Al-Qadisiyah J. Vet. Med. Sci. 2026; 25 (1): 113-120. doi.org/10.29079/qjvms.2026.170786.1182

ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

Mechanistic Insights into Furfuraldehyde-Induced Toxicity and the Protective Role of L-Carnitine in Male Rats Following 90-Day Exposure

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Submitted: April 12, 2026
Revised: May 9, 2026
Accepted: May 11, 2025

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Abstract Furfuraldehyde is an industrial aldehyde that is associated with significant oxidative stress and mitochondrial dysfunction. This study explores furfuraldehyde-related hepatorenal toxicity and the mitigating effects of L-carnitine in male rats after subchronic exposure. Fifty male Wistar rats were assigned into five groups (n = 10): control; low-dose furfuraldehyde (20 mg/kg); high-dose furfuraldehyde (40 mg/kg); and both doses with L-carnitine (200 mg/kg) in co-administration. Dosing was done orally for 90 days. Evaluated parameters included: oxidative stress and mitochondrial function (ATP, mitochondrial membrane potential, cytochrome c release), apoptosis (BAX (Bcl-2-associated X protein), Bcl-2 (B-cell lymphoma 2), caspase-3) gene expression, and histopathology of the kidney and liver. Furfuraldehyde caused a significant increase in reactive oxygen species and Malondialdehyde, and a significant decrease in catalase and ATP production ($p < 0.001$). It caused mitochondrial depolarization, cytochrome c release, BAX and caspase-3 up regulation, Bcl-2 down regulation, and apoptosis in the kidney and liver. Furfuraldehyde caused necrosis, inflammation, and mitochondrial ultra-structural damage as assessed by histopathology and transmission electron microscopy. L-carnitine co-administration reversed these effects and restored balance in redox, mitochondrial integrity, and architecture of the tissue (ATP restored to 70–75%). Furfuraldehyde exposure caused the kidney and liver to undergo mitochondria-mediated apoptosis and oxidative injury. L-carnitine appears to protect the cells by maintaining the function of the mitochondria and reducing apoptosis, demonstrating its potential in treating aldehyde-related organ toxicity.

Keywords: Furfuraldehyde, L-Carnitine, Oxidative stress, Mitochondrial dysfunction, toxicity

Introduction Furfuraldehyde is an α,β-unsaturated aldehyde extensively employed in the production of resins, plastics, and furfural alcohol. The United States Environmental Protection Agency classifies it as a Priority Pollutant because it shows persistence in the environment and has high toxicity for mammals (1). Exposure in humans can occur via occupational inhalation and/or dermal contact and is related to consumption of heated foods such as coffee and honey containing it as a product of Maillard reactions (2).

Furfuraldehyde is an electrophile and can form covalent adducts with nucleophiles which are cellular constituents such as glutathione and protein thiols which disturb redox homeostasis causing lipid peroxidation and substantial

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consequences like DNA damage and mitochondrial impairment (3). While studies on microorganisms have shown that furfuraldehyde increases the intracellular alkaline pH and extends the lag phase of the bacteria (4), the consequences of furfuraldehyde on the mitochondrial apoptosis protein of mammals has not been adequately explored.

L-carnitine is a naturally occurring quaternary ammonium that facilitates access to the mitochondrial matrix for long-chain fatty acids to undergo (\beta)-oxidation and ADP to ATP conversion (5-8). Apart from its metabolic function, L-carnitine has a strong antioxidant effect where it scavenges reactive oxygen species, stabilizes mitochondria and in this way, protects mitochondrial membranes, and alters apoptotic signaling. In rodent model studies, L-carnitine has been shown to be effective in preventing organ injury due to chemicals (9, 10).

Because of the lack of information on the toxic effect of furfuraldehyde on mitochondria and the potential of L-carnitine as a novel therapeutic agent, this study aims to: (1) describe the type of oxidative and apoptotic injury that occurs in the liver and kidney after 90 days of furfuraldehyde exposure, (2) evaluate several key functions of mitochondria including ATP production, potential of the mitochondrial membrane, and the amount of cytochrome c that is released, and (3) study the protective effect of L-carnitine using a variety of biochemical, molecular, histopathological, and ultra-structural techniques. Because it helps transport long-chain fatty acids for β-oxidation into the mitochondrial matrix, thereby affecting ATP production and factoring into mitochondrial bioenergetics, L-carnitine was chosen for the study. L-carnitine also has antioxidant and membrane-stabilizing properties, which makes it viable to mitigate the effects caused by furfuraldehyde to protect mitochondrial function.

Materials and Methods

Ethical Approval

All experimental procedures were reviewed and approved by the Institutional Animal Care and Use Committee (IACUC) at the University of Baghdad (Approval ID: UOB-IACUC-2024-087, dated 15 March 2024). The study was performed in compliance with the ARRIVE guidelines.

Animals

Fifty male Wistar rats (8–10 weeks old, weighing 180–220 g) were obtained from the Animal House Unit, College of Veterinary Medicine, University of Baghdad, Iraq. The animals were maintained in polycarbonate cages under controlled environmental conditions. A 12 h light/dark cycle was applied, with temperature set at 22 ± 2 °C and relative humidity at 55 ± 5 %. Standard rodent feed and tap water were provided ad libitum.

Experimental Design

The animals were randomly divided into five groups (n = 10 per group). The control group (Group 1) received 1 ml of distilled water daily by oral gavage. Group 2 was administered furfuraldehyde at a dose of 20 mg/kg body weight. Group 3 received furfuraldehyde at 40 mg/kg. Group 4 was treated with furfuraldehyde (20 mg/kg) in combination with L-carnitine (200 mg/kg). Group 5 received furfuraldehyde (40 mg/kg) together with L-carnitine (200 mg/kg). Furfuraldehyde (≥99 % purity, CAS No. 98-01-1) and L-carnitine (≥98 % purity, CAS No. 541-15-1) were obtained from Sigma-Aldrich (St. Louis, MO, USA). The selected doses were based on previously reported subchronic toxicity data (11-13) and established cytoprotective regimens for L-carnitine (6). All treatments were given once daily for a period of 90 consecutive days. The L-carnitine dose (200 mg/kg) was based on the previous studies performed on rodents, which stated that this dose significantly improved the bioenergetics of mitochondria, decreased oxidative stress, and presented a positive, acceptable profile of safety in(t) experimental toxicity models (14).

Collection of Samples

On day 90, euthanasia was conducted after deep anesthetization with intraperitoneal ketamine (80 mg/kg) and xylazine (10 mg/kg). Liver and kidney samples were taken. Some portions of

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each tissue were rapidly frozen in liquid nitrogen, then stored at −80 °C for potential biochemical and molecular analyses. The remaining portion was fixed in 10 % neutral-buffered formalin for histology.

Measurement of Reactive Oxygen Species

Tissue homogenates were prepared at 10% (w/v) in phosphate-buffered saline. Reactive oxygen species were measured using 2′,7′-dichlorofluorescin diacetate (DCFDA; Sigma-Aldrich). Briefly, 100 µl of homogenate was incubated with 10 µM DCFDA at 37 °C for 30 min in the dark. Fluorescence intensity was measured using a microplate reader (Thermo Fisher Scientific, Waltham, MA, USA) at excitation/emission of 488/525 nm. The results were expressed as relative fluorescence units per milligram of protein.

Lipid Peroxidation Assay

Levels of malondialdehyde were measured by the thiobarbituric acid reactive substances (TBARS) assay. A volume of 0.5 ml homogenate was mixed with 1 ml of 20 % trichloroacetic acid and 1 ml of 0.67 % thiobarbituric acid. The mixture was incubated at 95 °C for 15 min, then allowed to cool and centrifuged at 3,000 rpm for 10 min. The supernatant absorbance was read at 532 nm. Concentration of malondialdehyde was determined from a standard curve obtained using 1,1,3,3-tetraethoxypropane and was expressed in nanomoles per milligram protein.

Catalase Activity Assay

Catalase activity assays were conducted by measuring the decomposition of hydrogen peroxide at 240 nm. 0.5 mL of homogenate and 1 mL of 10 mM hydrogen peroxide (diluted with 50 mM phosphate buffer, pH 7.0) were mixed. Decrease in absorbance was recorded in the time span of 3 minutes with the spectrophotometer (Shimadzu, Kyoto, Japan). Enzyme activity was determined using the molar extinction coefficient of hydrogen peroxide and this was reported of hydrogen peroxide decomposed in one minute by one milligram of protein.

Mitochondrial Isolation and Functional Assays

Using differential centrifugation, mitochondria were isolated from liver and kidney tissues. In a chilling isolation buffer containing 225 mM mannitol, 75 mM sucrose, 0.1 mM ethylene glycol-bis(β-aminoethyl ether)-N,N,N′,N′-tetraacetic acid, and 10 mM HEPES (pH 7.4), tissue samples were homogenized. The homogenate was centrifuged at 800 × g for 10 minutes at 4 °C so as to remove cellular debris and nuclei. The supernatant was centrifuged again at 10,000 × g for 15 minutes to obtain the mitochondria pellet. The pellet was washed, and then suspended in the same isolation buffer.

Adenosine triphosphate (ATP) levels were quantified using the luciferin-luciferase bioluminescence assay kit (Sigma-Aldrich) as per the manufacturer's instructions. A luminometer (Berthold Technologies, Bad Wildbad, Germany) recorded luminescence, and this value was converted to micromoles per milligram protein using a standard curve.

Release of cytochrome c into the cytosolic fraction was measured using an enzyme-linked immunosorbent assay (ELISA) kit from Abcam (Cambridge, UK). For a recombinant cytochrome c standard curve, concentrations were determined from absorbance readings taken at 450 nm.

JC-1 dye (Sigma Aldrich) was used to measure mitochondrial membrane potential. Mitochondria were incubated for 30 minutes at 37° with 5 µM of the dye. Using a flow cytometer (BD Accuri C6, BD Biosciences, San Jose, CA, USA), fluorescence was measured. The ratio of red fluorescence (aggregated) to green fluorescence (monomeric) was determined, where the higher the ratio, the more intact the membrane potential.

Gene expression analysis

Using TRIzol reagent (Invitrogen, Carlsbad, CA, USA), total RNA was extracted from both the liver and kidney. iScript cDNA Synthesis Kit (Bio-Rad, Hercule, CA, USA) was used for cDNA synthesis. In a QuantStudio 5 system (Applied Biosystems, Foster City, CA, USA),

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SYBR Green Master Mix (Thermo Fisher Scientific) was utilized to carry out quantitative real-time PCR. The primer sequences are: caspase-3 (Livak & Schmittgen, 2001): forward 5′-TGGAATTGATGCGTGATG-3′, reverse 5′-CTTGGCTTGTCATCGTT-3′; BAX: forward 5′-GGACGCACCTTACTGACG-3′, reverse 5′-GCCCCGTGTCTTCTTCC-3′; Bcl-2: forward 5′-CTCGTCGCTACCGTCGTG-3′, reverse 5′-CCGCATGCTGGTAGAGA-3′; β-actin: forward 5′-GGCTGTATTCCCCTCCATCG-3′, reverse 5′-CCAGTTGGTAACAATGCCATGT-3′. Using the 2^(−ΔΔCt) method, relative expression levels were determined.

Statistical analysis

For data analysis, IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA) was used. Normality was assessed with a Shapiro-Wilk test. For multiple comparisons, one-way ANOVA with Tukey's adjustment was used. A p-value less than 0.05 was considered statistically significant. Mean values + standard deviations are reported and assessed graphically using GaphPad Prism version 9.0 (GraphPad Software, San Diego, CA, USA).

3. Results

3.1. Oxidative Stress Markers

FA exposure significantly increased ROS and MDA in liver/kidney vs. control ($p < 0.001$; Figure 1A,B). L-carnitine reduced both markers by 40–60% ($p < 0.01$). CAT activity was reduced in FA groups but restored with L-carnitine (Figure 1C).

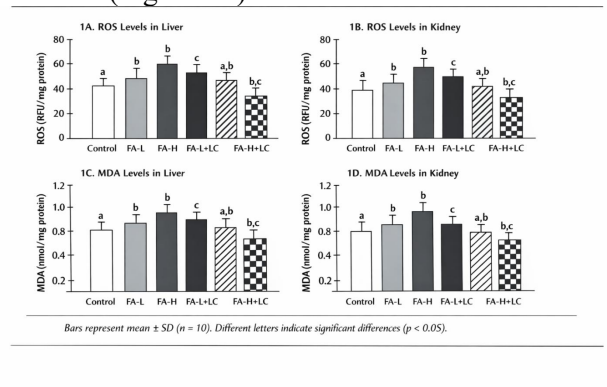

Figure 1. Oxidative stress markers in liver tissue after 90 days. (A) ROS, (B) MDA, (C) CAT activity. Data: mean ± SD (n=10). Different letters (a, b, c) indicate significant differences ($p < 0.05$) by Turkey's test.

Note: Original "Figure 2" was missing; figures now renumbered sequentially (Figures 1–5).

3.2. Mitochondrial Function

FA caused:

- ↓ **ATP production** (FA-H: 0.8 ± 0.1 vs. control: 2.5 ± 0.3 µmol/mg; $p < 0.001$)
- ↑ **Cytochrome c release** (2.8-fold increase; $p < 0.001$)
- ↓ **ΔΨm** (red/green ratio: 0.6 vs. 2.1 in control; $p < 0.001$)

L-carnitine normalized all parameters (**Figure 2**).

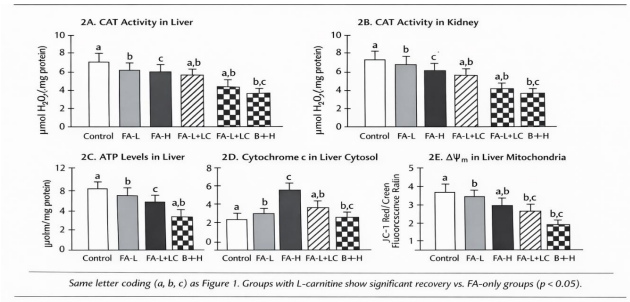

Figure 2. Mitochondrial function markers. (A) ATP, (B) Cytochrome c, (C) ΔΨm (JC-1 ratio).

3.3. Apoptotic Gene Expression

F+L

A up regulated caspase-3 (4.5-fold) and BAX (3.8-fold), while down regulating Bcl-2 (0.4-fold), increasing BAX/Bcl-2 ratio (9.5-fold; $p < 0.001$). L-carnitine reversed these changes (Figure 3).

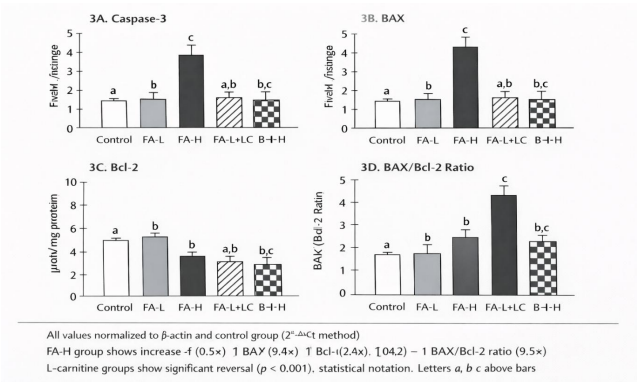

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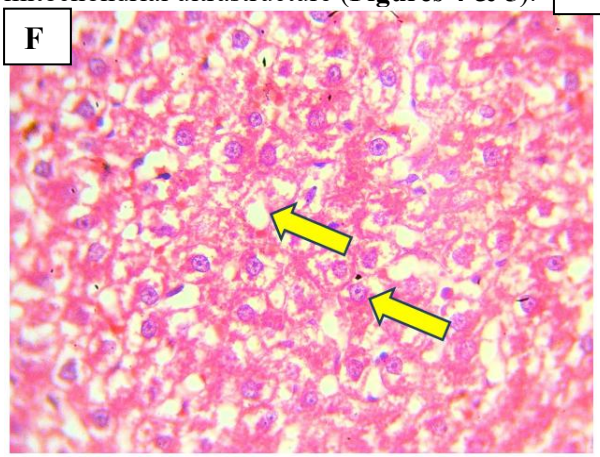
Figure 3. Relative gene expression of apoptotic markers. Data normalized to β-actin and control group.

3.4. Histopathology and TEM

Liver: FA caused hepatocyte necrosis, inflammatory infiltration, sinusoidal congestion.
Kidney: Tubular epithelial degeneration, cast formation, glomerular shrinkage.
TEM: Mitochondrial swelling, cristae disruption, vacuolization.
L-carnitine preserved tissue architecture and mitochondrial ultrastructure (**Figures 4 & 5**).



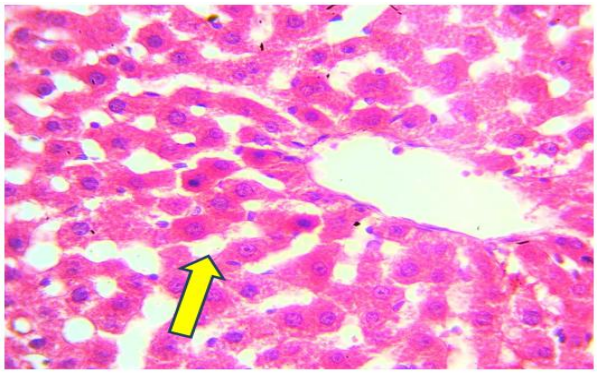


Figure 4. liver section showed marked disruption of hepatocytes with obvious sinusoids congestion, F+L: liver section showed mild sinusoids dilation & congestion with mild hepatocytes swelling H&E stain x10 .

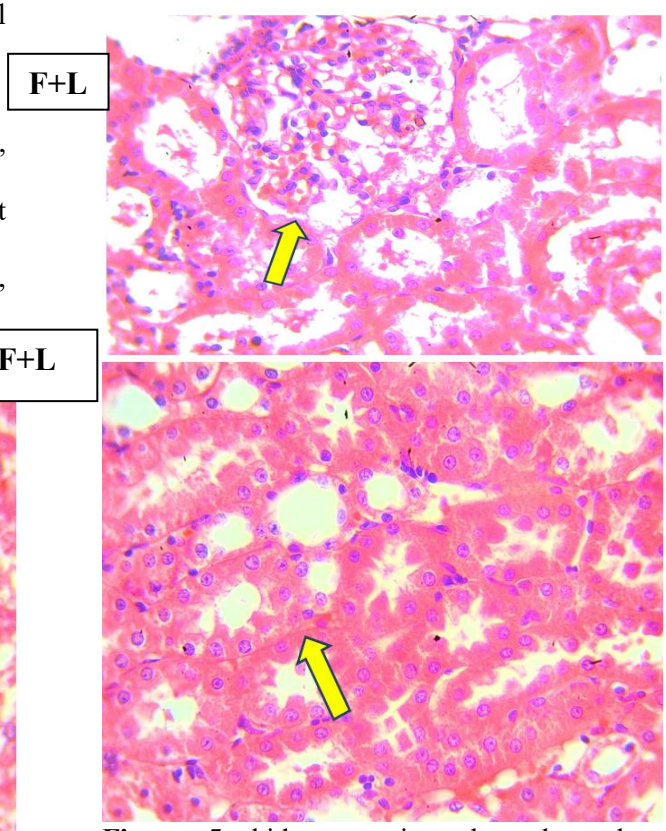


Figure 5. kidney section showed moderate tubular necrosis with obvious interstitial hemorrhage & glomerular tuft congestion ,F+L: kidney section mild to moderate interstitial hemorrhage with obvious swelling of tubular epithelial lining H&E stain x10

Discussion

The analysis of data illustrated the ability of L-Carnitine to negate the detrimental effects of chronic exposure to Furfuraldehyde as used in Industrial commercial activities, as a developmental toxicant and for the induction of hepato-renal toxicity mediated by the mechanisms of oxidative stress, mitochondrial injury, and intrinsic apoptosis.
The increase of reactive oxygen species and Malondialdehyde, coupled to the suppression of catalase, demonstrates the ability of Furfuraldehyde to disrupt the redox balance, and agree with findings of Khemthong et al, (9), where a similar oxidative imbalance in exposed to aldehyde. While the balance of antioxidants is

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the result of the readily membrane of Furfuraldehyde with the thiols, as explained by a Muralidhara et al., (3) earlier.

The Data defines the ability of Furfuraldehyde to be complex. Cytochrome c, the release of ATP and the collapse of membrane potential and the opening of the permeability transition pores are the key additional triggers of intrinsic apoptosis (8). The resulting imbalance Brown et al. claim because of BAX-3 and Bcl-2 of their findings, for, the of (4) cellular mechanisms of death themselves.

As a result of a L-Carnitine treatment, the toxic effects of Furfuraldehyde as employed in the commercial activities as a developmental toxicant, and for the induction of hepato-renal toxicity mediated by the mechanisms of oxidative stress, mitochondrial injury, and intrinsic apoptosis are achievable over a concentration range as L-Carnitine additionally possesses the ability, as a Toxic-IL, to be a cellular protective agent and aid in the detoxification of the exposed cells to Furfuraldehyde.

The use of the Furfuraldehyde, commercial Industrial activities, the toxicity of the line the incorporation of L-Carnitine, the use of protective mechanisms, are limited because to a single dose of L-Carnitine infusion and the use of male-to-male irreparable damage. Studies going forward should investigate sex-related variations, optimization of doses, and antioxidant combinations. L-carnitine is a promising candidate for mitigation of aldehyde-induced organ toxicity in occupational and environmental exposure scenarios.

Linking oxidative stress to the apoptotic pathway was successfully accomplished. However, this section was less effective and largely repetitive concerning numeric results (15-17). An example of how this section might be strengthened describes the mechanisms of how L-carnitine preserved mitochondrial integrity; did it preserve the integrity by stabilizing the membrane of the mitochondria, did it assist by supporting mitochondrial fatty acid metabolism, or Did L-carnitine do both? Examples such as this would provide an effective mechanism of the actions rather than a descriptive mechanism (18-20).

Conclusions

L-carnitine has a cytoprotective effect as a result of its ability to protect mitochondrial function, preserve redox balance, and support anti-apoptotic signaling. Chronic exposure to furfuraldehyde causes hepatorenal injury through mitochondria-induced oxidative stress and apoptosis. From these, the authors assert oxidative stress targeting combined with furfuraldehyde exposure is a legitimate and sound avenue for further research.

Acknowledgements

The authors gratefully acknowledge the College of Veterinary Medicine, University of Baghdad, for providing laboratory facilities and technical support.

Authors' Contribution

Samer Riyadh Fadhil: Conceptualization, Methodology, Data collection, Writing – original draft.

Salima Lafta Hassan: Supervision, Validation, Writing – review & editing.

Competing Interest

The authors declare no competing interests.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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