p < 0.01$). Moreover, the radius of RBCs decreased more when the lead concentration and time experiment increased ($p < 0.01$). In particular, in group L9 (90 mg lead/kg bw), the radius of RBCs was the smallest ($p < 0.01$). This means that the RBC size tended to shrink under the effect of lead, in which the lead concentration of 90 mg/kg bw had the greatest decrease in size.

Blood biochemical Assays

The biochemical parameters are shown in **Figure 2**. The AST level in the L7 and L9 groups decreased slightly in both experimental weeks 4 and 8, while this parameter in the L5 group tended to increase compared to the two control groups. However, this difference was not statistically significant ($p > 0.05$) compared to the control groups as well as among all exposed lead groups, except for the L7 group, which had AST levels that decreased in week 8 compared to week 4 ($p = 0.018$), and this group also had the lowest AST level of the three groups drinking lead. Similarly, the ALT level in the L7 group was the lowest among the 5 experimental groups in general and all 3 groups receiving lead in particular, but this difference was not statistically significant ($p > 0.05$).

The BUN level decreased slightly in the three groups receiving lead compared to the two control groups in both experimental week 4 and week 8, but the difference was not statistically significant ($p > 0.05$). However, the BUN levels in the L7 and L9 groups at week 8

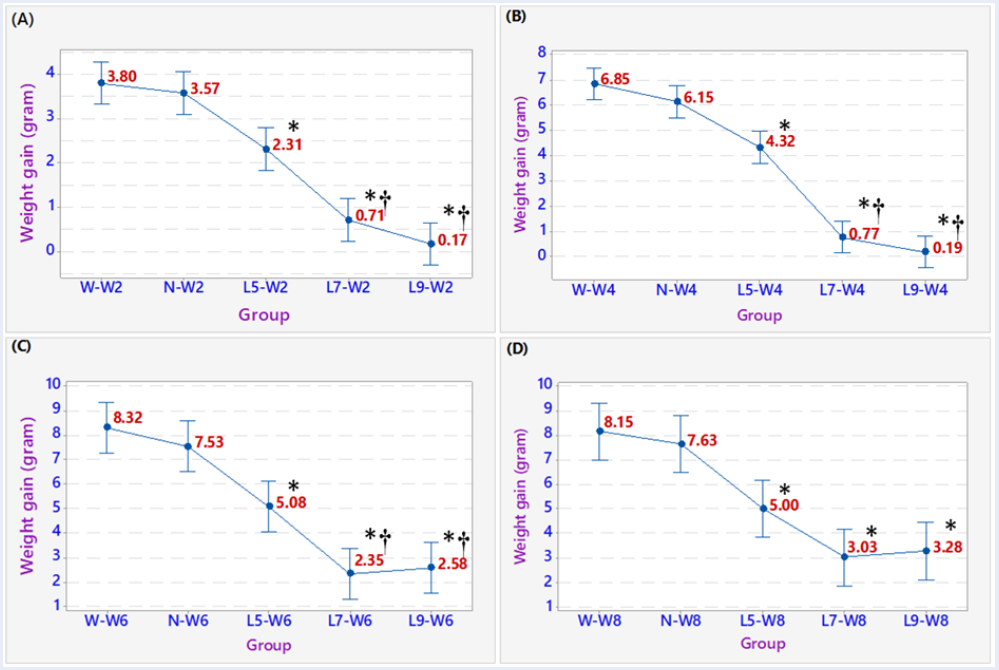

Figure 1: **The weight gain of mice after every 2 weeks**. **A**: after 2 weeks; **B**: after 4 weeks; **C**: after 6 weeks; **D**: after 8 weeks. (*): $p < 0.01$ compared to control groups; : $p < 0.01$ compared to group L5. Data are represented as the mean ± 95% CI, n = 12

were lower than those at week 4. Meanwhile, the CRE level in the three groups receiving lead was equivalent to that in the two control groups at week 4 ($p > 0.05$); at week 8, the CRE level in the L5 and L7 groups was still equivalent to that at week 4 ($p > 0.05$), while this level in the L9 group increased compared to that at week 4 ($p < 0.05$).

Histological structure analysis

The liver tissue structure in mice of five experimental groups exhibited tissue damage after 4 and 8 weeks of the experiment (**Figure 3**). These damages included portal inflammation, foci of inflammation, necrosis, apoptosis, and degeneration of hepatocytes. However, the severity of the damage varied among the five groups. The control groups (W and N) had a clear central venous area, while the lead-intake groups (L5, L7 and L9) had more obvious areas of inflammation, degeneration, and necrosis. In the two control groups, inflammatory areas appeared around the portal but did not spread beyond it, with limited infiltration of inflammatory cells in the lobules at sites of liver cell necrosis. Scattered small point necrosis was observed in the lobules, and there were signs of hepatocyte degeneration and apoptosis (**Figure 3** W, N). However, in the lead-intake groups, after 8 weeks, there was a wider spread of the area of necrosis and portal inflammation, as well as the appearance of degenerative areas. The necrotic area was heavily infiltrated by inflammatory cells; furthermore, there was widespread inflammation around the portal veins, which was accompanied by marginal necrosis; the degeneration of hepatocytes was characterized by the lack of a cell septum, the accumulation of glycogen, and the infiltration of inflammatory cells (**Figure 3** L5, L7 L9). The degenerative areas appeared in discrete, seamless or large areas. The types of damage appearing in each tissue sample are random.

The kidney tissue structure in mice differed between the groups that were given lead orally and those that were not. The control groups (W and N) had normal renal tissue structures, with visible glomeruli and tubules. Nonetheless, the renal tissue structure of the groups administered lead-drinking treatments (L5, L7, and L9) presented with apparent lesions. The renal vein was surrounded by inflammatory cells, resulting in the formation of inflammatory foci. Additionally, in groups L7 and L9, inflammatory areas spread and disrupted the tubular structure (**Figure 4** L7, L9 in the W4). Following eight weeks of experimentation, the kidney tissue in the lead-drinking groups

Table 1: Blood cell counts after 4 and 8 weeks of treatment with lead

	Group	Time point		
		Week 0	Week 4	Week 8
RBC (x10^6 cells/mm^3)	W	8.73 ± 0.18a,A	8.75 ± 0.38a,A	8.81 ± 0.14a,A
	N	8.75 ± 0.17a,A	8.78 ± 0.17a,A	8.76 ± 0.29a,A
	L5	8.74 ± 0.10a,A	8.72 ± 0.18a,A	7.39 ± 0.14bc,B
	L7	8.79 ± 0.31a,A	7,91 ± 0.30b,B	7.19 ± 0.51b,C
	L9	8.77 ± 0,21a,A	7.93 ± 0.55b,B	7.77 ± 0.37c,B
WBC (x10^3 cells/mm^3)	W	8.03 ± 0.48a,A	8.19 ± 0.18a,A	8.18 ± 0.44a,A
	N	8.04 ± 0.17a,A	8.13 ± 0.19a,A	8.09 ± 0.30a,A
	L5	8.10 ± 0.11a,A	9.41 ± 0.25b,B	9.61 ± 0.17b,B
	L7	8.02 ± 0.35a,A	8.80 ± 0.68c,B	11.80 ± 0.96c,C
	L9	8.01 ± 0.50a,A	9.60 ± 0.52b,B	10.40 ± 0.36b,B
PLT (x10^5 cells/mm^3)	W	9.76 ± 0.68a,A	9.76 ± 0.56a,A	9.81 ± 0.77a,A
	N	9.08 ± 0.15a,A	9.91 ± 0.13a,AB	10.00 ± 0.14a,B
	L5	9.74 ± 0.43a,A	12.68 ± 0.83b,B	12.56 ± 0.96b,B
	L7	9.74 ± 0.36a,A	12.85 ± 0.33bc,B	12.60 ± 0.58b,B
	L9	9.86 ± 0.18a,A	13.37 ± 0.34c,B	15.02 ± 0.29c,C
Radius of RBC (μm)	W	3.04 ± 0.16a,A	3.03 ± 0.18a,A	3.03 ± 0.17a,A
	N	3.04 ± 0.17a,A	3.03 ± 0.18a,A	3.03 ± 0.18a,A
	L5	3.05 ± 0.17a,A	2.84 ± 0.18b,B	2.83 ± 0.21b,C
	L7	3.04 ± 0.18a,A	2.85 ± 0.20b,B	2.82 ± 0.24b,C
	L9	3.04 ± 0.18a,A	2.82 ± 0.19c,B	2.79 ± 0.20c,C

^{a,b,c}: statistically significant compared in column in one group
^{A,B,C}: statistically significant compared in row

still showed extensive inflammatory areas, disrupted tubular structure, and damaged glomeruli (**Figure 4** L5, L7, L9 in the W8). The L9 group suffered the most damage out of the three lead-drinking groups.

The spleen tissue structure in mice was normal in all five groups, with distinct separation between the red pulp, white pulp, and blood vessels and no signs of damage. However, after four weeks of the experiment, the lead-drinking groups showed an invasion of the red pulp into the white pulp, which increased gradually with increasing lead concentration after eight weeks (**Figure 5** L5, L7 and L9). Meanwhile, the imaging results in the two control groups remained unchanged from the four-week mark (Figure 5 W and N). Consequently, the presence of lead resulted in an increase in the red to white pulp ratio, with the most significant effect observed in group L9 among the three lead-drinking groups.

In brief, lead exposure in mice resulted in harm to their liver and kidney tissue. Liver inflammation, degeneration, and necrosis were the most detrimental effects observed at a lead concentration of 90 mg/kg bw, while the kidneys showed the formation of inflammatory foci, extensive inflammatory areas, disrupted tubular structure, and damaged glomeruli. Additionally, mice exposed to lead exhibited red pulp invasion of white pulp in their spleen.

DISCUSSION

Effect of lead on body weight gain

The weight gain discrepancy observed between the lead group and the control group indicates that lead has an impact on the growth and weight gain of mice. These findings align with recent studies, including Alwaleedi (2016), Sun (2017), and Ibrahim (2012), which also found that the rats that drank

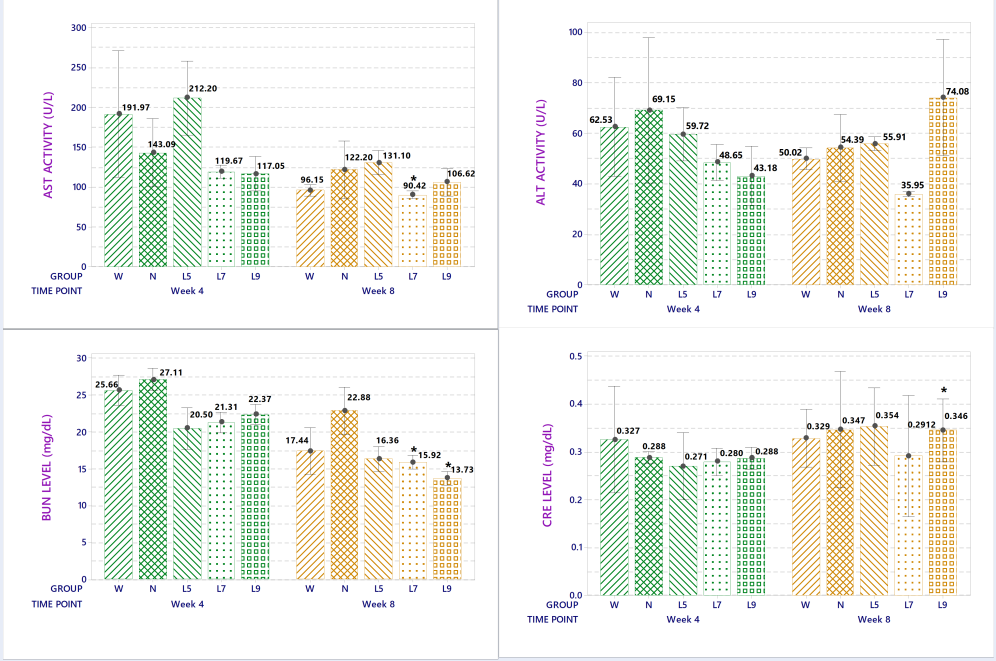

Figure 2: **AST and ALT level, BUN and CRE level after every 4 weeks.** Aspartate aminotransferase (AST), alanine aminotransferase (ALT), Blood urea nitrogen (BUN), creatinine (CRE). Data are represented as the mean ± SE, n = 3. (*): $p < 0.05$ compared to the result of the group at week 4, respectively.

lead had lower body masses than those in the control group[9,27,28]. The ability of lead to induce oxidative stress and bind to sulfhydryl groups of various enzyme systems may be the reason for the slow increase in mouse weight[5]. As a result, lead ions interfere with the absorption and metabolism of essential nutrients in mice[9].

Effect of lead on complete blood count

Studies indicate that nearly all of the lead (99%) in the body is concentrated in red blood cells, with only a small fraction (1%) present in the plasma[5]. In this study, we observed a decrease in both the number and size of red blood cells, which may be caused by intravascular hemolysis resulting from an increase in free radicals and lipid peroxidation in the blood[10]. In addition, lead can also bind to the sulfhydryl group of free radical scavenging enzymes such as SOD, GSH, and CAT, leading to reduced activity and increased levels of malondialdehyde (MDA) and H_2O_2 inside red blood cells[10,21,23]. Moreover, lead can inhibit the activity of enzymes important to heme biosynthesis, such as ALAD and ALAS, as well as GA3PD and G6PD[7,9,11,12]. Histological analysis of mouse spleens revealed that after 8 weeks of exposure, groups L5 and L7 experienced invasion of red pulp, leading to a decrease in the white pulp area and potentially contributing to the observed decrease in red blood cell count. However, there were no abnormal changes observed in the spleen of group L9 after 4 or 8 weeks of exposure, suggesting that the primary cause of the decrease in red blood cell count was likely intravascular hemolysis resulting from lead exposure.

The number of WBCs in mice taking lead generally increased over time, which could be due to their inflammatory response to lead exposure or increased natural WBC production in lymphatic organs[9,15]. Lead exposure can also lead to increased oxidative stress, resulting in the mobilization of proinflammatory cytokines and MDA for the proinflammatory response[29]. Therefore, the increase in mouse WBC count may be due to the mouse's general inflammatory response rather than inflammation caused by lead accumulation in the organs. This response could activate the defense mechanism against oxidative stress caused by long-term lead exposure. This study did not determine the peripheral WBC proportion of mice. Future studies should analyze the WBC proportion to strengthen the hypothesis of the proliferation of proinflammatory cytokines.

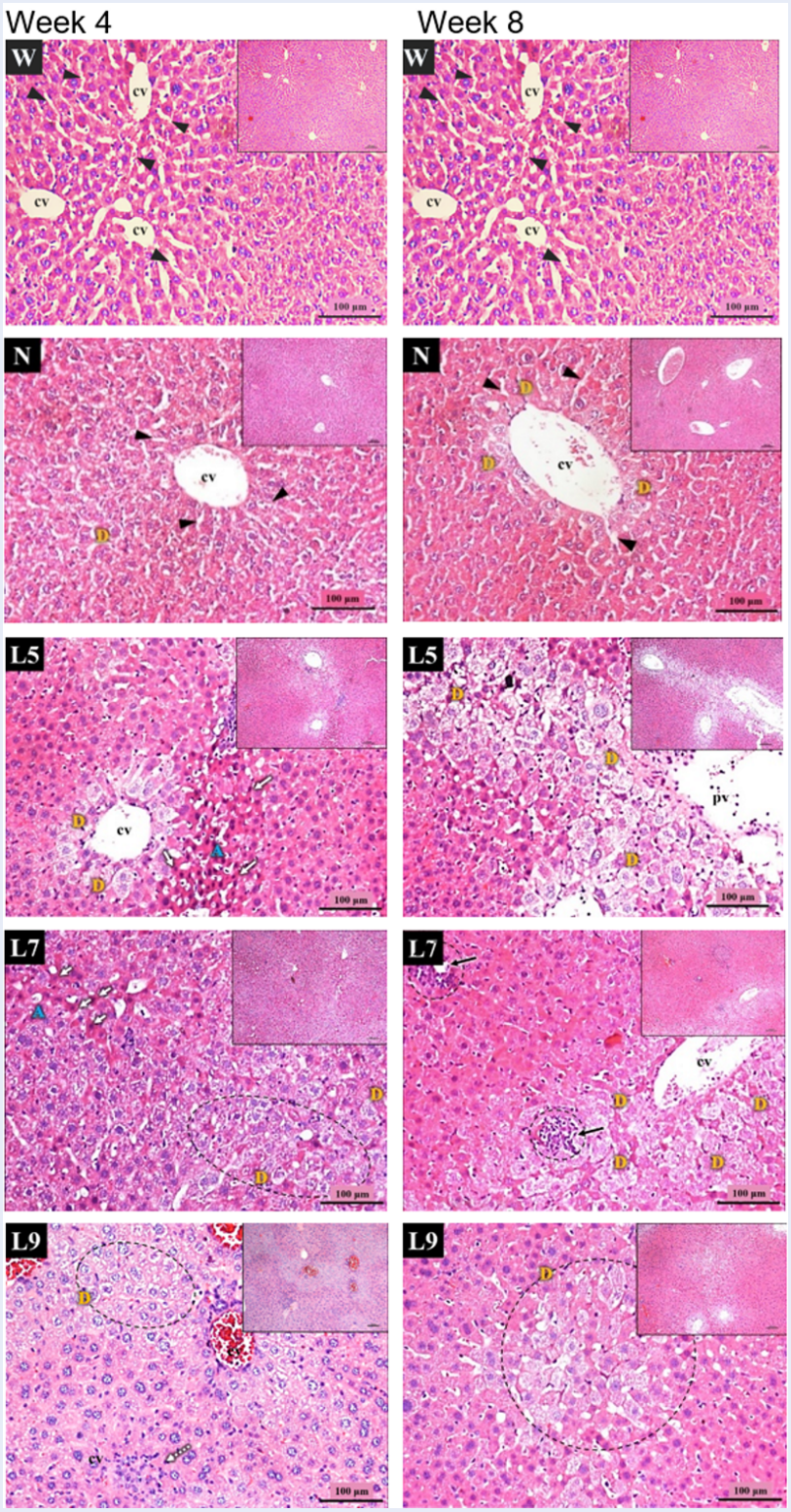

Figure 3: **Effect of lead on microstructures of mice liver after every 4 weeks exposure (x20).** scale bar 100 μm
W: control 1; N: control 2; L5: received 50 mg Pb/kg bw; L7: received 70 mg Pb/kg bw; L9: received 90 mg Pb/kg bw; cv: central vein; pv: portal vein; D: degeneration; A: apoptosis; Black arrow: necrosis; Asterisk: inflammation

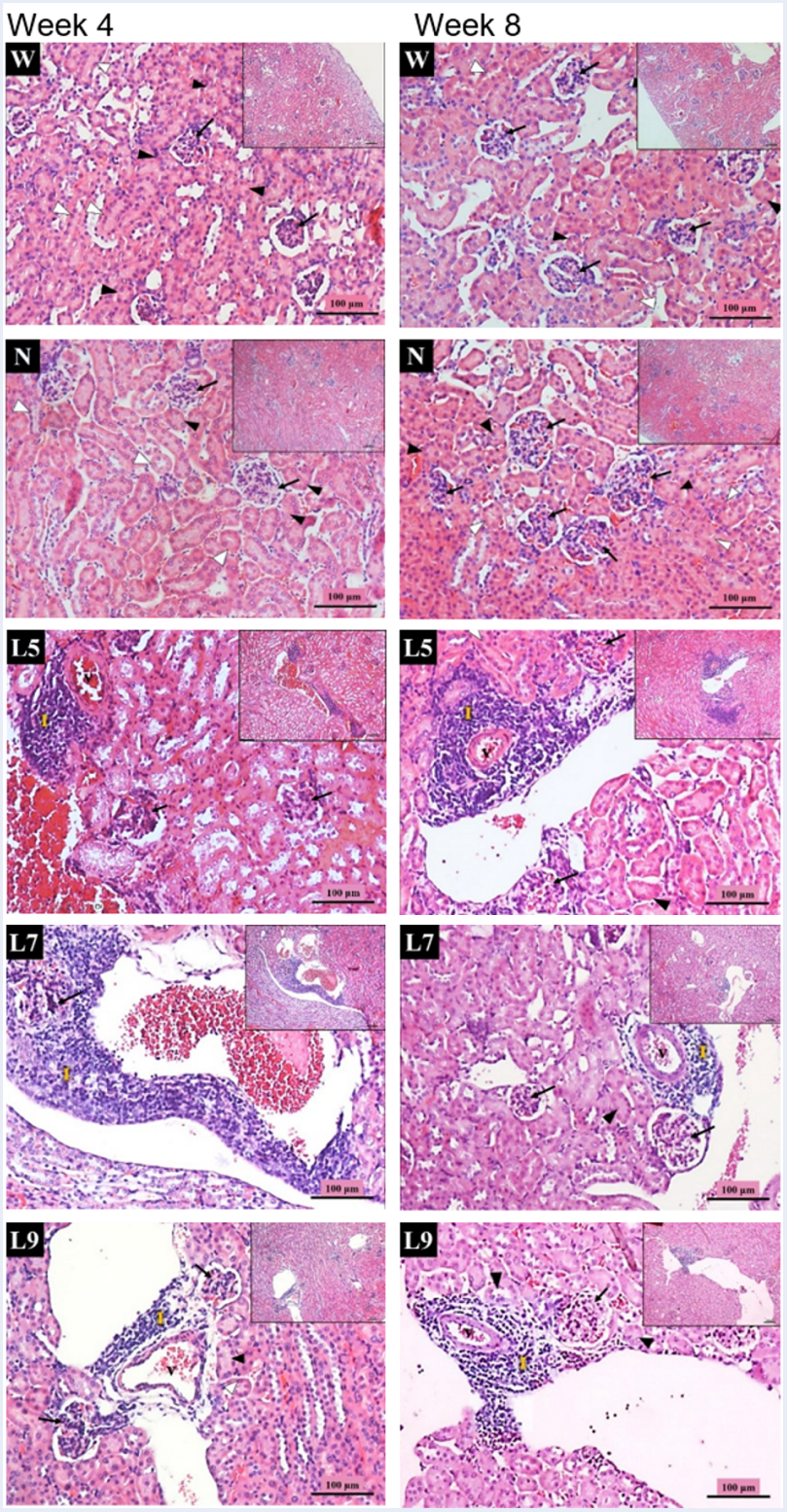

Figure 4: **The histological structure of mouse kidneys tissue after every 4 weeks (x20), scale bar 100 μm**. W: control 1; N: control 2; L5: received 50 mg Pb/kg bw; L7: received 70 mg Pb/kg bw; L9: received 90 mg Pb/kg bw; Arrow: glomerulus; Black arrow head: proximal tublule; White arrow head: distal tubule; V: vein; I: inflammation; IF: infiltration

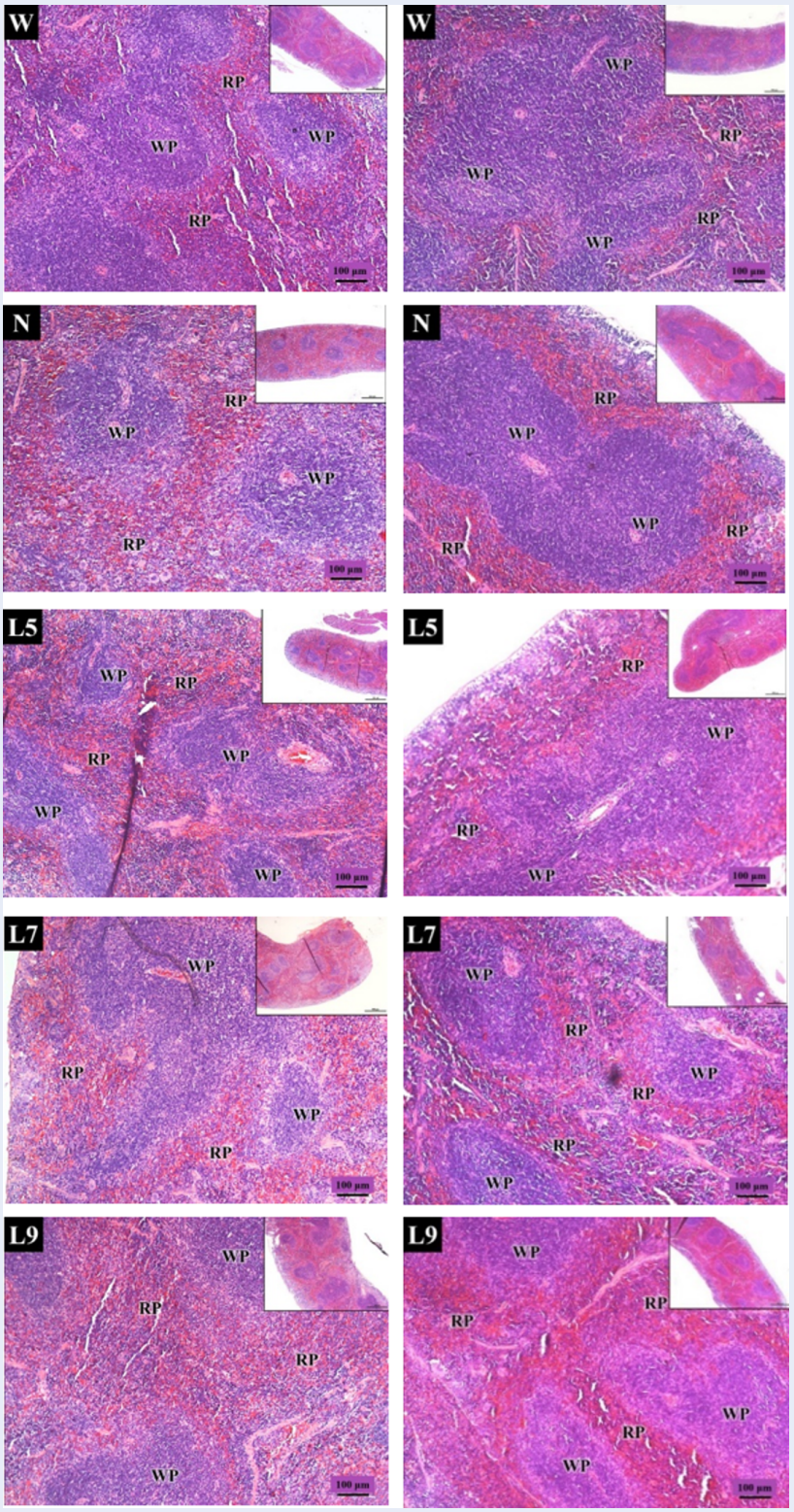

Figure 5: **The histological structure of mouse spleen tissue after every 4 weeks (x10), scale bar 100 μm**. W: control 1; N: control 2; L5: received 50 mg Pb/kg bw; L7: received 70 mg Pb/kg bw; L9: received 90 mg Pb/kg b. WP: white pulp; RP: red pulp

Lead poisoning may cause thrombocytopenia, which in turn may result in an increase in platelet proliferation [9,15]. Lead is able to compete with ions such as Ca^{2+}, Mg^{2+}, Fe^{2+}, and Na^{+} [3], which can disrupt the intracellular homeostasis of Ca^{2+} and activate protein kinase C [41] [30]. This disruption can lead to intravascular platelet rupture. Furthermore, the absence of noticeable signs of platelet capture and destruction in the mouse spleen histological analysis provides additional support for the aforementioned cause of the elevated platelet count.

Effect of lead on liver, kidneys and spleen

In this study, it was found that lead-containing treatments resulted in a reduction in liver enzyme levels (AST and ALT), which is different from some previous studies that showed an increase in liver enzyme levels in cases of lead poisoning [9,18]. This difference could be attributed to impaired liver function resulting from histological damage. Liver tissue damage can hinder the release of liver enzymes, leading to lower enzyme levels.

While a decrease in BUN level and an increase in CRE level may suggest liver dysfunction, it is not conclusive. Some studies have reported an increase in BUN and CRE levels in albino mice exposed to lead [9,10,12]. In terms of tissue structure, mainly phlebitis or tubular or renal tubular structures were destroyed in the lead-dose treatments. Therefore, the decrease in BUN index could be due to liver damage, and the increase in CRE index could be due to kidney damage. Further analysis of redox parameters and other biochemical parameters is necessary to better understand the histological and hematological results of liver and kidney structures.

The spleen tissue structure in mice after four weeks of the experiment showed that the lead-drinking groups showed an invasion of the red pulp into the white pulp, which increased gradually with increasing lead concentration after eight weeks. Exposure to lead can result in the invasion of red pulp into white pulp, which is a common phenomenon. The main reason is that lead affects the body's immune system. When lead is absorbed into the body, it is distributed to various tissues, including the bone marrow. The harmful effects of lead on bone marrow cells weaken the immune system, causing white pulp cells to lose their ability to prevent the growth of red pulp cells. Consequently, red pulp cells invade the white pulp, causing structural changes in the spleen tissue of animals exposed to lead [31].

CONCLUSION

This study provides evidence that exposure to lead at the tested concentrations can have various negative effects on the health of mice, including reduced weight gain, changes in blood cell counts, altered biochemical parameters, and tissue damage. Notably, the concentration of 90 mg/kg bw had the most significant effect among the three concentrations tested. However, further investigation of hematological and biochemical parameters is necessary to draw more comprehensive conclusions regarding the effects of lead on mice. These results have important implications for understanding the potential health risks associated with lead exposure and the need to develop strategies to mitigate its toxicity.

ABBREVIATIONS

AST: Aspartate aminotransferase
ALT: Alanine aminotransferase
RBC: Red blood cell
BW: Body weight
BUN: Blood urea nitrogen
CRE: Creatinine
H&E: Hematoxylin and eosin
L: Lead
MDA: Malondialdehyde
PLT: Platelet
WBC: White blood cell

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AUTHOR'S CONTRIBUTIONS

Huyen NTT and Chi TNL wrote the manuscript. Quan TK revised the manuscript. Huyen NTH and Quan TK planned and designed the experiments. Chi TNL, Tri TV and Hang NTT collected and analyzed the data. NTTH and Quan TK supervised the study and finalized the manuscript. All authors read and confirmed the publication of the article.

FUNDING

None.

AVAILABILITY OF DATA AND MATERIALS

Data and materials used and/or analyzed during the current study are available from the corresponding author on reasonable request.

ETHICS APPROVAL

All experimental procedures were approved by the Ethical Committee on Animal Experimentation of the University of Science, VNUHCM (ACUCUS, No. 1170B/KHTN-ACUCUS).

CONSENT FOR PUBLICATION

Not applicable.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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