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Original article

Integrated Molecular, Histological, and Physiological Evaluation of the Effects of Energy Drink on Liver and Kidney Tissues

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Abstract
In recent years, the consumption of energy drinks (EDs) has experienced a worrying increase worldwide; they have been associated with many health issues, such as elevated blood pressure, cardiovascular difficulties, head pain, disturbed sleep, drug overuse, tension and excitement, and liver and kidney toxicity. The purpose of the current research is to estimate the impact of ED consumption at the different doses on renal and hepatic functions in rats. Thirty male Wistar rats were randomly assigned to three groups (10 rats per group). The first group (control) received distilled water. Two groups received ED at different doses (0.5 and 3 mL/kg body weight) for 30-days. All treatments were administered by oral gavage. At the end of the experimental period, blood samples were collected to assay liver and kidney function tests. Liver and kidney samples were processed for histological evaluation. Exposure to ED at different doses induced highly significant increases in serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and blood urea nitrogen (BUN) levels compared with the control group ($P < 0.001$). In contrast, serum creatinine (CREA) levels showed no statistically significant difference compared with the control group ($P = 0.900$). Furthermore, in comparison to controls, exposure to these two ED doses. Pronounced histopathological changes were observed in hepatic and renal tissues of the ED-treated rats. In conclusion, the investigation discovered that energy drink consumption significantly elevates hepatocyte function markers (ALT, AST, and ALP) and kidney injury markers (UREA and CREA) and tissue damage. These findings point to potential adverse liver and renal effects that should be considered in the development of regulatory policies on energy drink consumption.
Keywords. Energy Drinks, Liver, Kidney, Rats, Toxicity, Renal Function, Hepatic Function.

Introduction

Energy drinks (EDs) first appeared in Europe and Asia in the 1960s due to consumer demands for dietary supplements that provide energy [1]. (EDs) are drinks marketed to increase energy levels, improve cognitive function, and enhance physical performance [2]. These drinks commonly contain a combination of components such as caffeine (50–500 mg per serving), glucuronolactone (600–1200 mg), taurine (1000–2000 mg), guarana (which provides extra caffeine ginseng (200–400 mg), and sugars (20–40 g), among other substances [3].

The consumption of these ingredients can lead to harmful health effects, with the most prevalent being increased blood pressure and heart rate, therefore elevating the risk of cardiovascular events in susceptible individuals [4]. Moreover, there have been reports of Corrected QT Interval prolongation following intake, which could make consumers more vulnerable to cardiac arrhythmias [5]. Studies indicate that abundant intake of energy drinks can trigger insomnia, depression, and anxiety [6,7], as well as withdrawal symptoms upon stopping use [8]. In addition, there have also been reports of damage and toxicity of the liver [9,10], with some severe cases requiring transplantation [11]. The kidneys and liver are essential organs that help keep the body in balance and handle detoxification. Earlier research has suggested that drinking too many EDs could have a harmful impact on both kidney and liver function. For example, high caffeine intake has been associated with increased urination, dehydration, and possible kidney injury [12]. In addition, the high sugar load and other additives found in EDs may play a role in liver inflammation and fat buildup, or steatosis [13].

Still, the exact ways EDs affect kidney and liver health aren't fully clear, and more research is needed to sort out the mechanisms involved and how effects change with different doses. Several animal studies have looked into how caffeine or certain ED ingredients influence kidney and liver function. However, there are still few broad studies that assess the impact of full ED formulations on these organs, particularly with careful dose-dependent comparisons. This study set out to evaluate how ED consumption affects kidney and liver function in rats. We expected that ED intake would cause unfavorable changes in renal and hepatic function, and that the extent of these effects would depend on the dose. We also planned to assess the histopathological changes in kidney and liver tissues from rats treated with EDs.

Materials and methods

Animals

Thirty male Wistar rats (8 weeks old, weighing 200-250g) were obtained from the animal house of Tripoli University. Before being tested, the rats were housed in standard cages (5 rats per cage) under controlled environmental conditions (22 ± 2 °C, 55 ± 5% humidity, 12-hour light/dark cycle). The experiments were carried out in line with the recommendations of International Laboratory Animal Use and Care [14].

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Drugs and Chemicals

Energy Drink

The brand of ED used in this study was "Red Bull", a product of Rauch Trading AG, Switzerland (manufactured for Red Bull GmbH, Austria). It was purchased from a local store in Tripoli, Libya.The ED was diluted in distilled water to achieve the desired concentrations.

Treatments

For the purpose of evaluating object identification, three groups of ten rats each were randomly assigned. For aduration of thirty days, rats were administered oral gavage with each of the following treatments.

- Group 1 (G1): distilled water (1 ml/100 g bw) was given to this normal control group.
- Group 2 (G2): received aLow dose of the energy drink at (0.5 mL/kg body weight).
- Group 3 (G3): received a high dose of the energy drinkat (3 mL/kg body weight).

The energy drink was administered orally once daily for 30 days. The doses were chosen based on [15].

Sample Collection

At the end of the 30-days treatment period, the rats were anesthetized with ketamine (50 mg/kg) and xylazine (5 mg/kg). Blood samples were collected from the retro-orbital plexus into serum separator tubes. The blood samples were centrifuged at 3000 rpm for 10 minutes, and the serum was stored at -80 °C until analysis. After blood collection, the rats were euthanized by cervical dislocation.

Histological Examination

At the end of the experiment, all rats were sacrificed by decapitation. Liver and kidney samples were immediately excised and processed for light microscopy and histological investigation using standard methods. Specimens were fixed in 10% neutral formalin and stained with hematoxylin and eosin.

Biochemical Analysis

Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), urea, and creatinine levels were measured using commercially available kits (Roche Diagnostics, Germany) on an automated biochemical analyzer (Cobas c311, Roche Diagnostics).

Statistical analysis

Data were expressed as mean ± standard deviation (SD). Statistical analysis was performed using SPSS version 25.0. One-way analysis of variance (ANOVA) followed by Tukey's post-hoc test was used to compare the means of different groups. A p-value < 0.05 was considered statistically significant.

Results

Liver function

The effects of ED consumption on serum liver enzyme activities are showed in (Table 1). Significant differences were demonstrated among the experimental groups for all measured liver enzymes. The activity of alanine aminotransferase (ALT) recorded a significant increase corresponding to the escalation of ED dose. In the control group, the mean ALT level was 36 ± 2.5 U/L, which increased to 50 ± 5 U/L in the low-dose group and then to 74 ± 1 U/L in the high-dose group of ED. Also, aspartate aminotransferase (AST) activity exhibited a marked increase across the study groups (P = 0.027). In comparison with the control group, the low dose group of ED increased AST activity to 154 ± 6.2 U/L, whereas the high dose group recorded the highest mean value (162 ± 8.7 U/L) in AST activity. Similarly, Alkaline phosphatase (ALP) activity was observed with increasing doses of ED. Mean ALP levels escalated from 128 ± 4.5 U/L in the control group to 182 ± 9 U/L in the low-dose group of ED, reaching 319 ± 2.5 U/L in the high-dose group of ED. These increases were highly statistically significant (P < 0.001), indicating a dose-dependent elevation in ALP activity.

Table 1. Effect of energy drink consumption on serum liver enzyme activities.

Groups	AST (U/L) Mean ± SE	ALP (U/L) Mean ± SE	ALT (U/L) Mean ± SE
Control	137.00 ± 10.00	128.00 ± 4.50	36.00 ± 2.50
Low dose	154.00 ± 6.20	182.00 ± 9.00	50.00 ± 5.00
High dose	162.00 ± 8.70	319.00 ± 2.50	74.00 ± 1.00
P-value (P)	0.027	0.000086	0.00002
Decision	*Sig (P < 0.05)**	*Ex-Sig* (P < 0.001)***	*Ex-Sig* (P < 0.001)***

Data are expressed as mean ± SE (N=10 in each group), Sig = Significant (P < 0.05), Very-Sig** = Very significant (P < 0.01), Ex-Sig*** = Extremely significant (P < 0.001) versus those of the control group.*

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Kidney function

The effects of ED consumption on kidney function parameters are presented in (Table 2). The measured renal function markers included serum creatinine and urea levels. No significant differences were noticed in serum creatinine levels between the experimental groups (P = 0.900). In the control group, the mean creatinine level was 0.44 ± 0.16, while the low-dose and high-dose groups of ED recorded mean values of 0.43 ± 0.07 and 0.47 ± 0.04, respectively. In contrast, serum urea levels exhibited a significant increase among the study groups (P = 0.0018). The control group had the mean urea level at 25 ± 1.9, which elevated to 42 ± 0.65 in the low-dose group of ED and reached 46 ± 3.4 in the high-dose group of ED, indicating a dose-dependent elevation in serum urea concentration.

Table 2 Effect of energy drink consumption on kidney function parameters creatinine and urea.

Groups	Creatinine (U/L) Mean ± SE	Urea (U/L) Mean ± SE
Control	0.44 ± 0.16	25.00 ± 1.90
Low dose	0.43 ± 0.07	42.00 ± 0.65
High dose	0.47 ± 0.04	46.00 ± 3.40
P-value (P)	0.900	0.0018
Decision	*N.S. (P > 0.05)*	*Very-Sig** (P < 0.01)*

Data are expressed as mean ± S.E (N=10 in each group), N.S. = non-significant. Sig = Significant (P < 0.05), Very-Sig** = Very significant (P < 0.01), versus those of the control group.*

Histopathological Analysis

The liver and kidney were examined histologically using hematoxylin and eosin staining to evaluate potential morphological alterations associated with the administration of ED at different doses (low dose of 0.5 ml/kg and high dose of 3 ml/kg).

Histopathology of liver and kidney in the control group

Liver tissue

Liver sections from the control group showed a normal lobular arrangement with a clearly defined central vein and regularly aligned hepatic cords. Hepatocytes appeared polygonal with uniform cytoplasm and centrally placed nuclei. Sinusoids were narrow and evenly distributed and contained macrophages (Kupffer cells), without congestion or dilation. No inflammatory cells, necrosis, or degenerative changes were observed. The overall architecture was well preserved and consistent with normal liver histology (Figure 1 a& b).

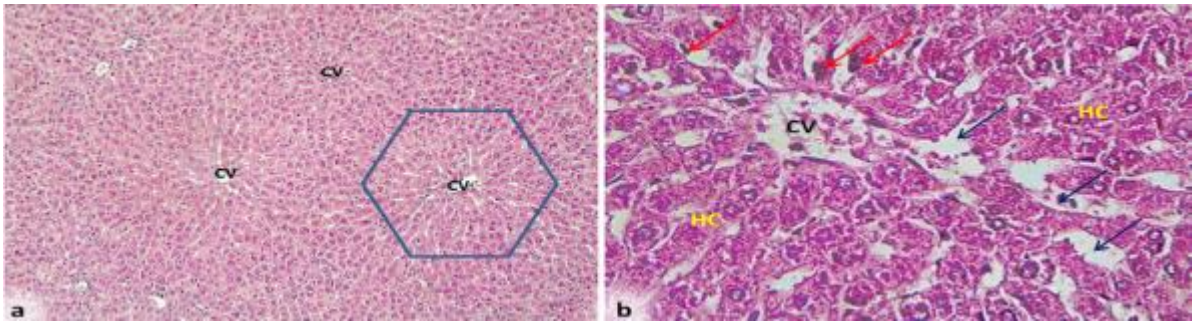

Figure 1; a) Normal hepatic architecture showing hepatic lobule (blue), a well-defined central vein (CV), (H&E,100x). b) Regularly arranged hepatic cords (HC), and intact hepatocytes with uniform cytoplasm. Sinusoids appear narrow with Kupffer cells (red arrows), (H&E,400x).

Kidney tissue

Kidney sections from the control group showed a normal cortical arrangement with well-defined glomeruli and intact renal tubules. Glomerular tufts appeared compact with clear Bowman's spaces. The proximal and distal tubules displayed uniform epithelial lining and narrow lumens, with no evidence of degeneration or congestion. The interstitial tissue was unremarkable, without inflammatory cells or edema. Overall, the renal architecture was preserved and consistent with normal kidney histology (Figure 2 a &b).

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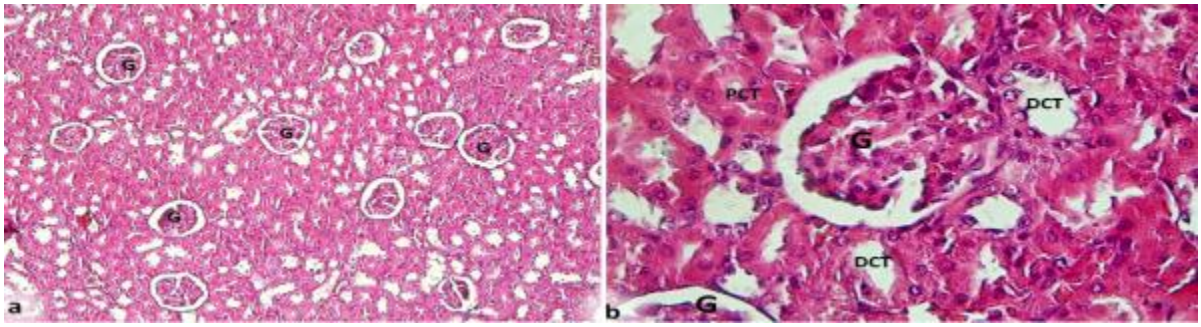

Figure 2; a) Normal renal architecture showing glomeruli (G), (H&E,100x). b) A well-defined glomerulus (G), proximal convoluted tubule (PCT), distal convoluted tubule (DCT) (H&E,400x).

Histopathology of the liver and kidney in rats that were given the low-dose group of ED:

Liver tissue

Liver sections showed a generally preserved lobular pattern with a distinct central vein in the middle of the field (Figure 4 a). Hepatocytes around the central vein appeared mostly intact, although many exhibited mild cytoplasmic vacuolation, and some central veins showed mild congestion (Figure 4b). No necrotic foci were observed, and inflammatory cells were minimal. Overall, the tissue displayed mild hepatocellular alterations consistent with early, low-grade effects of the administered drink, without evidence of severe structural damage (Figure 4a &b).

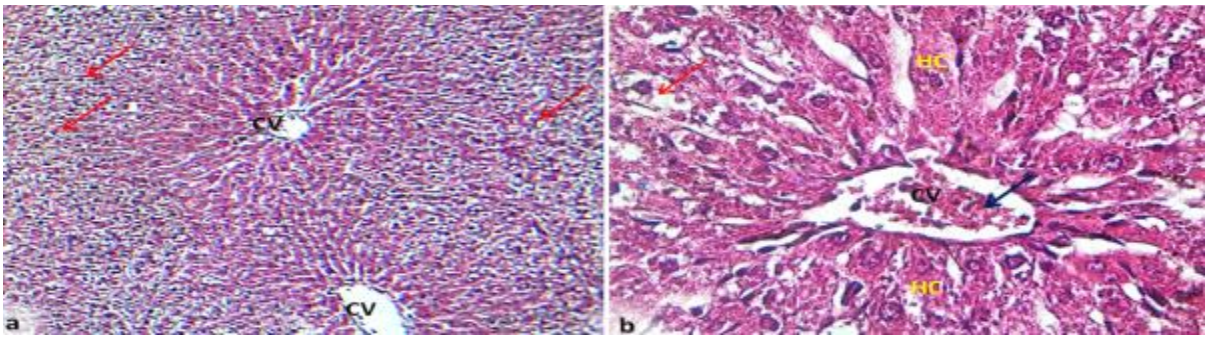

Figure 4; a) Preserved lobular pattern with a distinct central vein (CV). Hepatocytes show mild swelling and fine granular cytoplasmic changes (red arrow), along with moderate sinusoidal dilation. (H&E,100x). b) Regularly arranged hepatic cords (HC), and intact hepatocytes with uniform cytoplasm and mild congestion (H&E,400x).

Kidney tissue

Kidney sections from the same rat showed generally preserved cortical architecture with distinct renal corpuscles and well-defined glomeruli. The proximal and distal tubules appeared largely intact (Figure 5a), although some tubules exhibited mild epithelial swelling, congestion, and slight luminal dilation. Higher-magnification examination revealed focal tubular injury, including early vacuolation and mild epithelial desquamation, without evidence of extensive necrosis or significant inflammatory cell infiltration (Figure 5b). These findings indicate mild renal stress and low-grade tubular alterations associated with exposure to the low dose of ED, without major disruption of nephron structure (Figure 5a & b).

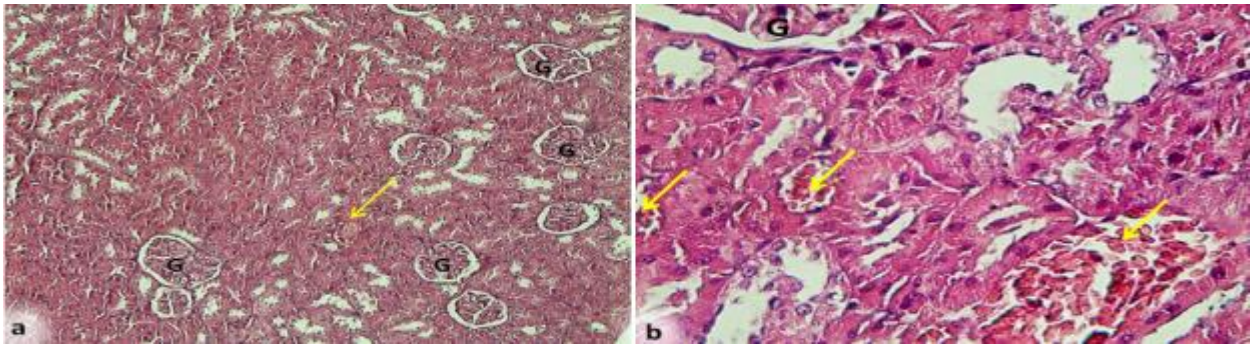

Figure 5; a) Preserved glomeruli (G). Mild interstitial congestion (yellow arrow) (H&E,100x). b) Degenerative changes as mild epithelial swelling and congestion (yellow arrow), (H&E,400x).

Histopathology of the liver and kidney in rats that were given the high-dose group of ED

Liver tissue

In the high-dose group, liver tissue displayed noticeable changes. Some central veins were dilated and congested (Figure 7 a &b).

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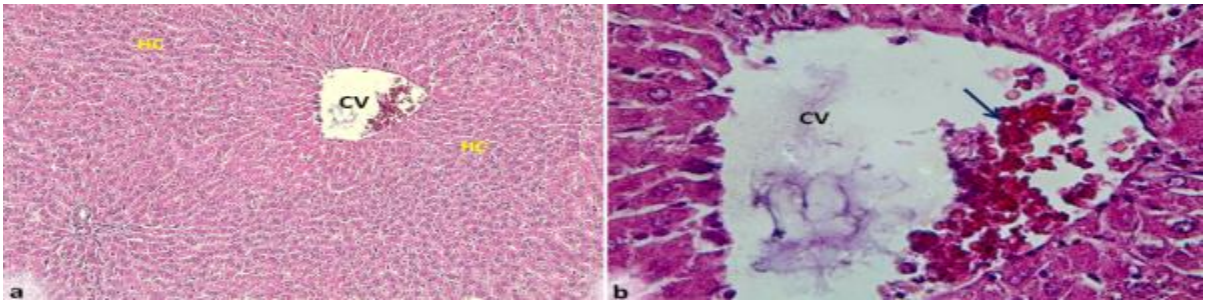

Figure 7; a) Preserved lobular pattern with a dilated, congested central vein (CV), (H&E,100x). b) Regularly arranged hepatic cords with a dilated, congested central vein (CV), (H&E, 400x).

Kidney tissue

The glomeruli and tubular epithelium showed no degenerative changes. The interstitium exhibited mild edema and congestion (Figure b a& b).

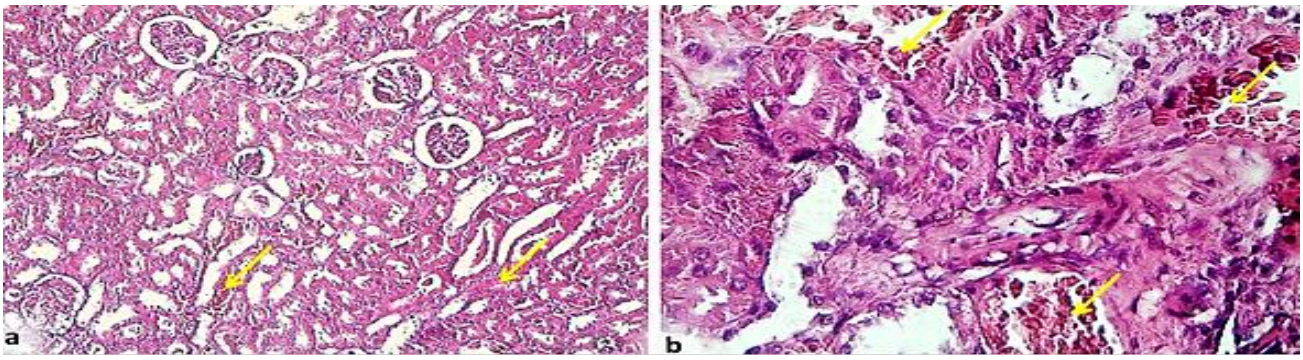

Figure 8; a) Preserved glomeruli (G). Mild interstitial congestion (yellow arrow), (H&E,100x). b) Congested interstitial space (yellow arrow), (H&E, 400x).

Discussion

This study has demonstrated that oral administration of ED to rats for 30 days resulted in varying degrees of liver and kidney damage. This was evident in the significant ED-induced elevations in serum AST, ALT, and ALP, creatinine, and urea levels. The liver and biological parameters are among the most significantly impacted areas and serve as reliable markers for liver damage due to toxic substances, especially in young people who frequently consume these beverages. This was consistent with a prior study that observed that energy drinks are carbonated beverages designed to enhance energy, enhance alertness, improve attitude, and stimulate cognitive function [16]. They typically contain a mix of ingredients, including caffeine [17,18].

Similar increases have been reported in serum AST, ALT, and ALP of rats exposed to caffeinated EDs [1]. In agreement with the current study,[19] showed that the influence of energy drinks on numerous physiological factors, including the metabolism of molecules and detoxification, is a responsibility of the liver, as well as the presence of high contents of macromolecules and other chemicals in the ED can cause severe stress on this critical organ [20, 21].

Also, the present study exhibited a significant change in the average serum urea and creatinine in rats treated with low and high doses of energy drink compared to the control group. These findings align with [23], who noted that a low dose of an energy drink can cause mild to moderate kidney damage, while higher doses may lead to more severe injury. These increases indicate impaired kidney function. These findings may be attributed to the high sugar content of energy drinks, which can increase osmotic stress on renal tissues, as well as their high caffeine content, which exerts a diuretic effect and may raise renal workload [22].

Moreover, the higher serum creatinine and urea levels seen in the treated groups point to reduced kidney function. This could be linked to the high sugar load in energy drinks, which may add osmotic stress to renal tissues, along with their elevated caffeine content, which has a diuretic effect and can increase the kidneys' workload [23]. In addition, these findings align with earlier reports highlighting the negative effects of high caffeine levels in beverages on a range of biochemical measures, including the extent of renal function impairment among induced energy-drink groups. This was reflected in experimental groups exposed to energy drink intake, where elevated urea, creatinine, and uric acid levels were observed [24, 25]. Increased serum urea and creatinine levels have been recorded in rats exposed to chronic energy drink intake, indicating renal dysfunction and possible structural damage by oxidative stress to kidney tissues [26].

The pattern of variations seen in liver and kidney function parameters of rats exposed to the different doses of ED was in agreement with the lesions in the photomicrographs of these tissues. The lesions seen here are most likely linked to the harmful effects of ED. It's fair to suggest that these changes arose from tissue injury driven by ED-induced oxidative stress. These findings line up with an earlier report showing signs of hepatotoxicity and changes in liver ultrastructure in rats given

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different types of EDs [25]. Additionally, Khayyat and colleagues reported that rats treated with EDs showed hepatic cytoplasmic vacuolations, likely from lipid droplet accumulation, which they attributed to degenerative changes within hepatocytes [27].

Conclusion

This study indicated that chronic consumption of energy drinks has hepatotoxicity, nephrotoxicity, and dose-dependent toxicity. likely due to caffeine alone or the combined toxicity of caffeine with taurine and/or sugar, depending on the drink's concentration and the length of consumption.

Conflict of interest. Nil

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