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Role of Sodium Butyrate Supplement on Reducing Hepatotoxicity Induced by Lead Acetate in Rats

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A B S T R A C T

Lead has always been a health risk in developing countries. Lead severely affects liver function. Butyrate is effective in treating inflammatory disorders in animals. Thus, this study aimed to determine whether sodium butyrate mitigates lead acetate-induced hepatotoxicity. In this research, 40 adult female albino rats were randomly assigned to one of four treatment groups for a duration of 35 days as follows: group 1 served as a control, group 2 received sodium butyrate (SB) orally at 200 mg/kg daily, group 3 received lead acetate (LA) orally at 50 mg/kg daily, and group 4 received both SB and LA (SB+LA) orally. Blood was collected for complete blood picture (CBC) and some serum biochemical evaluations. Liver samples were collected for histopathological examination. The rats that exposed to lead acetate showed a significant (P<0.05) elevation in globulin, total bilirubin, total serum protein, and total white blood cells with a decrease in total red blood cells, haemoglobin, and packed cell volume, while weight gain shows a significant (P<0.05) decrease in this group. Histologically showed pre-vascular infiltration of the nuclear cell. Body weight of Rat's gavage with sodium butyrate showed a substantial (P<0.05) increase, as well as there, were improvements in red blood cells RBC, haemoglobin, and packed cell volume PCV with the normal histological structure of the liver and no pathological lesion in hepatocyte. The fourth group (SB+LA) showed a significant (P<0.05) decrease in total bilirubin, indirect bilirubin, and total white blood cells, while other tests in this group showed nearly the control group as a result of the effect of SB. In conclusion, sodium butyrate consumption effectively reduces the harmful effects of lead acetate and prevents liver damage.

Keywords: lead, toxicity, butyric acid, bilirubin, globulin, rat

INTRODUCTION

Prolonged lead (Pb) exposure, even in small amounts, has been linked to a variety of health problems, including hematological, nephrotic, and liver diseases. Lead toxicity, which occurs as a result of occupational and environmental contamination, continues to be a problem for global health (1-4). Dust, drinking water, and many foods are among the sources of this lead toxin exposure for people and animals.

The liver has been seen as a target organ that is contaminated by enteral Pb absorption via the portal vein (5). Several mechanisms have been proposed to explain the liver damage caused by Pb. Pb can cause changes in hepatic biotransformation and activity that may lead to hepatocyte dysfunction, nucleic acid synthesis, and cholesterol metabolism (5, 6). Structural disorganization of these cells, including necrosis and hydropic degeneration, is brought on by the metal's accumulation in the hepatic cells (7). This

toxin may be blamed for the decline in the antioxidant defense system (8). Considering the significance of oxidative damage caused by Pb, a treatment approach aimed at strengthening the body's antioxidant defenses may help in preventing this toxin (7). Reactive oxygen species (ROS) production is in excess compared to an antioxidant system's ability to scavenge them, which results in oxidative stress (9). As a byproduct of energy metabolism, various types of liver cells continuously produce ROS, including hydrogen peroxide (H_2O_2) and superoxide anion radicals ($O2^{\bullet-}$) (10). When ROS are present in high concentrations, they oxidatively modify cellular macromolecules (DNA, lipids, proteins, etc.), which cause an accumulation of damaged macromolecules and damage the liver (11, 12). Additionally, ROS signaling may play significant roles in typical physiological activities, including controlling cellular homeostasis, or it may take part in an unfavorable reaction that encourages metabolic dysfunction and an inflammatory response, based on the types of cells, tissue environment, and ROS sources (13, 14). As a result, although molecular pathways are not yet fully characterized, the development of non-alcoholic fatty liver disease (NAFLD) may be caused by both indiscriminate oxidative biomolecular damage and improper redox signaling (15).

One of the short-chain fatty acids (SCFAs) is butyrate, which can be produced by gut bacteria fermenting dietary polysaccharides under anaerobic conditions (16, 17). In addition to being crucial for preserving intestinal health, SCFA also reaches the bloodstream and has an immediate impact on peripheral tissue metabolism (18). In type 2 diabetic mice, butyrate supplementation in drinking water guards against fat-rich diet-induced glucose intolerance and metabolic problems (19). Sodium butyrate regulates the skeletal muscle mitochondrial adaptation brought on by a fat-rich diet in mice, with a rise in the proportion of type 1 fibers that exhibit higher-oxidation activity and insulin sensitivity (20). Lowering oral butyrate restores diet-induced insulin resistance in rats by increasing energy intake and enhancing fat oxidation through burning brown adipose tissue (21). Hence, this study evaluated sodium butyrate's ability to prevent lead acetate toxicity from damaging the liver.

MATERIALS AND METHODS

Ethics

The experimental design and procedures used in this study were reviewed and approved in accordance with animal welfare ethical standards by the Scientific Committee of the Department of Physiology, Biochemistry, and Pharmacology, College of Veterinary Medicine, University of Baghdad, and the Ethics Committee of the College of Veterinary Medicine, University of Baghdad, Baghdad, Iraq.

Animals and Management

A total of 40 mature Wister Albino female rats ranging in weight from 190 to 200 g and in age from 8 to 9 weeks were obtained from and housed at the animal house of the College of Veterinary Medicine, University of Baghdad. Animals were housed under standard conditions of controlled temperature (25±2 °C), relative humidity (50-60%), and photoperiods (12 h light-dark cycle). Two weeks of acclimation were performed on the rats before the start of the study. Commercial pellets and water were provided to the animals *ad libitum* throughout the entire experiment.

Study Design

Sodium butyrate (MyBioSource®, USA) supplement was given to rats at a dose of 200 mg/kg BW (22) by weighted 500 mg from this supplement and dissolved in 5 mL distilled water and given 0.2 mL for each 100 g BW of rats. Lead acetate from (Sigma-Aldrich, St. Louis, MO, USA) was given to rats at a dose of 50 mg/kg BW (23) by dissolving 250 mg of lead acetate in 10 mL distilled water and gavaged 0.2 mL from this solved for each 100 g BW of rat both chemicals were given daily for 35 days

The animals were randomly allocated into four groups of ten animals each for a duration of 35 days as follows: group 1 served as a control, group 2 was given sodium butyrate (SB) an oral dose of 200 mg/kg daily, group 3 was given lead acetate (LA) of an orally dose of 50 mg/kg daily, and group 4 was given both sodium butyrate and lead acetate (SB+LA) orally.

Sample Collection and Laboratory Tests

Body weight was measured twice weekly for 35 days of the experiment to calculate the weight gain.

At the end of the study, rats were anesthetized using intramuscularly injected a mixture of ketamine-HCL 90 mg/kg BW and xylazine 40 mg/kg BW (Alfasan, Woerden, Holland). Blood was drawn from the pre-orbital sac and divided into two parts. The first part of blood with EDTA for complete blood count (CBC) samples. Second part was without EDTA for serum harvesting.

Blood samples were analyzed by automated blood analyzer (Mythic™ 18 Vet, Orphée Switzerland) to measure red blood cell count (RBC), hemoglobin concentration (Hb), packed cell volume (PCV), and total white blood cell count (WBC). Total serum protein (TSP) concentration (albumin and globulin), and total serum bilirubin (TSB, direct and indirect bilirubin) were measured using commercially available colorimetric assay kits (BioSource, USA) according to the manufacture's instruction.

On day 35, following blood collection, all animals were sacrificed by diethyl ether and liver samples were harvested, fixed in 10% buffered formalin, embedded in paraffin, sectioned at 5 µm, and stained with hematoxylin and eosin (H&E) for evaluation under light microscopy (40).

RESULTS

Body Weight

Figure (1) shows the Body weight levels in rats were given lead acetate treatment (LA) which shows a reduction in body weight expressed by a significant value (P<0.05) when compared to control, Sodium butyrate (SB) group a significant (P<0.05) is increased in the SB group in comparison with the LA group and the combination (SB+LA) after 35 days.

Total Serum Protein

Figure 2A shows the total serum protein (TSP) values in LA-treated rats had a significant (P<0.05) increase in TSP activity compared to the control and SB-treated groups and a combination of lead acetate with sodium butyrate (SB+LA) were decreased significantly (P<0.05). The results in Figure 2B shows the serum albumin activity of the control and treated groups during the experiment. After 35 days of exposure to lead acetate, albumin significantly (P<0.05) decreases when compared to the control and SB-treated group. Figure 2C shows serum globulin activity elevated considerably (P<0.05) in rats exposed to LA in comparison to control and SB-treated groups, the results also showed that the SB group shows non-significant differences to the end of the treatment when compared to the control group.

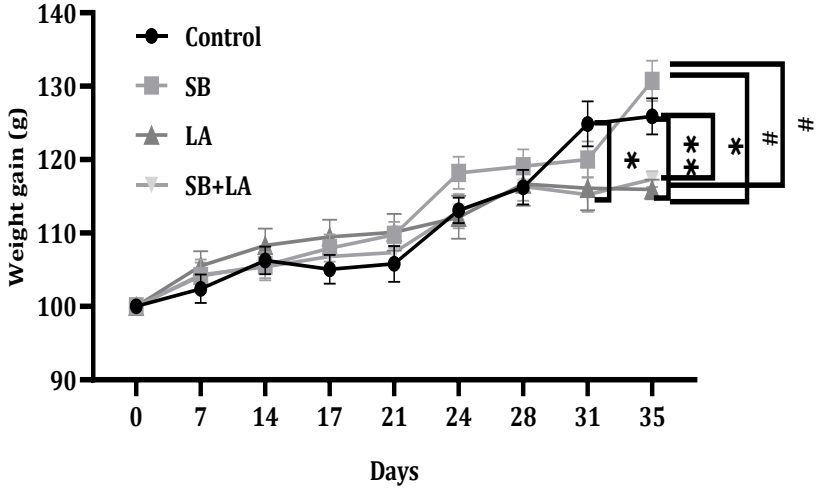

Figure 1. Effect of sodium butyrate, lead acetate, and their combination on body weight gain in adult female rats. Mean±SEM, n=10. Control, rats received only drinking water; SB, rats orally received sodium butyrate at 200 mg/kg BW; LA, rats orally received lead acetate at 50 mg/kg BW; SB+LA, rats orally received both sodium butyrate at 200 and lead acetate at 50 mg/kg BW. *P≤0.05, **P≤0.01, ***P≤0.001, #P≤0.0001

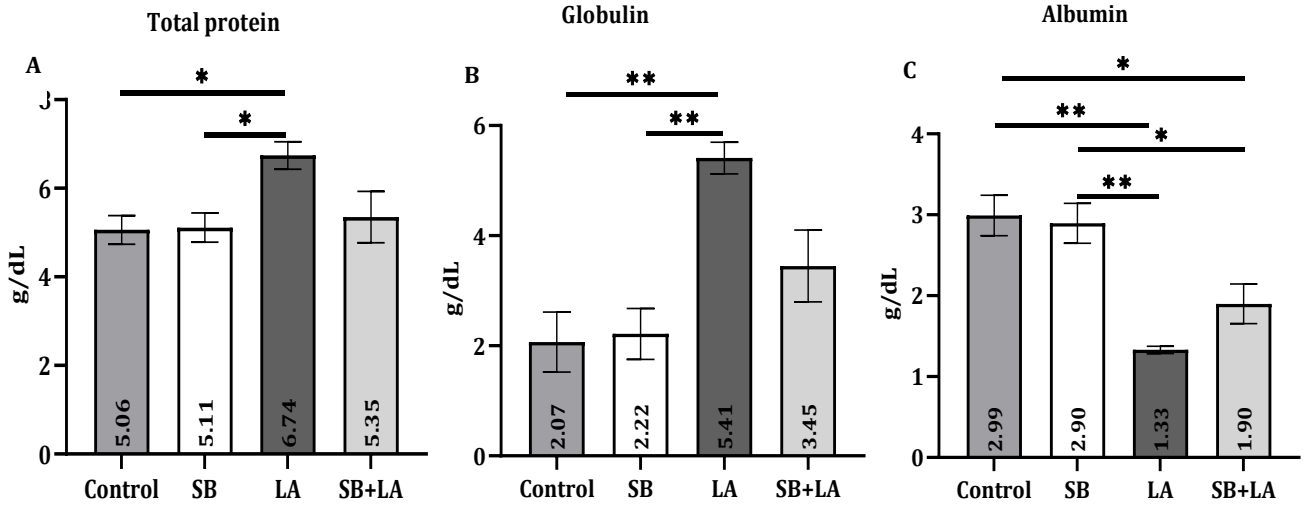

Figure 2. Effect of sodium butyrate, lead acetate, and their combination on (**A**) serum total protein (g/dL), (**B**) serum albumin (g/dL), and (**C**) serum globulin in adult female rats. Mean±SEM, n=10. Control, rats received only drinking water; SB, rats orally received sodium butyrate at 200 mg/kg BW; LA, rats orally received lead acetate at 50 mg/kg BW; SB+LA, rats orally received both sodium butyrate at 200 and lead acetate at 50 mg/kg BW. *P≤0.05, **P≤0.01, ***P≤0.001, #P≤0.0001

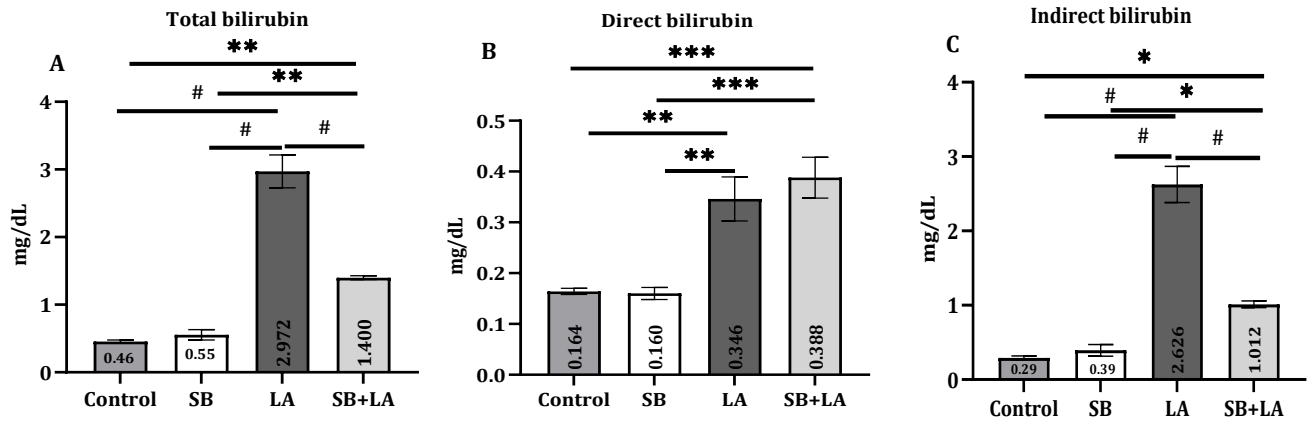

Figure 3. Effect of sodium butyrate, Lead acetate, and their combination on (**A**) total serum bilirubin (mg/dL), (**B**) direct serum bilirubin (g/dL), and (**C**) indirect serum bilirubin (mg/dL) in adult female rats. Mean±SEM, n=10. Control, rats received only drinking water; SB, rats orally received sodium butyrate at 200 mg/kg BW; LA, rats orally received lead acetate at 50 mg/kg BW; SB+LA, rats orally received both sodium butyrate at 200 and lead acetate at 50 mg/kg BW. **P*≤0.05, ***P*≤0.01, ***P≤0.001, #*P*≤0.0001

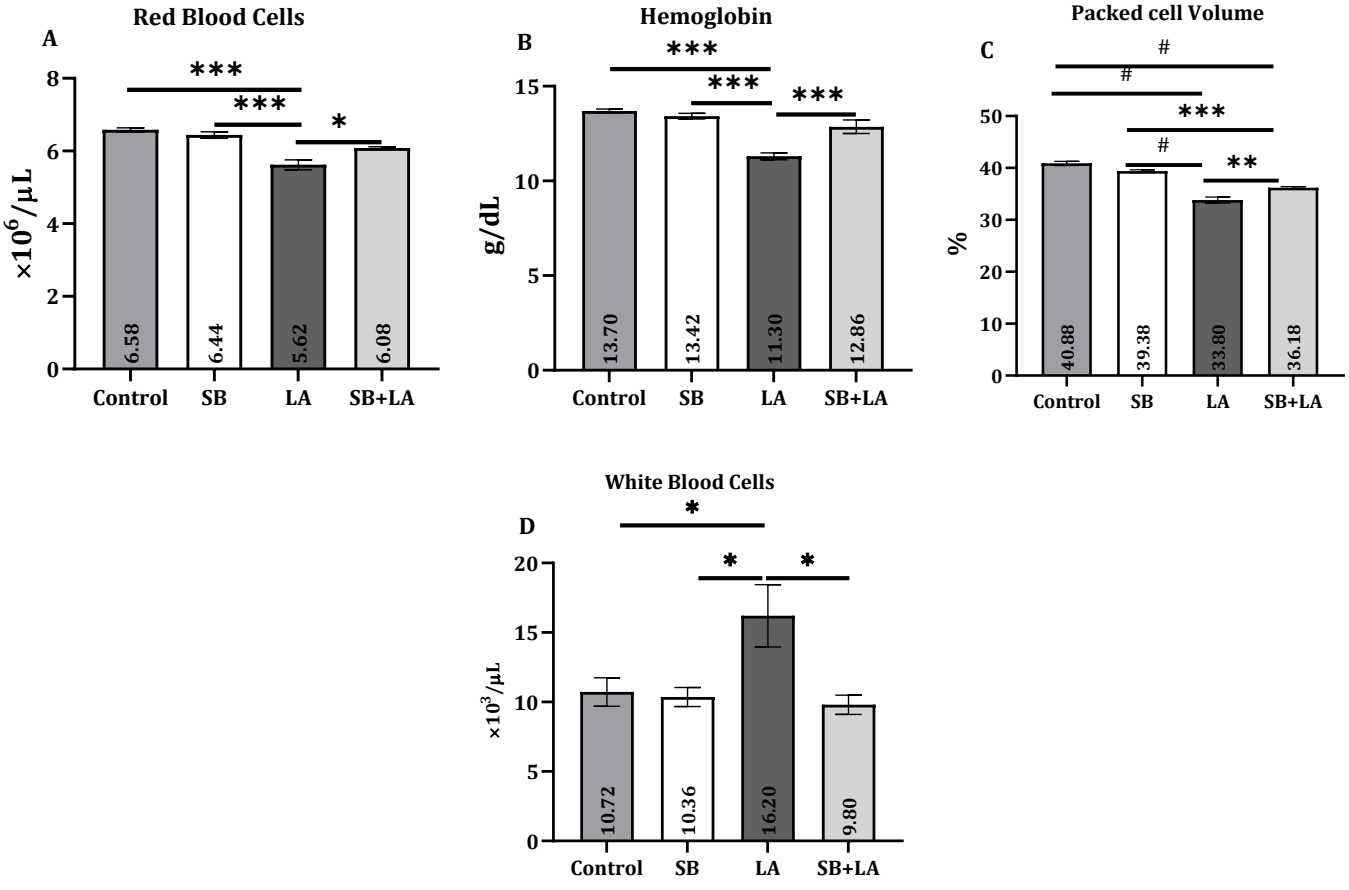

Figure 4. Effect of sodium butyrate, Lead acetate, and their combination on (**A**) total red blood cells count (×10⁶ µL), (**B**) hemoglobin (g/dL), (**C**) pocket cell volume (g/dL), and (**D**) total white blood cells count ((×10³ µL) in adult female rats. Mean±SEM, n=10. Control, rats received only drinking water; SB, rats orally received sodium butyrate at 200 mg/kg BW; LA, rats orally received lead acetate at 50 mg/kg BW; SB+LA, rats orally received both sodium butyrate at 200 and lead acetate at 50 mg/kg BW. **P*≤0.05, ***P*≤0.01, ***P≤0.001, #*P*≤0.0001

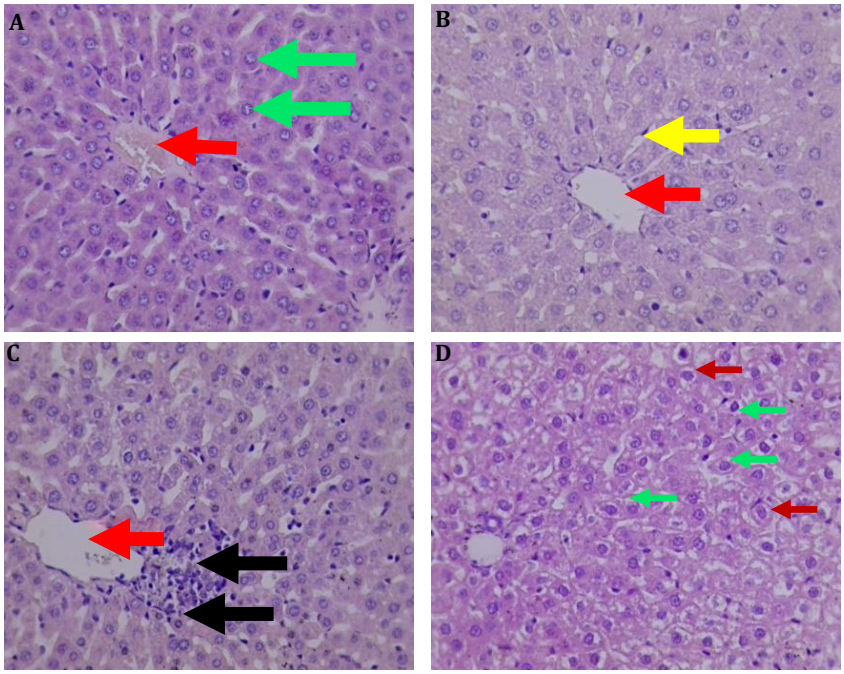

Figure 5. Representative histology images of liver from (**A**) control rats showing no-clear pathological lesion appears in the structure of the liver with normal congestion of blood vessels and normal hepatocyte, (**B**) rats orally given 200 mg/kg BW sodium butyrate showing normal hepatocyte without pathological changes, (**C**) rats orally given 50 mg/kg BW lead acetate showing pre-vascular mild filtration of the nuclear cell (macrophage) and lymphocyte, (**D**) rats orally given both sodium butyrate (200 mg/kg BW) and lead acetate (50 mg/kg BW) showing cellular swelling with loss of sinusoid. H&E, 40×

Total Serum Bilirubin

Results of current study showed that there was a significant increase ($P<0.0001$) in total serum bilirubin (TSB) in LA-treated group compared with the control and SB-treated groups (Table 3A). TSB was significantly decreased ($P<0.0001$) in the SB+LA-treated group compared with the LA-treated group. Additionally, oral administration of LA significantly increased ($P<0.0001$) the serum direct bilirubin concentration in comparison with the control and SB-treated groups, but not with SB+LA-treated group (Figure 3B). There was a significant increase ($P<0.0001$) in serum indirect bilirubin in the LA-treated group when compared to the control, SB, and combination group (Figure 3C

Complete Blood Count

The results showed that rats orally exposed to LA at 50 mg/kg BW significantly reduced ($P<0.001$) the red cells count, and hemoglobin compared to control; and treatment groups (Figure 4A, B). Compared to control and SB-treated groups, significantly lower ($P<0.001$) pocket cell volume was recorded in LA and SB+LA-treated groups (Figure 4C)

Figure 4D shows that there was a significant elevation ($^{*}P<0.05$) in WBC in rats given LA compared with the control, SB, and combination groups.

Histopathological Study of the Liver

In the livers of control rats, histopathological examination shows a normal histological section, there is no-clear pathological lesion appears in the structure of the liver with normal congestion of blood vessels, normal hepatocyte, and normal hepatic cord (Figure 5A). Meanwhile, SB rats in figure (5B) shows the normal histological structure of hepatocytes, no-clear pathological lesions appear in the structure of the liver with normal congestion of blood vessels. Histopathological sections of the liver of rats were given 50 mg/kg. BW of LA showed pre-vascular mild filtration of the nuclear cell (Macrophage and Lymphocyte) with slight Keratolysis (Figure 5C). Sections in the liver of rats gavaged with both (SB+LA) show acute cellular swelling with loss of sinusoid and rounded edges of the hepatocyte (Figure 45D).

DISCUSSION

Lead intoxication has substantially inferior on the health of normal rats, which was seen when rats were exposed to lead, their body weight was dramatically reduced (24). Butyrate or its salt form had been shown to have a favorable influence on body weight gain in our study and previous studies (25, 26). Dietary SB supplementation resulted in a decrease in pH in the duodenum and jejunum, which enhance digestibility and decrease pathogen burden

by adjusting the pH in the small intestine (27), which could explain the improved growth performance.

Albumin and total proteins in the plasma levels decreased as a result of exposure to lead toxicity. This suggests that changes in total soluble protein levels were linked to changes in albumin levels. These results could be explained by the suppression of albumin production by certain enzymes in cellular activities, as well as the current study's low significant excretion of hormones that can control the production of proteins. Heavy metals, such as lead, can precipitate soluble protein, and albumin in the blood serves as a transporter for the toxin (28, 33).

The considerable decline in RBC counts and Hb levels occurs concurrently with the lead acetate group. Elevation in the bilirubin level of rats indicates that some RBCs have been lysed as a result of lead intoxication, which is consistent with the increase in blood bilirubin levels brought on by lead. This could result from the heme oxygenase being induced (28, 29). This metal's restriction of heme synthesis is likely the cause of this anemia (30). RBC membrane's stress from oxidants and lipid peroxidation which is caused by lead exposure and results in hemoglobin oxidation, which enhances the conversion from hemoglobin into met-Hb and can also result in RBC hemolytic. Rats were given Lead acetate showed significantly increased leukocytosis and in situations of severe liver damage, the gamma globulin fraction is dramatically elevated while the albumin fraction is noticeably decreased (31).

Because of its link to severe jaundice and the risk of permanent brain injury at high doses, bilirubin has long been cytotoxic (32). Exposure to lead causes fatty alterations in the liver's parenchyma, which degrade hepatocytes and cause nuclear pyknosis (34). The number of apoptotic cells in the liver increases. Lead toxicity also causes cellular enlargement, mitochondrial breakage and vacuolization, and cell body wrinkling (35). Lead's damaging effects on tissues have been demonstrated to be produced mostly by oxidative stress, which interferes with cell membrane integrity, lipid peroxidation, and broad tissue destruction (36). Due to cellular disruptions, necrosis, and infiltration of inflammatory cells in the liver, lead has been shown to impair the morphologic features of hepatocytes (37). Lead poisoning has also been linked to the dilatation of sinusoids and the accumulation of red blood cells in the liver (34). SB also showed anti-inflammatory activity in the current investigation, in the SB-treated group, the histopathological investigation revealed a considerable reduction in neutrophilic infiltration (38). SB's hepatoprotective impact may be due to an improvement in the gastrointestinal barrier (39), which reduces the transmission of gut endotoxins to systemic and portal circulation. Lead acetate increases oxidative stress and causes hepatotoxicity, sodium butyrate supplement act as a treatment against the toxic effect of lead acetate on the liver via decreasing the inflammation and elevation of antioxidant agents.

Sodium butyrate supplement has a positive effect on liver function and maintains the normal physiological function of the liver against lead acetate's toxic effect.

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N/A.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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تأثير مكمل الصوديوم بيوتيريت في تقليل تسمم الكبد المحدث بواسطة خلات الرصاص في كبد الجرذان

رسل موفق أحمد، أميرة كامل محمد

فرع الفسلجة والكيمياء الحياتية و الادوية، كلية الطب البيطري، جامعة بغداد، بغداد، العراق

الخلاصة

الرصاص له مخاطر صحيه في البلدان الناميه ,وله تأثير عكسي على وظائف الكبد.البيوتريتفعال في علاج الاضطرابات الالتهابيه في الحيوانات والهدف من هذه الدراسه التحقق من قدره الصوديوم بيوتريت في تقليل تسمم الكبد بواسطه خلات الرصاص في هذا البحث ,40 من جرذان الالبينو الاناث البالغه قسمت الى اربع مجاميع متساوية في تجربه استمرت 35 يوم مجموعه1 الكونترول, مجموعه جرعت صوديوم بيوتيريت بجرعه 200ملغم\كغم يوميا، مجموعه3 اعطيت خلات الرصاص بجرعة 50ملغم/كغم وزن الجسم تجريع فموي يوميا,المجموعه 4 اعطيت كلاهما الصوديوم وخلات الرصاص .جمعت عينات دم لعمل صور دم كامله وعينات مصل الدم لقياس البليروبين الكلي البليروبين المباشر وغير المباشر,البروتين الكلي,الالبومين ,الكلوبيولين وتحضير الكبد للفحص النسيجي. الجرذان المعرضه لخلات الرصاص اظهرت زياده حقيقيه في الكلوبيولين ,البليروبين الكلي ,البروتين الكلي في مصل الدم وكريات الدم البيض مع نقصان في كريات الدم الحمراء, الهيموغلوبين, وحجم الخلايا المرصوصه، اما الوزن فاظهر نقصان واضح في هذه المجموعه, نسيجيا لوحض ارتشاح وعائي للخلايا الالتهابيه وزن جسم الجرذان التي جرعت الصوديوم بيوتيريت لوحظ زياده كبيره ,تحسن في كرياث الدم الحمر, الهيموغلوبين, وحجم الخلايا المرصوصه مع تركيب نسيجي طبيعي للكبد وعدم وجود اذى مرضي في خلايا الكبد. في المجموعه الرابعه لوحظ نقصان واضح في البيليروبين الكلي والبيليروبين الغير مباشر وكريات الدم البيض اما باقي الاختبارات في هذه المجموعه اظهرت نتائج قريبه من مجموعه الكونترول نتيجه لتأثير الصوديوم بيوتريتز. يمكن الاستنتاج ان تناول الصوديوم بيوتيريت يقلل من فعاليه التأثير المؤذي لخلات الرصاص ومنع تلف الكبد.

الكلمات المفتاحية: خلات الرصاص، حمض البيوتيريك، بيليروبين،كلوبيولين.