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Antioxidant effect of L-carnitine on experimentally induced acute toxic lesion of rabbits with Acetaminophen (paracetamol)

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Article info

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The prospects of the use of L-carnitine as an antioxidant to treat acute toxic conditions caused by administering high doses of Acetaminophen (paracetamol) were analyzed in the paper. Rabbits were chosen as the objects of the study. Thus, the aim of this research was to determine the effect of Acetaminophen (paracetamol) acute toxicity on several indicators of rabbits' bloodstream and the role of L-carnitine as an antioxidant against paracetamol toxicity (hepatotoxicity). The experiment was conducted at the Faculty of Medical Laboratory Technology, University of Al-Zahrawi from the period of 7/11/2024 to 01/02/2025.Ten male rabbits of local (hybrid) breeds, weighing 1-1.5 kilograms, aged 6-11 months, were divided into two groups and kept in special cages. All animals were examined to make sure that they were free of injuries and pathologies before the experiment began, and anti-helminthic Albendazole 3 % and the injection solution of Ivermectin (0.1 ml/rabbit) preparations were administered to the rabbits. To prevent eimeriosis, the animals were treated with Amprolium, 0.6 ml/l of drinking water for 4 days. Group 1 (n=5) was the control one; medicines were not used for the rabbits of this group. The animals in group 2 (n=5) were treated with Acetaminophen (paracetamol) at an acute toxic dose (2000 mg/kg), after that they were treated with L-carnitine (250 mg/kg) for three weeks. During the experiment, the experimental and control groups of animals had the same keeping conditions and the identical diet. As a result of the conducted studies, a significant increase in the bloodstream of the liver enzymes (ALT by 45.8%, AST by 54.0%) was found when treated with a toxic dose of Acetaminophen (paracetamol) at the background of the sharp decrease in the erythrocytes and leucocytes number and hemoglobin level (by 20.0, 18.7, and 40.3%, respectively). It was determined that the use of L-carnitine as an anti-oxidant means resulted in the normalization of ALT and AST levels in the bloodstream as well as the number of erythrocytes and leucocytes and the level of hemoglobin. In particular, on the 21st day of the research, the ALT and AST indicators decreased in the blood serum, and in the blood, the amount of the erythrocytes and leucocytes and hemoglobin level increased (by 16.3, 21.9, and 29.6%, respectively) compared to the indicators after Acetaminophen administration.

Keywords: Acetaminophen (paracetamol), blood parameters, hepatotoxicity, L-carnitine.

Антиоксидантні властивості L-карнітину за умови експериментального токсичного ураження кролів ацетамінофеном (парацетамолом)

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У роботі проаналізовано перспективність використання L-карнітину як антиоксидантного засобу за гострих токсичних станів викликаних введенням підвищених доз ацетамінофену (парацетамолу). В якості об'єкта дослідження було обрано кролів. За мету роботи поставлено визначити впливу гострої токсичності ацетамінофену (парацетамолу) на окремі показники кровоносного русла кролів та встановлення ролі L-карнітину як антиоксиданту за отруєння ацетамінофеном. Експеримент проводився на факультеті медичної лабораторної техніки Університету Аль-Захраві, у період з 11.07.2024 по 01.02.2025 років. Для досліду використано десять самців кролів місцевих порід, вагою в межах 1–1,5 кілограма та віком від 6 до 11 місяців. Тварини за принципом аналогів були розділені на дві групи. Обидві групи упродовж досліду утримувалися у спеціальних клітках. Всіх тварин перед проведенням досліду обстежили на наявність травм та патологій, з профілактичною метою проведено антигельмінтні обробки з використанням Альбендазолу 3 % та ін'єкційного розчину Івермектину. З метою виключення еймеріозу, тваринам протягом 4 днів з питною водою використовували Ампроліум. Кролів з 1 групи обрано як контрольну групу (лікарських препаратів не отримували). У кролів 2 групи для досліду було викликано гостру токсичність ацетамінофеном (парацетамолом). Препарат застосовували у дозі 2000 мг/кг після чого, кролям упродовж трьох тижнів в якості антиоксидантного засобу використовували L-карнітином (250 мг/кг). Дослідна та контрольна групи тварин під час експерименту мали однакові умови утримання та однаковий раціон. В результаті проведених досліджень встановлено, що введення кролям токсичної дози ацетамінофену (парацетамолу) викликало у кровоносному руслі дослідних тварин значне підвищення рівня печінкових ферментів – АЛаТ та АсАТ (на 45,8 та 54,0 % відповідно) на фоні різкого зниження кількості еритроцитів, лейкоцитів та рівня гемоглобіну (на 20,0, 18,7 та 40,3 % відповідно). Визначено, що застосування в якості антиоксидантного засобу L-карнітину призводило до нормалізації в кровоносному руслі рівня АлАТ та АсАТ а також кількості еритроцитів, лейкоцитів й гемоглобіну. Зокрема, на 21 добу дослідження у сироватці крові відбулося зниження АлАТ та АсАТ (на 31,3 та 50,6 % відповідно) а в крові підвищення кількості еритроцитів, лейкоцитів та рівня гемоглобіну (на 16,3, 21,9 та 29,6 % відповідно) порівняно до показників після введення ацетамінофену.

Ключові слова: ацетамінофен (парацетамол), показники крові, гепатотоксичність, L-карнітин.

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Introduction

The top concern for physicians is hepatotoxicity as a result of administering pharmaceutical products. A biomarker is a measurable, quantifiable characteristic that is used to diagnose a disease or assess how well a treatment is working. A variety of biomarkers are employed to identify liver lesion and detect liver disease [1]. To diagnose liver disease (toxicity), we must measure liver enzymes, especially aminotransferase, which includes AST and ALT. They serve as indicators of hepatocellular lesion. By catalyzing the transfer of amino groups from aspartic acid or alanine to ketoglutaric acid, they contribute to gluconeogenesis by generating pyruvic acid and oxaloacetic acid, respectively [2]. Acetaminophen (paracetamol) use has been associated with liver failure, which has resulted in liver transplantation or death in some cases. Acetaminophen (paracetamol) hepatotoxicity is usually associated with large dosages of Acetaminophen (paracetamol) that exceed the authorized maximum dose [3]. This effect could be caused by taking more than one medicine that contains Acetaminophen (paracetamol) as a component. Regular Acetaminophen (paracetamol) use has also been linked to liver lesions in individuals. There are many antioxidants against toxic materials like vitamin C [4], β-glucan [5], quercetin [6], *Olea europaea* [7], L-carnitine [8]. L-carnitine has antioxidant properties that have been discovered through its ability to protect antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (G-px) from further peroxidative damage [8], lipid peroxidation (LPO) [9], and DNA damage [10]. By lowering the generation of hydrogen peroxide (H2O2), L-carnitine has a preventive effect against LPO [11]. It is linked to the buffering of excessive acyl-CoA, which can be harmful to cells [8]. L-carnitine vital nutrient (L-3-hydroxy-4-N-N-N-trimethylaminobutyrate) is used by the body to turn fat into energy. It transports fatty acids through the inner mitochondrial membrane so that they can undergo β-oxidation later on [12]. It is also an antioxidant that reduces metabolic stress in the cells. The studies have reported that L-carnitine has an effective 1.1-diphenyl-2-picryl-hydrazyl (DPPH) free radical scavenging, superoxide anion radical scavenging, hydrogen peroxide scavenging, and the total reducing power [13].

The aim of the study

The aim of the study was to determine the effect of acute toxicity of Acetaminophen (paracetamol) on hepatotoxicity and the role of L-carnitine as an antioxidant against paracetamol toxicity (hepatotoxicity).

Materials and methods

Materials and Chemicals

The animals were administered prophylactically with an internal, hepatic, and intestinal, Albendazole 3 % anti-helminthic preparation and Ivermectin 0.1 ml/rabbit was injected under the skin to prevent internal and external worms. The rabbits were also injected with Amprolium at a dose of 0.6 ml/liter of drinking water for 4 days to prevent eimeriosis. Those doses as preventive ones did not induce toxicity [14].

Study Area

The experiment was conducted at the College of Medical Laboratory Technology, University of Al-Zahrawi, from the period 7/11/2024 to 02/01/2025.

Study Design

Ten male rabbits, local (hybrid) breeds, weighing 1–1.5 kg, aged 6–11 months, were divided into two groups and kept in special cages. All animals were examined to make sure that they were free of injuries and pathologies before. The experiment started with G1 treated with water and pellets; this group was used as the control one. G2 was treated with Acetaminophen (paracetamol), an acute single toxic dose (2000 mg/kg) [15] orally for one day until the toxic signs appeared (vomiting, anoxia, nausea, and convulsions. Then the rabbits in G 2 were treated with L-carnitine (250mg/kg) [16] orally for three weeks.

Blood Tests

Three milliliters of blood were taken from all the experimental animals directly from the heart [17] at the start of the experiment, after the toxic dose, and finally after 3 weeks of L-carnitine treatment (the end of the experiment). Tubes containing anticoagulants were used for blood parameters. A collection of blood in tubes without anticoagulant (gel tubes) was then centrifuged at 3,000 cycles for 20 minutes to separate blood serum that was used to conduct laboratory tests.

1. Complete Blood Count (CBC).

A blood sample was taken and placed in the Urit-2900 device, and the measurement was done automatically.

2. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST).

After putting a serum sample into the DC-40-Mindray apparatus, the measurement was carried out automatically.

Statistical Analysis

The SAS (Statistical Analysis System, version 9.1) was used to statistically analyze the experimental data. For Experiment 2, the one-way ANOVA was used, and least significant differences (LSD) were employed to determine whether there were any significant differences between the group means. $P<0.05$ was regarded as statistically significant, and the findings were presented as mean ± standard errors [18].

Ethic Statement

The research was approved by the ethics committee of the University of Kerbala, College of Veterinary Medicine, under the number UOK.VET.HE.2024.089.

Results and discussion

1 The effect of toxic doses of Acetaminophen (paracetamol) and L-carnitine on alanine aminotransferase (ALT) (IU/L):

The effects of oral supplementation of treated L-carnitine and toxic dose of Acetaminophen (paracetamol) on male rabbits at libitum are shown in ***Table 1***.

Table 1
The effect of Acetaminophen (paracetamol) toxic dose and L-carnitine on alanine aminotransferase, IU/L.

Parameters of groups	Mean ± SE			LSD Values
	zero time start of the experiment	toxic dose of paracetamol for G2 only	three weeks after L-carnitine treated	
G1	45±1.14[a]	44.4±1.4[a]	43.8±0.8[a]	3.6
G2	45±1.14[b]	83±4.37[a]	57±6.26[b]	13.74

Note: letters indicate a significant difference between periods in each group in the horizontal direction. Values represent mean ± SE (N=10).

G1 does not significantly differ in any of the ensuing areas. G2 exhibits a notable rise in toxicity followed by a notable decline in response to L-carnitine treatment.

2. The effect of Acetaminophen (paracetamol) toxic dose and L-carnitine on aspartate aminotransferase (AST) (IU/L):

The effects of oral supplementation of L-carnitine treatment and toxic dose of Acetaminophen (paracetamol) on male rabbits at libitum are shown in ***Table 2***.

Table 2
The effect of Acetaminophen (paracetamol) toxic dose and L-carnitine on aspartate aminotransferase (AST) (IU/L)

Parameters of groups	Mean ± SE			LSD values
	zero time start of the experiment	toxic dose of paracetamol for G2 only	three weeks after L-carnitine treatment	
G1	40.2±1.4[a]	36.6±2.2[a]	36.6±1.8[a]	5.6
G2	30±1.73[b]	66.8±5.53[a]	33±1.64[b]	10.7

Note: letters indicate a significant difference between periods in each group in the horizontal direction. Values represent mean ± SE (N=10).

G1 does not significantly differ in any of the ensuing areas. G2 exhibits a notable rise in toxicity followed by a notable decline in response to L-carnitine treatment.

3. The effect of Acetaminophen (paracetamol) toxic doses and L-carnitine on erythrocytes, packed cell volume, hemoglobin, and leucocytes:

The results show a significant decrease in the blood parameter (RBCs, Hb, PCV, and WBCs) when treated with the toxic dose of Acetaminophen (paracetamol), then followed by a significant increase in the blood indicators (RBCs, Hb, PCV, and WBCs) when treated with L-carnitine. The results are represented in ***Table 3***.

Table 3
The effect of Acetaminophen (paracetamol) toxic dose and L-carnitine on blood parameters in rabbits

Parameters *in group*	Mean ± SE			LSD values
	zero time start of the experiment	toxic dose of paracetamol for G2 only	three weeks after L-carnitine treatment	
RBCs, 10^6/mm^3 *G1*	6.5± 0.32[a]	6.5±0.39[a]	6.5±0.25[a]	1.02
RBCs, 10^6/mm^3 *G2*	6.5±0.73[a]	5.2±0.19[b]	6.2±0.33[a]	1.08
PCV, % *G1*	41.0±1.72[a]	41.2±2.24[a]	41.2±1.82[a]	5.99
PCV, % *G2*	41.0±1.44[a]	35.5±1.91[a]	41.9±0.78[a]	4.78
Hb, g/dL *G1*	12.5±0.57[a]	12.6±0.51[a]	12.6±0.50[a]	1.63
Hb, g/dL *G2*	12.3± 0.57[a]	10.0±0.30[b]	12.8±0.47[a]	1.43
WBCs, 10^3/mm^3 *G1*	6.3±0.69[a]	6.2±0.64[a]	6.4±0.61[a]	2.00
WBCs, 10^3/mm^3 *G2*	6.4±1.55[a]	3.8±1.08[b]	5.4±1.07[a]	1.73

Note: RBCs – red blood cells (erythrocytes), PCV – packed cell volume, Hb – hemoglobin, WBCs – white blood cells (leucocytes); letters indicate a significant difference between periods in each group in the horizontal direction; Values represent mean ± SE (N=10).

As a result of the conducted research, there were no significant differences between the animals in G1 in all periods in all parameters, while there was a significant decrease in all parameters in the animals of G2 after being treated with Acetaminophen (paracetamol) toxic dose, then a significant increase in all parameters after being treated with L-carnitine for two weeks.

What happened to rabbits after being given a toxic dose of paracetamol 2000 mg/kg after 24 hours of pathological symptoms, which were similar to the signs observed by Roth et al. (1999) [19]. A high rate of toxicity in rabbits' blood led to the loss of appetite, lethargy, and imbalance, and as the liver is the main center for paracetamol metabolism, the increase in its dose leads to the destruction of hepatocytes, which provide ready food for the body from the energy stored in the liver, causing the appetite disruption and idleness .Vomiting is caused by intestinal problems

caused by liver impairment and the change in food metabolism [20, 21].

The increase in the level of AST and ALT may be attributed to the hepatotoxicity of paracetamol 2000 mg/kg, which leads to the breakdown of hepatocytes secreting these enzymes present in the cytosol of the hepatocyte into the bloodstream [22], and this indicates that the toxic dose of paracetamol led to injury of the liver [23]. The beneficial effects of L-carnitine supplementation on lipid profile, inflammatory biomarkers, and oxidative stress have been demonstrated in previous studies [24].

L-carnitine participates in the transportation of long-chain fatty acids from the cytoplasm in liver cells to the mitochondria and thus increases the carnitine supplementation, which resulted in a significant decrease in AST, ALT and GGT levels. Consistent with our findings, the previous studies have shown an increase in carnitine content in the liver, especially in patients with liver diseases. A review [25] noted that L-carnitine is involved in the regulation of hepatotoxic processes induced by valproic acid, and L-carnitine supplementation had a protective effect against hepatotoxicity. In another study, L-carnitine increased tissue survival and survival after hepatocyte injury [26].

In an experimental study, the administration of L-carnitine resulted in preservation of liver enzymes (ALT, AST, and GGT) after the induction of hepatocellular injury [27].

Oxidative stress plays an important role in the pathogenesis of paracetamol-induced liver injury [28]. This study showed that paracetamol poisoning caused adverse effects on hematopoietic organs as represented by a decrease in hematological parameters including RBC number, Hb content, PCV %, TLC, platelet amount and neutrophil. These findings are consistent with those of Desnoyers (2000) [29], who showed that the changes in the analyzed blood parameters could be due to the oxidative stress caused by paracetamol, which has a damaging effect on the immune and circulatory organs and red blood cells. Paracetamol inhibits hematopoiesis and causes hematotoxicity, mainly methemoglobinemia and hemolytic anemia. This may be due to the destruction of red blood cells because of increased lipid peroxidation in cell membranes [30]. The disorder and anemia can be overcome by the proposed role of L-carnitine in improving erythrocyte survival by improving erythrocyte membrane stability [31]. Matsumura et al., 1996 [32] found a significant opposite correlation between the total serum carnitine concentration and erythrocyte fragility and weekly maintenance doses of erythropoietin in 26 patients. The studies have shown that supplementation of L-carnitine to maintenance dialysis patients was associated with improvements in red blood cell deformation, membrane stability and hematocrit [33]. There is more data in the literature to support a trial of L-carnitine in anemic patients on maintaining dialysis requiring very high doses of erythropoietin, although the evidence is inconclusive due to the small sample size, small effect size and the lack of treatment times. There are several randomized, placebo-controlled trials that evaluated the effect of L-carnitine on hemoglobin or hematocrit. One study was conducted before erythropoietin became available, and all patients received folate, vitamin B12, and sodium ferric gluconate at the end of each dialysis session, in addition to a placebo or L-carnitine, for 12 months [34].

Conclusions

There are negative effects of acute toxic doses of Acetaminophen (paracetamol) on liver enzymes (AST and ALT), as they increase, indicating liver toxicity. Moreover, hematological parameters (RBCs, WBCs, PCV, and Hb) decrease, indicating the oxidative effect of paracetamol toxicity. There are positive effects of L-carnitine in treating acute toxic effects of acetaminophen (paracetamol), which ensures antioxidant effects against free radicals. For that, we recommended to avoid taking Acetaminophen (paracetamol) in high doses, on the other hand, using L-carnitine to treat toxic doses and oxidative stress.

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Conflict of interest

The author state that there is no conflict of interest.

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