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Protective effects of Sodium Tetraborate on fatty acid profiles in rat hearts subject to Lead toxicity

Efecto protector del tetraborato de sodio sobre los perfiles de ácidos grasos en el corazon de ratas, expuestas a toxicidad por plomo

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ABSTRACT

This study was designed to observe the effects of sodium tetraborate on preventing lead metal toxicity damage to fatty acids in rat heart tissue. After the heart tissues were homogenized with Tris-HCl buffer, the separated pellet part was prepared for fatty acid analysis. The fatty acids in the obtained lipid extract were converted to methyl ester form and analyzed with SHIMADZU GC 2010 PLUS gas chromatography. When comparing the lead group with the lead + sodium tetraborate group, partial increases were observed in saturated fatty acid levels such as myristic acid (14:0), pentadecanoic acid (15:0), and palmitic acid (16:0), while varying increases were seen in some monounsaturated fatty acid levels such as palmitoleic acid (16:1, n-7), and oleic acid (18:1, n-9). Moreover, demonstrated varying levels of increases in all polyunsaturated fatty acids excluding the arachidonic acid (20:4, n-6) and adrenic acid (22:4, n-6) fatty acids. As the most notable fatty acid change in our study, arachidonic acid (20:4, n-6) fatty acid increasing in the lead group while decreasing in the sodium tetraborate group demonstrates that sodium tetraborate decreased the negative effects of oxidative stress on fatty acids. The results of this study provide supporting evidence for the potential use of sodium tetraborate in drug design to prevent the toxic effects of lead.

Key words: Lead; sodium tetraborate; fatty acids; toxic effect; heart

RESUMEN

Este estudio fue diseñado para observar los efectos del tetraborato de sodio en la prevención del daño causado por la toxicidad del metal plomo a los ácidos grasos en el tejido cardíaco de ratas. Tras homogeneizar el tejido cardíaco con tampón Tris-HCl, el sedimento separado se preparó para el análisis de ácidos grasos. Los ácidos grasos del extracto lipídico obtenido se transformaron en éster metílico y se analizaron mediante cromatografía de gases SHIMADZU GC 2010 PLUS. Al comparar el grupo plomo con el grupo plomo + tetraborato de sodio, se observaron aumentos parciales en los niveles de ácidos grasos saturados como ácido mirístico (14:0), ácido pentadecanoico (15:0) y ácido palmítico (16:0), mientras que se observaron aumentos variables en algunos niveles de ácidos grasos monoinsaturados como ácido palmitoleico (16:1, n-7) y ácido oleico (18:1, n-9). Además, se observaron aumentos variables en todos los ácidos grasos poliinsaturados, excluyendo los ácidos grasos ácido araquidónico (20:4, n-6) y ácido adrenérgico (22:4, n-6). El cambio más notable en los ácidos grasos en nuestro estudio, el aumento del ácido graso ácido araquidónico (20:4, n-6) en el grupo tratado con plomo, mientras que disminuyó en el grupo tratado con tetraborato de sodio, demuestra que tetraborato de sodio disminuyó los efectos negativos del estrés oxidativo sobre los ácidos grasos. Los resultados de este estudio proporcionan evidencia que respalda el uso potencial de tetraborato de sodio en el diseño de fármacos para prevenir los efectos tóxicos del plomo.

Palabras clave: Plomo; tetraborato de sodio; ácidos grasos; efecto tóxico; corazón

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INTRODUCTION

Studies revealing the effects of boron that have been surprising to date, which also in critical of human and animal health, are present in the literature [1]. For example, boron has been reported to play important roles in the activity of metabolic enzymes, the metabolism of steroid hormones and micronutrients such as calcium, magnesium and vitamin D [2].

In light of all of the above mentioned data, boron has begun to receive necessary attention in recent years within medical chemistry and medicine development [3]. Even if people ingest boron through their diet, excessive exposure to boron can negatively affect health. Although inhalation poisoning is rare, one animal study showed that inhaling cellulose containing 20 % boric acid resulted in decreased fetal weight [4]. In addition, dermal exposure to boric acid has proven fatal in some cases. Deaths have occurred after oral ingestion of boric acid, and weight loss and reproductive toxicity have been demonstrated in animals. On the other hand, it has also been reported that there is no evidence that boric acid is carcinogenic in mice [4].

Although the toxic effects of lead (Pb) are classified as acute and chronic, there is no sharp boundary between these two categories [5]. It has been suggested that the acute effects of low doses of Pb are mostly not felt, but repeated exposure to high doses of Pb can cause poisoning due to nervous system damage, starting with symptoms such as metallic taste in the mouth, stomach pain, vomiting and diarrhea, and can lead to coma, respiratory arrest and even death [6]. The clinical significance of Pb stems from its chronic effects on blood cells and nerve cells [5]. As a significant enzyme inhibitor, Pb, once it enters cells, prevents selenium and sulfur-containing enzymes from exhibiting antioxidant activity [7]. The impairment of protoporphyrin metabolism leads to anemia; the loss of cellular oxidation and reduction balance, and loss of synaptic performance. All these functional deficiencies result in neurological symptoms such as loss of intelligence and memory, difficulty concentrating, and problems with the heart, kidneys, and liver [8, 9, 10].

Analyses of the Third National Health and Nutrition Examination Survey (NHANES III) conducted between 1988 and 1994 reported an association between blood Pb levels < 10 mg/dL and a higher risk of cardiovascular disease death; a negative slope was observed when whole blood Pb levels were < 2000 µg/dL, a positive slope was observed when whole blood Pb was > 0,25 and < 5000 µg /dL, and then a negative slope was observed when > 5000 µg /dL [11]. On the other hand, Menke *et al*. [12] reported that all-cause mortality, myocardial infarction mortality, and cancer mortality varied depending on whole blood Pb.

One study reported that subacute Pb exposure at the tested doses increased the O2- levels in the heart tissue of rats, and even at the lowest dose used in the experiment (0.1 mg Pb/kg body weight/day) increased O2- and Malondialdehyde levels compared to controls [13]. Malondialdehyde is the end product of lipid hydroperoxide degradation and is an indicator of the late stage of oxidative lipid damage [14, 15]. Oxidation of membrane lipids as a result of Pb exposure can Pb to alterations in membrane integrity, permeability, and function [16]. Pb has been shown to contribute to cardiovascular complications through changes in the structure of polyunsaturated fatty acids, induction of lipid peroxidation and disruption of membrane enzymes [17].

Boron has recently attracted great interest due to its pleiotropic effects such as activating immune responses, antioxidant activities, involvement in bone metabolism, enhancing animal performance and regulating various body systems [18]. It has been reported that boric acid is capable of acting as a coupling material capable of connecting heavy metals such as cadmium (Cd), that this may mean that boric acid could reduce Cd circulation and tissue accumulation, and that boric acid could thus reduce the amount of Cd in the body and reduce its toxicity [19]. This information has led to the idea that sodium tetraborate (ST), used as a boron source in this study, may also exhibit the same function against Pb, a heavy metal.

As studies report that metal toxicity has been correlated with oxidative stress [20] and the existence of a relationship between oxidative stress bioindicators and the fatty acid compound [21, 22] can be found in the literature. This study aimed to demonstrate the protective effects of ST, derived from boron, against potential damage to fatty acid levels caused by Pb-induced damage to cellular activities in heart tissues.

MATERIALS AND METHODS

Experimental design of animals

The husbandry of the Wistar Albino species of rats (*Rattus norvegicus*) that weighed (UTW-0632) 250 ± 300 g was conducted in the Bingöl University Animal Experiments Ethics Committee (BUHADEK:18.05.2021–2021/02). All experiments were conducted in accordance with the Laboratory Animal Husbandry and Usage Guidebook [23]. The provision of food and water was conducted in an ad libitum manner. Normal lighting and darkness were set (12L:12D). The temperature and relative humidity levels during the experiment were respectively, 20 ± 3 °C and between 40-60 %.

A total of 24 rats were utilized in the study and with each group having (n=6), the rats were split into 4 groups; those being Control (C) (0.5 mL, i.p isotonic water), (150 mg/kg/day/oral) (Merck, USA) [24], ST (100 mg/kg/day/oral) (Sigma, USA) [25], Pb + ST.

At the end of Pb and ST applications (7 days (d)), the rats were subjected to anesthesia through (60 mg/kg i.p.) Ketamine hydrochloride and 10 mg/kg Xylazine i.p. Heart tissues were extracted following median laparotomy, washed with phosphate buffered saline (PBS, PH:7.4), and preserved until the conduction of the fatty acid analysis. Heart tissues (1 grams (g)) were mechanically homogenized with a homogenizer (IKA T25 DIGITAL ULTRA-TURRAX, Germany) with Tris-HCl buffer and centrifuged (Hettich Zentaifugen, Germany) at 150 counts per seconds (s) (cps) for 10 s, at + 4 °C. Fatty acid analysis was performed on the pellet section separated in this way [26].

Preparation of methyl ester forms of fatty acids and their analysis by gas chromatography

Total lipid contents of samples were extracted after homogenization in 3:2 (v/v) hexane isopropanol mixtures according to procedures described by Hara and Radin [27]. All solvents contained 0.01 % butylated hydroxytoluene as an antioxidant. Fatty acid methyl esters were prepared from total lipid by acid catalyzed transmethylation at 55 °C for 15 12 h according to method of Christie and Tvrzicka [28, 29, 30].

Statistical method and analysis

Statistical analyses were conducted through the use of the SPSS 20.00 package program. One-way analysis of variance (ANOVA) was used to compare the control group with the experimental groups, and the LSD test was used for between-group comparisons. The results obtained in the study were calculated as mean ± standard error. A statistical significance level of $P < 0.05$ was accepted.

RESULTS AND DISCUSSION

TABLE I depicted the observed saturated fatty acid composition (SFA) within heart tissues. It was determined that the heart tissue fatty acid compound contained fatty acids such as myristic acid (14:0), pentadecanoic acid (15:0), palmitic acid (16:0), heptadecanoic (17:0), stearic acid (18:0), lignoceric acid (24:0). It was found that these fatty acids contained high amounts of palmitic acid and stearic acid. When the distribution of these fatty acids is inspected between groups, it was observed that the 14:0, 16:0, 17:0, and 18:0 amounts were, compared with the control group, higher in the Pb group at varying levels ($P > 0.05$). Conversely, the amounts of the 15:0 and 24:0 saturated fatty acids partially decreased in the Pb group ($P > 0.05$). When compared to the Pb group, on the other hand; the amounts of the 14:0, 15:0, and 16:0 fatty acids were not found to be different at a statistically significant level in the ST group while increases of varying levels were observed in the Pb + ST group ($P > 0.05$, $P < 0.05$, $P < 0.01$) (TABLE I). Other fatty acids, on the other hand (17:00, 18:0, and 24:00) demonstrated partial decreases relative to the Pb group ($P < 0.05$) (TABLE I, FIGS.1 and 2).

TABLE I

Saturated fatty acids concentration (SFA) (%)

SFA	C	Pb	ST	Pb + ST
14:0	0.32 ± 0.01	0.47 ± 0.08 ^{a}	0.29 ± 0.01 ^{a,b}	0.75 ± 0.30 ^{d,c}
15:0	0.18 ± 0.01	0.16 ± 0.02 ^{a}	0.17 ± 0.01^{a,a}	0.18 ± 0.03 ^{a,a}
16:0	13.53 ± 0.83	13.96 ± 1.02 ^{a}	13.65 ± 0.66 ^{a,a}	15.41 ± 1.93^{b,b}
17:0	0.44 ± 0,02	0.47 ± 0.08 ^{a,a}	0.43 ± 0.02 ^{a,a}	0.39 ± 0.01 ^{b,b}
18:0	14.87 ± 0.62	15.55 ± 0.83 ^{a}	15.39 ± 1.41 ^{a,a}	13.80 ± 1.31 ^{a,b}
24:0	0.96 ± 0.11	0.93 ± 0.15 ^{a,a}	1.30 ± 0.43 ^{a,b}	0.84 ± 0.19 ^{a,b}

a: $P > 0.05$, b: $P < 0.05$ c: $P < 0.01$, d: $P < 0.001$. 2nd letterings represent comparisons relative to the Pb group. SFA: Saturated fatty acids, 14:0 = myristic acid, 15:0 = pentadecanoic acid, 16:0 = palmitic acid, 17:0 = heptadecanoic acid, 18:0 = stearic acid, 24:0 = lignoceric acid. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

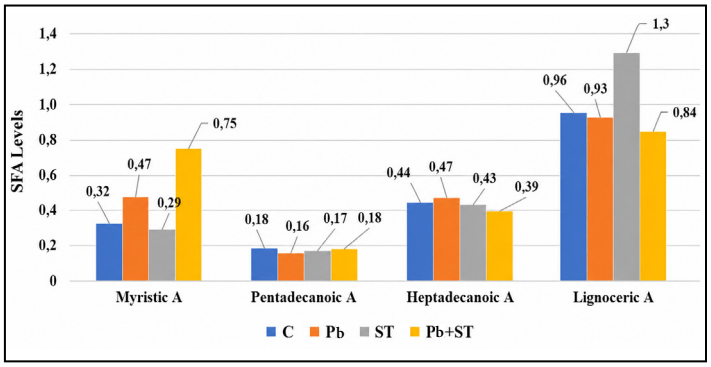

FIGURE 1. Graphical comparison of saturated fatty acids concentration a C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

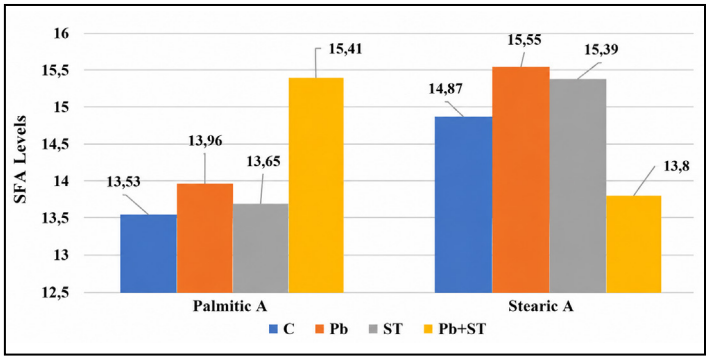

FIGURE 2. Graphical comparison of saturated fatty acids concentration b C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

The monounsaturated fatty acid composition (MUFA) observed within the heart tissue was depicted in TABLE II. Mono fatty acid compounds were observed to contain fatty acids such as pentadecanoic acid (15:1), palmitoleic acid (16:1, n-7), heptadecanoic acid (17:1), octadecanoic acid (18:1, n-7) and oleic acid (18:1, n-9). It was determined that these fatty acids, on the other hand, contained high amounts of octadecanoic acid (18:1, n-7) and oleic acid (18:1, n-9).

These evaluation of the inter-group distribution of monounsaturated fatty acids (15:1, 16:1, n-7, and 18:1, n-9) when compared to C, demonstrated no statistically significant differences in the Pb group while increases were observed ($P > 0.05$). However, some monounsaturated fatty acids (17:1 and 18:1, n-7) observably decreased in the Pb group relative to the control group ($P > 0.005$, $P < 0.001$).

These results were evaluated to show the differences in the next major comparison: the comparison of the Pb and Pb + ST groups. When a comparison with the Pb group is drawn, on the other hand; it can be observed that while the ST group demonstrated a distinct increase in the 18:1, n-7 fatty acid ($P < 0.01$), the other fatty acids demonstrated the opposite circumstance for 15:1, 16:1, n-7, 17:1, and 18:1, n-9 ($P > 0.05$).

Compared to the Pb group, the Pb + ST group demonstrated increases of varying levels in the amounts of the palmitoleic acid (16:1, n-7) and oleic acid (18:1, n-9). monounsaturated fatty acids ($P > 0.05$, $P < 0.001$) while demonstrating the opposite circumstance for the others ($P > 0.05$, $P < 0.05$) (TABLE II, FIGS. 3 and 4). In conclusion, it was determined that ST only showed

a positive increase in palmitoleic acid (16:1, n-7) and oleic acid (18:1, n-9) levels, which are monounsaturated fatty acids, against Pb.

TABLE II

Concentration of monounsaturated fatty acids (MUFA) (%)

MUFA	C	Pb	ST	Pb + ST
15:1	1.81 ± 0.17	1.99 ± 0.21 ^{a}	1.90 ± 0.14 ^{a,a}	1.77 ± 0.36 ^{a,a}
16:1, n-7	0.54 ± 0.01	0.73 ± 0.82 ^{a}	0.38 ± 0.06 ^{a,a}	0.89 ± 0.55 ^{a,a}
17:1	1.11 ± 0.08	1.09 ± 0.09 ^{a}	1.01 ± 0.11 ^{a,a}	0.91 ± 0.13 ^{b,b}
18:1, n-7	3.27 ± 0.22	2.60 ± 0.27 ^{d}	3.02 ± 0.19 ^{a,c}	2.58 ± 0.26 ^{d,a}
18:1, n-9	11.10 ± 0.79	11.37 ± 1.47 ^{a,a}	11.00 ± 1.26 ^{a,a}	18.24 ± 1,57 ^{d,d}

Monounsaturated fatty acids concentration (MUFA) C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate. a: P > 0.05, b: P < 0.05 c: P < 0.01, d: P < 0.001. 2nd letterings represent comparisons relative to the Pb group. 15:1 = pentadecanoic acid, 16:1, n-7 = palmitoleic acid, 17:1 = heptadecanoic acid, 18:1, n-7 = octadecanoic acid, 18:1, n-9 = oleic acid. C = Control, Pb = Lead, ST = Sodium tetraborate Pb + ST = Lead + Sodium tetraborate

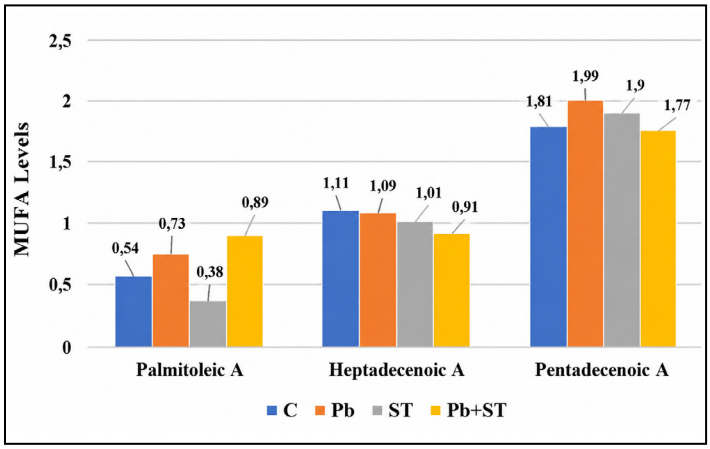

FIGURE 3. Graphical comparison of monounsaturated fatty acids concentration. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

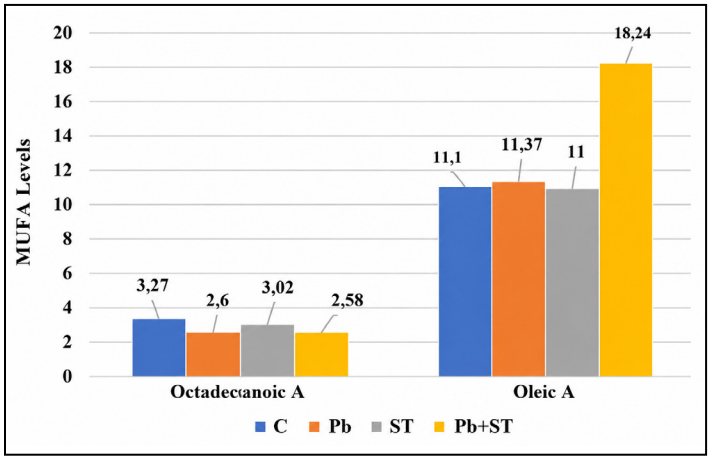

FIGURE 4. Graphical comparison of monounsaturated fatty acids concentration. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

TABLE III depicts the composition of the polyunsaturated fatty acid compound (PUFA) of the heart tissue. It was observed that the polyunsaturated fatty acid compound contained various unsaturated fatty acids such as lineoleic acid (18:2, n-6), linolenic acid (18:3, n-3 LNA), arachidonic acid (20:4, n6), docosapentaenoic acid (22:5, n-3), adrenic acid (22:4, n-6), and docosahexaenoic acid (22:6, n-3). Within these fatty acids, on the other hand, it was observed that the lineoleic acid (18:2, n-6), arachidonic acid (20:4, n6) and docosahexaenoic acid (22:6, n-3) fatty acids were contained at higher levels.

The inter-group comparison of the distributions of polyunsaturated fatty acids revealed that lineoleic acid (18:2, n-6c), docosapentaenoic acid (22:5, n-3) and docosahexaenoic acid (22:6, n-3) demonstrated decreases of varying levels in the Pb group compared to C (P > 0.05, P < 0.01). On the other hand, arachidonic acid (20:4, n-6) level demonstrated a distinct increase (P < 0.01). A comparison of polyunsaturated fatty acid levels relative to the Pb group reveals that while decreases in arachidonic acid (20:4, n6) levels were observed in both the St and the Pb + ST groups (P > 0.01, P < 0.001), with the Pb + ST group being more distinct within this context, the docosapentaenoic acid (22:6, n-3) amount demonstrated decreases of varying levels in both the ST and the Pb + ST groups (P < 0.05, P < 0.01) (TABLE III, FIGS. 5 and 6).

TABLE III

Amount of polyunsaturated fatty acids (PUFA) (%)

PUFA	C	Pb	ST	Pb + ST
18:2, n-6c	24.49 ± 1.97	23.87 ± 1.02 ^{a}	24.76 ± 1.43 ^{a,a}	24.44 ± 0.97 ^{a,a}
18:3, n-3 LNA	0.35 ± 0.05	0.35 ± 0.06 ^{a}	0.32 ± 0.05 ^{a,a}	0.39 ± 0.06 ^{a,a}
20:4, n-6	17.01 ± 1.60	17.78 ± 1.06^{c}	17.00 ± 1.79 ^{a,c}	14.13 ± 1.10 ^{d,d}
22:5, n-3	1.06 ± 0.10	0.88 ± 0.14 ^{b}	0.95 ± 0.13 ^{a,a}	0.93 ± 0,14 ^{a,a}
22:4, n-6	1.07 ± 0.13	1.14 ± 0.08 ^{a}	1.05 ± 0.08 ^{a,a}	1.06 ± 0.11 ^{a,a}
22:6, n-3	5.62 ± 0.43	4.32 ± 0.69 ^{c}	5.64 ± 0.63 ^{a,c}	4.90 ± 0.60^{c,b}

polyunsaturated fatty acids concentration (PUFA), C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST= Lead + Sodium tetraborate a: P > 0.05, b: P < 0.05 c: P < 0.01, d: P < 0.001. 2nd letterings represent comparisons relative to the Pb group. 18:2, n-6c = lineoleic acid, 18:3, n-3 LNA = linolenic acid, 20:4, n6 = arachidonic acid, 22:5, n-3 = docosapentaenoic acid, 22:4, n-6 = adrenic acid, 22:6, n-3 = docosahexaenoic acid. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

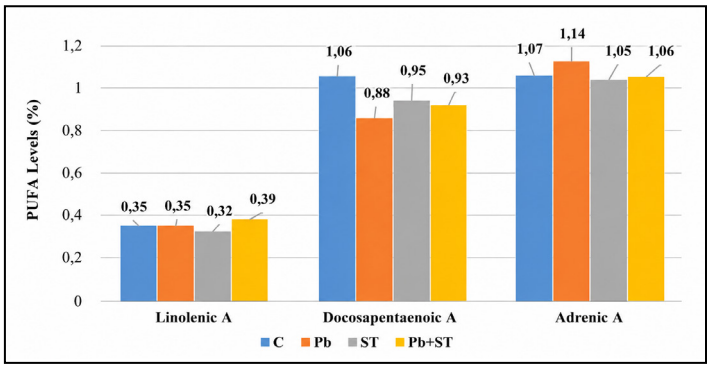

FIGURE 5. Graphical comparison of polyunsaturated fatty acids concentration. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

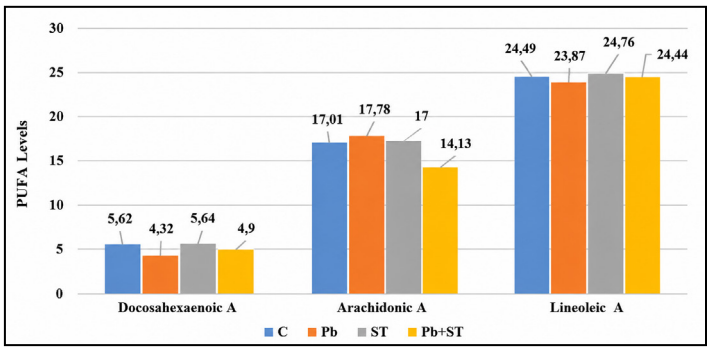

FIGURE 6. Graphical comparison of polyunsaturated fatty acids concentration. C = Control, Pb = Lead, ST = Sodium tetraborate, Pb + ST = Lead + Sodium tetraborate

A previously conducted study put forward the claim that the addition of calcium tetraborate partially prevented increases in the oxidative stress index (OSI) and the total oxidant (TOS) concentrations caused by Cadmium, and that this circumstance results in the calcium tetraborate (CTB) preventing oxidative protein, lipid, and DNA injuries in liver tissues. In this mentioned study, it was also reported that the boron treatment led to decreases in TOS and OSI while simultaneously decreasing the lipid oxidation and oxidative stress caused by ethanol at a significant rate [31]. The protective mechanism of boric acid against oxidative injuries has not yet been entirely grasped, while its role in cell membrane functions and enzymatic reactions are commonly known.

Considering the genetic marker diversity between gender, race and environment, lipids can be an indicator with much less inconsistency because they are common metabolic substances [32], so this study aimed to take advantage of this property of fatty acids. Many studies demonstrate that high amounts of Pb and oxidative stress bioindicators have a relationship with the fatty acid compound can be found in the literature [20, 33]. One of these studies reported that baby greenhead ducks and baby Canadian geese demonstrated increased lipid peroxidation in their livers after exposure to Pb through nourishment [34, 35].

In a study investigating the effects of Pb-induced toxicity on liver, kidney, brain, and heart tissues, and the activities of catalase (CAT), superoxide dismutase (SOD), glutathione, glutathione peroxidase, and glutathione reductase—important enzymes of the antioxidant system that serve as a defense against oxidative damage—in Wistar rats; inhibition of antioxidant enzyme activity levels was observed in rats exposed to Pb, and a significant decrease in glutathione levels was reported in these rats. Furthermore, it has been reported that lipid peroxidation, DNA degradation, and hematological parameters were significantly altered in rats treated with Pb acetate compared to controls [36]. In another study investigating the effects of Pb, it was observed that hematocrit levels and blood δ-aminolevulinic acid dehydratase (ALA-D) function decreased, and Pb levels increased in blood and tissues, lipid peroxidation levels increased in erythrocytes, plasma, and tissues, and caused protein oxidation in tissues [37].

In a study investigating the effects of chronic Pb exposure on the oxidative stress status of the heart and liver in rats, it was reported that Pb exposure increased the activity levels of important antioxidant enzymes, SOD and CAT, in the liver and heart tissues examined [38].

Some studies inspecting changes in the fatty acid compound in various tissues of animals exposed to Pb reported increases, especially in arachidonic acid (AA) [39, 40]. In this study, similar results were obtained to the studies mentioned above regarding the increase in arachidonic acid levels in the group treated with Pb. Additionally, in this study the increase of the AA fatty acid content in the Pb group and its decrease in the boron-supported group indicates that boron decreased the negative effects of oxidative stress on fatty acids. In a study, fatty acid changes in red deer and wild boar tissues in the Pb mining area were investigated and it was reported that significant differences were observed in the fatty acid composition of the red deer liver compared to the results in the control and mining areas [41]. This study additionally stated the most significant change in the fatty acid compound to be a decrease in the percentage of docosahexaenoic acid (22:6, n-3), an n-3 PUFA (Polyunsaturated Fatty Acid), in animals with high Pb concentrations. The concentrations of several metals studied here, especially Pb, were reported to be significantly higher in the tissues of red deer and wild boar from the Pb mining area than in the control sites in the same Province of Ciudad Real. An important issue in the study was that there was a significant correlation between the Pb concentration in the liver of red deer and the percentage of n-3 PUFA (n ¼ 70, r ¼ 0.341, p ¼ 0.004), however, no significant difference was reported between mining areas and the control group. At the conclusion of the study, they claimed that this decrease in fatty acid levels could be correlated with the increased oxidation of this unsaturated FA [41].

Another similar study conducted by Lim *et al*. [42], stated that decreases in the 22:6, n-3 fatty acid concentration of 3-week-old rats who were exposed to Pb through the milk of their mothers who were exposed to drinking water with 0,2 % Pb acetate during the breast-feeding period were observed. In the same study, baby rats who were once again exposed to Pb reportedly demonstrated decreases in n-3 PUFA concentrations in their livers.

In another study conducted as a follow-up to the afore mentioned one, livers of mothers exposed to Pb demonstrated a relative increase in the amount of the 22:6, n-3 fatty acid, while this increase was not present in the tissue concentration [43]. As this study also demonstrated a decrease in the percentage of docosahexaenoic acid (22:6, n-3), it demonstrates similarities to the other afore mentioned studies.

In a study, fatty acids in the brain of mice exposed to Pb were examined to observe the toxicological effects, and at the end of the study, it was reported that Pb exposure affected the composition of lipids in the brain of mice, especially C16:0, C18:0 and C18:1. This study is similar to in literature studies in that the change in C16:0, C18:0 fatty acids were significant in the Pb + CTB group compared to the Pb group. Additionally, differences in varying proportions in C18:1 (n-7 and n-9) fatty acids are observed in this study [32].

In another study, fatty acid contents were examined in Arbor Acre broiler (*Gallus gallus domesticus*) chicks fed diets containing 0, 500, 750, 1000, 2000 or 4000 ppm Pb. In the study, it was determined that increasing dietary Pb levels decreased the concentrations of 16:1 and 18:1 fatty acid, while increasing the concentrations of 20:4. This literature and our study are similar in terms of the decrease in the octadecanoic acid (18:1, n-7) ratio and the increase in the arachidonic acid (20:4) ratio in the Pb group. It was reported that the concentration of lineoleic acid (18:2) fatty acids was not significantly different from the control values for any Pb level, but the 18:2/20:4 ratio decreased from a control value of 3.3 to approximately 2 for all Pb treatments. In the same study, it was reported that the ratio of saturated/monoenoic fatty acids increased with dietary Pb levels above 1000 ppm. In this study, the increase in saturated fatty acids other than pentadecanoic acid (15:0) and arachidonic acid (20:4) fatty acids in the Pb group is similar to previous study [44].

CONCLUSION

The results of this study, like those in previous studies in the literature, showed a similarity in terms of the increase in the 20:4 fatty acid ratio following Pb application, which suggests that the increase in the 20:4 ratio in heart tissue may be due to Pb toxicity. While many studies regarding the healing effect of antioxidant vitamins or substances against Pb toxicity can be found, no studies inspecting the effect of ST against the toxic impact of Pb on the fatty acid amounts in heart tissues were present in the literature. The results of this study support research on both the toxic effects of Pb and the antioxidant effects of ST, offering insights into how ST could be used in drug or supplement design to counter the toxic effects of Pb.

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Conflict of interest

The authors declare no conflicts of interest.

BIBLIOGRAFIC REFERENCES

[1] Khaliq H, Juming Z, Ke-Mei P. The Physiological Role of Boron on Health. Biol. Trace. Elem. Res. [Internet]. 2018; 186:31–51. doi: https://doi.org/gfhcgt

[2] Sun P, Luo Y, Wu XT, Ansari AR, Wang J Yang K, Xiao K, Peng K. Effects of Supplemental Boron on Intestinal Proliferation and Apoptosis in African Ostrich Chicks. Int. J. Morphol. [Internet]. 2016; 34(3):830-835. doi: https://doi.org/q7cd

[3] Keklik E, Keklik M, Bakkaloğlu U, Yürük M, Çoksevim B. The Effect of Borax on Hematological Parameters and Immunoglobulin Values in Rats. West Indian Med. J. [Internet]. 2016; 65:1-14. doi: https://doi.org/q7cf

[4] Hadrup N, Frederiksen M, Sharma AK. Toxicity of boric acid, borax and other boron containing compounds: A review. Regul. Toxicol. Pharmacol. [Internet]. 2021; 121:104873. doi: https://doi.org/q7cg

[5] Sandhir R, Gill KD. Effect of lead on lipid peroxidation in liver of rats. Biol Trace Elem Res. [Internet]. 1995; 48(1):91-97. doi: https://doi.org/fs5xj3

[6] El-Sokkary GH, Kamel ES, Reiter RJ. Prophylactic effect of melatonin in reducing lead-induced neurotoxicity in the rat. Cell. Mol. Biol. Lett. [Internet]. 2003; 8(2):461-470. [cited 19 Apr 2026]. Available in: https://goo.su/T5Bk1t

[7] Solliway BM, Schaffer A, Pratt H, Yannai S. Effects of exposure to lead on selected biochemical and haematological variables. Pharmacol. Toxicol. [Internet]. 1996; 78(1):18-22. doi: https://doi.org/bk79m8

[8] Knowles SO, Donaldson WE. Dietary modification of lead toxicity: Effects on fatty acid and eicosanoid metabolism in chicks. Comp. Biochem. Physiol. C. Comp. Pharmacol. [Internet]. 1990; 95(1):99-104. doi: https://doi.org/fd3v

[9] Lawton LJ, Donaldson WE. Lead-induced tissue fatty acid alterations and lipid peroxidation. Biol. Trace Elem. Res. [Internet]. 1991; 28(2):83-97. doi: https://doi.org/b3jbkd

[10] Ribarov SR, Benov LC. Relationship between the hemolytic action of heavy metals and lipid peroxidation. Biochim. Biophys. Acta. [Internet]. 1981; 640(3):721-726. doi: https://doi.org/bj6h25

[11] Aoki Y, Brody DJ, Flegal KM, Fakhouri THI, Parker JD, Axelrad DA. Blood Lead and Other Metal Biomarkers as Risk Factors for Cardiovascular Disease Mortality. Medicine. [Internet]. 2016; 95(1):e2223. doi: https://doi.org/q7cm

[12] Menke A, Muntner P, Batuman V, Silbergeld EK, Guallar, E. Blood lead below 0.48 micromol/L (10 microg/dL) and mortality among US adults. Circulation. [Internet]. 2006; 114(13):1388–1394. doi: https://doi.org/csq6tt

[13] Javorac D, Tatovi´c S, Anđelkovi´c M, Repi´c A, Barali´c K, Djordjevic AB, Mihajlovi´c M, Stevuljevi´c JK, Đuki´c-´Cosi´c D, ´Cur˘ci´ M, Antonijevi´c B, Bula, Z. Low-lead doses induce oxidative damage in cardiac tissue: Subacute toxicity study in Wistar rats and Benchmark dose modelling. Food Chem. Toxicol. [Internet]. 2022; 161:112825. doi: https://doi.org/q7cq

[14] Fan Y, Zhao X, Yu J, Xie J, Li C, Liu D, Tang C, Wang C. Lead-induced oxidative damage in rats/mice: a meta-analysis. J. Trace Elem. Med. Biol. [Internet]. 2020; 58:126443. doi: https://doi.org/gp35nt

[15] Basaran N, Duydu Y, Bacanlı M, Gül-Anlar H, Dilsiz SA, Üstündağ A, Yalçın CÖ, Schwerdtle T, Bolt HM. Evaluation of oxidative stress and immune parameters of boron exposed males and females. Food Chem. Toxicol. [Internet]. 2020; 142:111488. doi: https://doi.org/q7cr

[16] Poreba R, Gac P, Poreba M, Andrzejak R. Environmental and occupational exposure to lead as a potential risk factor for cardiovascular diseases. Environ. Toxicol. Pharmcol. [Internet]. 2011; 31(2):267–277. doi: https://doi.org/dvgtks

[17] Jan AT, Azam M, Siddiqui K, Ali A, Choi I, Rizwanul-Haq QM. Heavy Metals and Human Health: Mechanistic Insight into Toxicity and Counter Defense System of Antioxidants. Int. J. Mol. Sci. [Internet]. 2015; 16(12):29592–29630. doi: https://doi.org/f756m8

[18] Abdelnour SA, Abd El-Hack M, Swelum AA, Perillo A, Losacco C. The vital roles of boron in animal health and production: a comprehensive review. J. Trace Elem. Med. Biol. [Internet]. 2018; 50:296–304.doi: https://doi.org/gfm5s4

[19] Kar I, Patra AK. Tissue bioaccumulation and toxicopathological effects of cadmium and its dietary amelioration in poultry-a review. Biol. Trace Elem. Res. [Internet]. 2021; 199(10):3846–3868. doi: https://doi.org/g37jv3

[20] Donaldson WE, Knowles SO. Is lead toxicosis a reflection of altered fatty acid composition of membranes? Comp. Biochem. Physiol. C Comp. Pharmacol. [Internet].1993; 104(3):377–379. doi: https://doi.org/c7n28b

[21] Hsu P.C, Guo YL. Antioxidant nutrients and lead toxicity. Toxicology. [Internet].2002; 180(1):33–44. doi: https://doi.org/bvs5zk

[22] Jomova K, Valko M. Advances in metal-induced oxidative stress and human disease. Toxicology. [Internet]. 2011; 283(2–3):65–87. doi: https://doi.org/fc7bv8

[23] Guide for the Care and Use of Laboratory Animals. National Research Council. 8th edition. Committee for the Update of the Guide for the Care and Use of Laboratory Animals. Washington, DC, USA: National Academies Press; 2011 [cited 20 Jan 2026]. Available in: https://goo.su/X95u9t

[24] Salcan I, Dilber M, Bayram R, Suleyman E, Erhan E, Karahan Yilmaz S, Yazici GN, Coban, A, Suleyman H. Effect of Taxifolin on Cobalt-induced Ototoxicity in Rats: A Biochemical and Histopathological Study. Int. J. Pharmacol. [Internet]. 2000 [cited Sep 15 2025]; 16(8):522-528. doi: https://doi.org/q7c3

[25] Beydilli H, Yilmaz N, Cetin ES, Topal Y, Celik OI, Sahin C, Topal H, Cigerci IH, Sozen H. Evaluation of the protective effect of silibinin against diazinon induced hepatotoxicity and free-radical damage in rat liver. Iran Red. Crescent. Med. J. [Internet]. 2015; 17(4):e25310. doi: https://doi.org/q7c4

[26] Demir E, Yılmaz Ö. The effects of bitter almond oil on some biochemical parameters in serum and erythrocytes of streptozotocin-induced Type-1 diabetic rats. Van Vet. J. [Internet]. 2015 [cited 01 Sep 2025]; 26(1):17-23. Available in: https://goo.su/njb2B

[27] Hara A, Radin NS. Lipid extraction of tissues with a low-toxicity solven. Anal. Biochem. [Internet]. 1978; 90(1):420–426. doi: https://doi.org/b3c4h8

[28] Christie WW. Gas chromatography and lipids. Glaskow: The Oil Pres; [Internet]. 1989. [cited 07 Sep 2025]. Available in: https://goo.su/7C872

[29] Ichihara K, Fukubayashi Y. Preparation of fatty acid methyl esters for gas-liquid chromatography. J. Lipid Res. [Internet]. 2010; 51(3):635-640. doi: https://doi.org/b5g5sk

[30] Tvrzicka E, Vecka M, Stankova Zak A. Analysis of fatty acids inplasma lipoproteins by gas chromatography flame ionisation detection Quantative aspects. Anal. Chim. Acta. [Internet]. 2002; 465(1-2):337-350. doi: https://doi.org/d624gw

[31] Cikler-Dulger E, Sogut I. Investigation of the protective effects of boric acid on ethanol induced kidney injury. Biotech. Histochem. [Internet]. 2020; 95(3):186–193. doi: https://doi.org/gjvtcj

[32] Jung JM, Lee J, Kim KH, Jang IG, Song JG, Kang K, Tack FMG, Oh JI, Kwon EE, Kim HW. The effect of lead exposure on fatty acid composition in mouse brain analyzed using pseudo-catalytic derivatization. Environ. Pollut. [Internet]. 2017; 222:182-190. doi: https://doi.org/f9zp3g

[33] Hsu PC, Guo YL. Antioxidant nutrients and lead toxicity. Toxicology. [Internet]. 2002; 180(1):33–44. doi: https://doi.org/bvs5zk

[34] Hoffman DJ, Heinz GH, Sileo L, Audet DJ, Campbell JK, Le Captain LJ. Developmental toxicity of lead-contaminated sediment to mallard ducklings. Arch. Environ. Contam. Toxicol. [Internet]. 2000;39(2):221–232. doi: https://doi.org/bsfjq7

[35] Hoffman DJ, Heinz GH, Sileo L, Audet DJ, Campbell JK, LeCaptain LJ, Obrecht HH. Developmental toxicity of lead-contaminated sediment in Canada geese (Branta canadensis). J. Toxicol. Environ. Health A. [Internet]. 2000; 59(4):235–252. doi: https://doi.org/bj953v

[36] Dewanjee S, Sahu R, Karmakar S, Gangopadhyay M. Toxic Effects Of Lead Exposure In Wistar Rats: Involvement Of Oxidative Stress And The Beneficial Role Of Edible Jute (*Corchorus olitorius*) Leaves. [Internet]. Food Chem. Toxicol. 2013; 55: 78–91. doi: https://doi.org/f4tq7x

[37] Reckziegel P, Dias VT, Benvegnú DM, Boufleur N, Barcelos RCS, Segat HJ, Pase CS, dos Santos CMM, Flores EMM, Bürger ME. Antioxidant Protection Of Gallic Acid Against Toxicity Induced By Pb In Blood, Liver And Kidney Of Rats. Toxicol. Rep. [Internet]. 2016; 3:351–356. doi: https://doi.org/g5jxpx

[38] Markiewicz-Gorka I, Januszewska L, Michalak A, Prokopowicz A, Januszewska E, Pawlas N, Pawlas K. Effects of chronic exposure to lead, cadmium, and manganese mixtures on oxidative stress in rat liver and heart. Arh. Hig. Rada. Toksikol. [Internet]. 2015; 66(1):51–61. doi: https://doi.org/gm75q6

[39] Knowles SO, Donaldson WE, Andrews JE. Changes in fatty acid composition of lipids from birds, rodents, and preschool children exposed to lead. Biol.Trace Elem.Res. [Internet]. 1998; 61:113–125. doi: https://doi.org/dw8zdh

[40] Osterode W, Ulberth F. Increased concentration of arachidonic acid in erythrocyte membranes in chronically lead-exposed men. J. Toxicol. Environ. Health A. [Internet]. 2000; 59(2):87–95. doi: https://doi.org/cgv3mn

[41] Reglero MM, Taggart MA, Monsalve-Gonzalez L, Mateo R. Heavy metal exposure in large game from a lead mining area:Effects on oxidative stress and fatty acid composition in liver. Environ. Pollut. [Internet]. 2009; 157(4):1388–1395. doi: https://doi.org/fshnqd

[42] Lim SY, Doherty JD, McBride K, Miller-Ihli NJ, Carmona GN, Stark KD, Salem NJr. Lead exposure and (n-3) fatty acid deficiency during rat neonatal development affect subsequent spatial task performance and olfactory discrimination. J. Nutr. [Internet]. 2005: 135(5):1019–1026. doi: https://doi.org/q7c9

[43] Lim SY, Loewke J, Doherty JD, Norman SJr. Preferential effect of lead exposure during lactation on non-essential fatty acids in maternal organs. Lipids. [Internet]. 2005b; 40(7):685–693. doi: https://doi.org/fm2cgj

[44] Donaldson, WE, Leeming TK. Dietary Lead: Effects on Hepatic Fatty Acid Composition in Chicks. Toxicol. Appl. Pharmacol. [Internet]. 1984; 73(1):119-123. doi: https://doi.org/c3s6df