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Original Article

Histological and Biochemical Effect of Branded Energy Drink (Fearless) on the Liver and Kidney of Albino Rats

Goyang J. Tsennoe[1], Gongmen T. Tokwap[2], Solomon M. Gamde[3*], Imoh Ibanga[4], James O. Adisa[5]

[1,2,5]Department of Medical Laboratory Science, University of Jos, Nigeria
[3]Department of Medical Laboratory Science, Plateau State University Bokkos, Nigeria
[4]Federal College of Medical Laboratory Science, Jos, Plateau State, Nigeria
Email: [1)] maimakojuliana57@gmail.com, [2)] gongment@gmail.com, [3)] solomonmatthias85@gmail.com, [4)] imohrich65@gmail.com, [5)] olaefeadisa@gmail.com

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Abstract

Energy drink consumption is rising globally due to their perceived mental and physical performance benefits. These carbonated beverages contain high levels of caffeine, sugar, and various additives. This study examined the effects of the energy drink (Fearless) on liver and kidney histology and biochemical parameters in albino rats. Fifteen Rattus norvegicus (mean weight 126.3±23.9g) were divided into three groups (n=5): Group A (control) received standard feed and water; Groups B and C received 10 ml/kg and 20 ml/kg body weight/day of the energy drink for 6 weeks, respectively. The body weights of animals were measured. Following sacrifice under light anesthesia, blood samples were collected for biochemical analysis, and liver and kidney tissues were processed via paraffin wax for histological examination. Body weights of animals descriptively indicated steady increased. Biochemical analysis showed no statistically significant changes in the kidney function serum sodium, potassium, chloride, urea, and creatinine inclusive compared to controls. However, liver function parameters showed statistically significant elevations ($P<0.05$) in AST, ALT, and ALP in the test groups. Liver histology revealed central vein congestion and hepatocyte necrosis in both dose groups. The low-dose kidney sections exhibited glomerular tuft changes, polymorphonuclear inflammatory infiltration, architectural distortion, and vascular congestion, while the high-dose group showed widened Bowman's capsule space and renal tubule necrosis with inflammatory infiltration. These findings suggest that chronic energy drink consumption adversely affects liver and kidney structure and function.

Keywords: Energy Drink, Haemorrhage, Histopathology, Inflammatory Cells.

1. Introduction

In recent years, beverages known as energy drinks (EDs) have grown far more widespread and are now a popular choice around the globe, especially among adults age 35 and under, who are drawn to their capacity to enhance both cognitive and physical functioning (Vercammen et al., 2019). Energy drinks are canned or bottled carbonated beverages that contain large amounts of caffeine and sugar along with additional ingredients. Although the primary desired effects such as increased energy, enhanced physical activity, reduced mental fatigue, and improved mood are attributed to stimulants like caffeine and taurine, these same ingredients can also exert hazardous effects when consumed in excess (Gheith, 2017). Caffeine exerts its effects by antagonizing adenosine receptors, thereby boosting sympathetic outflow, whereas taurine plays a role in calcium homeostasis and osmotic regulation. Disruptions to

these processes, however, can result in tissue injury (Curran & Marczinski, 2017). There are many brands of energy drinks, including Tiger, Red Bull, Power Horse, Fearless, Predator, Monster, and Rockstar, among others. These beverages typically contain caffeine, guarana, water, carbohydrates, vitamins, amino acids, and minerals (Higgins et al., 2018). The adverse effects associated with energy drink consumption are wide-ranging. Consumption of caffeinated energy drinks may induce nephrotoxicity (Greene et al., 2014), hematological disorders (Olaleru & Odeigah, 2015), hepatitis and pancreatitis (Uwaifo, 2019). Furthermore, the high sugar content results in obesity and diabetes (Adjene et al., 2014), while disturbances in taurine homeostasis may affect the brain, cardiovascular system, and even the skeletal system (Curran & Marczinski, 2017).

Recent studies have also linked energy drink consumption to allergic disorders (Wee et al., 2021). The growing popularity of energy drinks among adolescents and young adults has raised concerns regarding their general health and well-being. Regarding caffeine content, levels can reach up to 500 mg per 20 oz. (600 mL) serving which approximately 15 times the amount found in a 12-ounce (360 mL) serving of cola (Reissig et al., 2009; Seifert et al., 2011). Beverages containing guarana may have actual caffeine levels considerably higher than those listed on the label (Reissig et al., 2009). Responsible for removing toxins such as blood urea nitrogen (BUN), creatinine, and uric acid, the kidney also plays a key part in controlling extracellular fluid volume, osmolality, electrolyte balance, and hormone production. Its basic working unit is the nephron, which consists of the glomerulus, proximal tubules, distal tubules, and collecting ducts (Gounden et al., 2024). Given that the kidney is highly susceptible to damage from exogenous toxins including components of energy drinks assessing renal histology and biochemical markers is essential for evaluating potential nephrotoxicity. Given the above, this study aimed to investigate the biochemical and histological effects of the branded energy drink (Fearless) on the liver and kidney of albino rats.

2. Methods

2.1. Chemicals

The energy drink used was Fearless. It was purchased at the terminus market of Jos, Plateau state, Nigeria.

2.1.1. Animal Welfare Provision and Ethical Clearance

Compliance with the National Academy of Science's most recent animal welfare guidelines governed how this study was conducted. The protocol underwent review by the institutional Animal Care committee, and ethical approval was obtained from the Animal House facility at the Faculty of Pharmaceutical Sciences, Department of Pharmacology, University of Jos.

2.1.2. Experimental Animals

The animal house of the Faculty of Pharmaceutical Sciences, University of Jos, provided fifteen albino rat pups aged two weeks. These animals were kept in a dedicated experimental room under suitable housing conditions. Following a seven-day adaptation phase, during which standard diet and water were freely available, the experiment commenced. The ethics committee at the Faculty of Pharmaceutical Sciences, Department of Pharmacology, University of Jos, granted ethical clearance for the animal study with the reference number F12-00379.

2.1.3. Experimental Design

A total of three groups, each containing five rats, were formed for the study (see Table 1). Group A acted as the control, with access only to water and standard diet. Group B was treated with Fearless at 10 ml/kg/rat, and Group C received Fearless at 20 ml/kg/rat. The latter dosage mirrors the volume of one bottle of Fearless typically ingested per day by an adult human with a body weight of 70-75 kg. The beverage was given daily through oral route via intragastric gavage to all animals of group B and C for a period of six (6) weeks and after completing the experimental period. All animals were painlessly sacrificed under chloroform anesthesia and subjected to standard necropsy procedures. Liver and kidney were excised and they were fixed in buffered neutral formalin (10%) for more than 24 hours. 2mls of blood was collected from each Albino rat into plain tubes and labelled appropriately. The samples were allowed to clot and later retracted and centrifuged at 3000g for 5 minutes to obtain serum. The serum samples were then transferred to another corresponding labelled plain bottles using disposable Pasteur pipette respectively. Liver function test was then carried out using spectrophotometer.

Table 1. Experimental Design

Animals' Groups	Treatment Received
Control Group A	Received normal diet and water for 6 weeks
Energy Drink Group B	Received 10mls/kg Fearless for 6 weeks
Energy Drink Group C	Received 20mls/kg Fearless for 6 weeks

2.1.4. Biochemical analysis

The spun serum of the energy drink group and control were evaluated for liver transaminases alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), as well as total protein (TP), albumin (Alb), total bilirubin (TB), direct bilirubin (DB), urea, creatinine (Cr) and electrolytes; potassium (K+), sodium (Na+), chloride (Cl-), and bicarbonate (HCO_3) were analyzed using diagnostic kits from Randox laboratory (Gamde et al., 2023).

2.1.5. Histopathology analysis

The excised fixed liver and kidney organs was taken to Central Diagnostic Laboratory, National Veterinary Research Institute, Vom, Plateau State for grossing and tissue processing. After processing, the tissue samples were embedded in paraffin and thin tissue sections of 5µm were cut using the Rotary microtome. Prior to staining, sections were dewaxed and dehydrated. Staining of the liver sections were carried out. Stained sections were dehydrated, cleared and mounted prior to microscopic examination as described by Gamde et al. 2025. Sections were examined using a light microscope. Photomicrograph were taken with a Hirocam High Resolution Optics microscope digital camera system using X10 and X40 objectives.

2.1.6. Statistical Analysis

The data are presented as means along with their standard errors (SE). Prior to conducting the one-way ANOVA, the normality assumption was verified using the Shapiro-Wilk test, and the assumption of equal variances was confirmed with Levene's test; both assumptions held true. Differences between groups were analyzed using one-way analysis of variance (ANOVA), followed by Bonferroni-adjusted pairwise comparisons. All analyses were performed with SPSS version 23.0 for Windows (SPSS Inc., Chicago, IL, USA). A threshold p-value of less than 0.05 was used to determine statistical significance.

3. Results and Discussion

3.1. Research Results

3.1.1. General Observations

As seen in table 2, animals administered the energy drink and the control showed normal fur appearance, formed stool, and food and water intake. However, animals administered with energy drink were more active.

Table 2. General Observations

Groups	Treatment	Fecal droppings	Alertness	Fur appearance	Food intake	Water intake
I	Diet & water	Dark/formed	Active	Smooth	Normal	Normal
II	10mls/kg	Dark/formed	Very active	Smooth	Normal	Increased
III	20mls/kg	Dark/formed	Very active	Smooth	Normal	Increased

3.1.2. Biochemical assessment

In this study, mean concentration of aspartate transaminase (AST), alanine transaminases (ALT), alkaline phosphatase (ALP) of the control groups compared with the low dose treatment group showed that the P-value is significant, suggesting that at lower doses the energy drink significantly alters these key indicators of liver function (Table 3). Similarly, the high dose treatment group also demonstrated significant alterations in AST, ALT, and ALP compared to the control group (Table 4). In addition, mean concentration of urea, sodium, potassium and creatinine from the control groups to the low dose treatment group showed a measure of stability and the P-value is also not significant, suggesting that at lower doses, the energy drink does not significantly alter these key indicators of kidney function (Table 5). Likewise, the high dose treatment group showed no significant difference in urea, sodium, potassium, and creatinine compared to the control group (Table 6). However, the mean chloride level was observed to be higher in the control group compared to the low-dose group, though this difference also lacked statistical significance (Table 5).

Table 3. Liver Function Test Biomarkers of Low Dose Group (10mls/kg)

Parameters	Groups	N	Mean±SD	t-test	p-value
AST IU/L	Control	4	19.50±1.91	6.062	0.001
	Low Dose	4	26.50±1.29		
ALT IU/L	Control	4	14.00±1.63	4.243	0.005
	Low Dose	4	20.00±2.31		
ALP IU/L	Control	4	58.75±3.92	3.992	0.007
	Low Dose	4	77.57±8.57		
TP g/L	Control	4	62.65±2.96	5.989	0.001
	Low Dose	4	52.43±1.70		
ALB g/L	Control	4	33.66±1.90	2.352	0.057
	Low Dose	4	30.94±1.32		
TB mg/dL	Control	4	0.50±0.10	0.884	0.411
	Low Dose	4	0.56±0.10		
DB mg/dL	Control	4	0.36±0.05	1.029	0.343
	Low Dose	4	0.43±0.12		

Key: N= Frequency, SD= Standard deviation, P-value= level of significant ALP- Alkaline phosphatase, ALT- Alanine aminotransferase and AST- Aspartate aminotransferase, ALB- Albumin, Total bilirubin -TB, Total protein -TP, Direct bilirubin -DB

Table 4. Liver Function Test Biomarkers of High Dose Group (20mls/kg)

Parameters	Groups	N	Mean±SD	t-test	p-value
AST IU/L	Control	4	19.50±1.91	14.147	0.001
	High Dose	5	36.40±1.67		
ALT IU/L	Control	4	14.00±1.63	12.097	0.001
	High Dose	5	25.80±1.30		
ALP IU/L	Control	4	58.75±3.92	3.025	0.019
	High Dose	5	102.00±28.00		
TP g/L	Control	4	62.65±2.96	2.777	0.027
	High Dose	5	51.34±7.61		
ALB g/L	Control	4	33.66±1.90	2.345	0.051
	High Dose	5	29.15±3.42		
TB mg/dL	Control	4	0.50±0.10	2.083	0.076
	High Dose	5	0.73±0.21		
DB mg/dL	Control	4	0.36±0.05	3.066	0.018
	High Dose	5	0.52±0.09		

Keys: N= Frequency, SD= Standard deviation, P-value= level of significance ALP- Alkaline phosphatase, ALT- Alanine aminotransferase and AST- Aspartate aminotransferase, ALB- Albumin, Total bilirubin -TB, Total protein -TP, Direct bilirubin -DB

Table 5. The Mean of Electrolytes, Urea, and Creatinine of Low Dose Group

Parameters	Groups	N	Mean±SD	t-test	p-value
Urea in mg/dl	Low dose	4	37.5275±1.30936	0.661	0.533
	Control group	4	38.0375±0.81798		
creatinine in mg/dl	Low dose	4	0.6075±0.04646	2.006	0.092
	Control group	4	0.5000±0.09661		
Sodium in mEq/L	Low dose	4	137.675±3.5929	0.152	0.884
	Control group	4	137.350±2.3014		
Potassium in mEq/L	Low dose	4	3.8225±0.74710	0.092	0.930
	Control group	4	3.7725±0.79529		
Chloride in mEq/L	Low dose	4	73.8275±5.53019	0.923	0.392
	Control group	4	78.0100±7.17829		

Key: N= Frequency, SD= Standard deviation, P-value= level of significant

Table 6. The Mean of Electrolytes, Urea, and Creatinine of High Dose Group

Parameters	Groups	N	Mean±SD	t-test	p-value
Urea in mg/dl	High dose	5	39.9420±2.73616	1.329	0.226
	Control group	4	38.0375±0.81798		
creatinine in mg/dl	High dose	5	0.4720±0.05215	0.560	0.593
	Control group	4	0.5000±0.9661		
Sodium in mEq/L	High dose	5	140.520±3.2275	1.648	0.143
	Control group	4	137.350±2.3014		
Potassium in mEq/L	High dose	5	3.6360±0.40648	0.337	0.746
	Control group	4	3.7725±0.79529		
Chloride in mEq/L	High dose	5	85.8500±7.45194	1.593	0.155
	Control group	4	78.0100±7.17829		

Table 7. Mean Body Weight of the Experimental Animals

Group	Day 1	Day 7	Day 14	Day 21	Day 28	Day 35	Day 42
Control	80.68g	102.28g	125.10g	131.60g	155.00g	153.00g	157.02g
Low Dose	87.38g	102.04g	117.68g	125.28g	132.94g	137.22g	155.18g
High Dose	74.24g	91.86g	106.28g	119.42g	137.40g	142.06g	144.20g

3.1.3. Histological Assessment

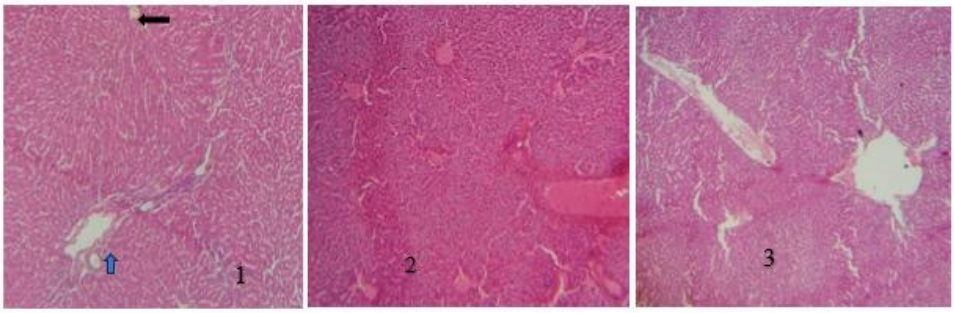

Figure 1. Plate 1, 2, and 3

Histological examination (Figure 1) of the liver across all groups revealed dose-dependent changes. The control group (Plate 1) displayed normal hepatic architecture, with a central vein surrounded by radiating cords of hepatocytes and an intact portal triad containing bile ducts. In the low-dose group (Plate 2), disruption of normal architecture was evident, with multiple central veins and hepatocyte necrosis observed. The high-dose group (Plate 3) demonstrated more severe pathological changes, including a prominently engorged central vein containing blood and extensive hepatocyte necrosis. All sections were stained with Haematoxylin and Eosin at X100 magnification.

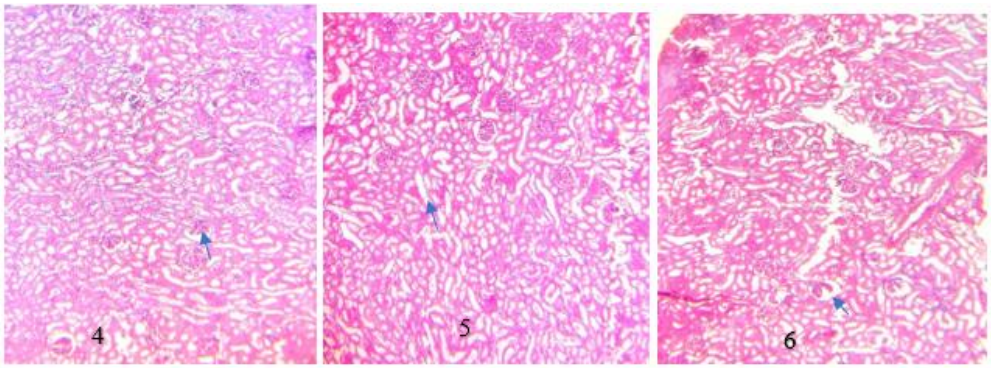

Figure 2. Plate 4, 5, and 6

Histological examination (Figure 2) of the kidney similarly revealed progressive, dose-dependent alterations. The control group (Plate 4) exhibited normal renal architecture, with intact glomerular tufts enclosed within Bowman's capsule, well-defined medullary tubules, and only mild inflammatory changes. The low-dose group (Plate 5) showed notable histopathological changes, including preserved but affected glomerular tufts, diffuse polymorphonuclear inflammatory cell infiltration, distortion of normal tissue architecture, and vascular congestion. The high-dose group (Plate 6) displayed the most severe alterations, characterized by glomerular tufts with markedly widened Bowman's capsule space and renal tubule necrosis accompanied by inflammatory infiltration. All sections were stained with Haematoxylin and Eosin at X100 magnification.

3.2. Discussion

The study was conducted to assess the histopathologic and biochemical changes in the liver and kidney of albino rats treated with a branded energy drink, Fearless, over a six-week period. No abnormal behaviors were observed in any group during the experiment. In terms of alertness, low-dose and high-dose treatment groups were notably more active compared to the control group, which received only standard diet and water and was observed to be calmer

and less active. This behavior is typical for rats not exposed to stimulants and reflects a baseline level of activity without external influence. The heightened activity in the treatment groups could be attributed to the stimulant effects of the energy drink, particularly ingredients such as caffeine, taurine, and other vitamins. This is in agreement with the findings of Higgins et al. (2018), which states that energy drinks, which commonly contain caffeine, can increase alertness and physical activity.

Feeding behavior remained normal across all groups, indicating that the energy drink did not significantly affect the appetite or food intake of the rats. This supports the claims of Valle et al. (2018) suggesting that while energy drinks influenced activity levels, they may not necessarily impair the animals' ability to feed or digest their diet. Water consumption increased in both treatment groups compared to the control group. The elevated water intake might be a compensatory response to the diuretic effects of caffeine, which can increase urine production and cause dehydration (Armstrong, 2002).

The mean body weight of the weaned albino rats across all groups exhibited a steady increase throughout the six-week study period, with no significant variation recorded between the control and treatment groups. The absence of significant differences in weight gain implies that the energy drink did not disrupt the normal metabolic processes that contribute to growth. This is consistent with findings where moderate consumption of energy drinks did not lead to significant changes in body weight in animal models (Buxton & Hagan, 2012), and may reflect the rats' ability to adapt to the components of the drink without experiencing adverse effects on normal growth (Higgins et al., 2018). It is important to note, however, that these results are specific to the six-week duration and controlled conditions of this study, and longer-term exposure may yield different outcomes.

As shown in Tables 3 and 4, the mean concentrations of aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP) were significantly elevated in both the low-dose and high-dose treatment groups compared to the control ($P<0.05$). This suggests that at both dose levels, the energy drink significantly altered these key indicators of liver function. The AST and ALT elevations at low dose are in agreement with the work of Boone et al. (2005), which identifies these transaminases as sensitive indicators of hepatocellular injury released due to cytoplasmic membrane leakage or necrosis. The ALP elevation, rising from a mean of 58.75 ± 3.92 IU/L in the control to 77.57 ± 8.57 IU/L at low dose and 102.00 ± 28.00 IU/L at high dose, may reflect mild cholestatic influence or enzyme induction, common in young rats where bone-derived ALP contributes substantially (Giknis & Clifford, 2006). In toxicity contexts, ALP rises can accompany hepatocellular changes in mixed injury patterns (Hall, 2007). The significant decrease in total protein, from 62.65 ± 2.96 g/L in the control to 52.43 ± 1.70 g/L at low dose and 51.34 ± 7.61 g/L at high dose, correlates with compromised hepatic synthetic capacity, a functional consequence of liver impairment (Thapa & Walia, 2007).

Marked increases in AST and ALT were observed at high dose, with AST rising to 36.40 ± 1.67 IU/L and ALT to 25.80 ± 1.30 IU/L compared to control values of 19.50 ± 1.91 IU/L and 14.00 ± 1.63 IU/L respectively, in agreement with Ozer et al. (2008), who identified elevated AST and ALT as classic markers of hepatocellular damage reflecting leakage from injured hepatocytes. In rat toxicity studies, such increases in transaminases strongly correlate with histopathologic evidence of necrosis or inflammation (Boone et al., 2005). The significant rise in direct bilirubin at high dose (0.52 ± 0.09 mg/dL vs. 0.36 ± 0.05 mg/dL in the control, $P=0.018$) suggests a cholestatic component, potentially involving bile duct injury or impaired biliary excretion (Hall, 2007). Giknis and Clifford (2006) observed similar findings, suggesting that cholestasis in rodents can accompany hepatocellular injury in mixed-pattern

hepatotoxicity. The decrease in total protein and trending albumin reduction indicate compromised hepatic synthetic capacity, common in moderate-to-severe liver injury where protein production is downregulated (Thapa & Walia, 2007). Total bilirubin levels showed no statistically significant difference at either dose level (P=0.411 at low dose; P=0.076 at high dose).

As shown in Tables 5 and 6, mean concentrations of urea, sodium, potassium, and creatinine from the control group to the low-dose treatment group showed a measure of stability, and the P-values were not significant. This suggests that at lower doses, the energy drink does not significantly alter these key indicators of kidney function. The mean chloride level was slightly higher in the control group (78.01 ± 7.18 mEq/L) compared to the low-dose group (73.83 ± 5.53 mEq/L), though this difference also lacked statistical significance (P=0.392). The stability of most biochemical markers at low doses might indicate that the kidney's compensatory mechanisms are still effective under mild stress, in line with the findings of Rahmadi et al. (2024), who concluded that low doses do not cause substantial changes in renal biomarkers.

In contrast, the high-dose group exhibited increased mean concentrations of urea (39.94 ± 2.74 mg/dL), sodium (140.52 ± 3.23 mEq/L), and chloride (85.85 ± 7.45 mEq/L) compared to the control, implying that higher doses of the energy drink may overwhelm the kidney's ability to maintain homeostasis, though these differences did not reach statistical significance (P>0.05). Potassium and creatinine levels in the high-dose group remained comparable to the control (3.64 ± 0.41 mEq/L vs. 3.77 ± 0.80 mEq/L, and 0.47 ± 0.05 mg/dL vs. 0.50 ± 0.10 mg/dL respectively), suggesting a selective impact of the energy drink on specific biochemical pathways. Notably, creatinine was higher in the low-dose group (0.61 ± 0.05 mg/dL) than in the high-dose group (0.47 ± 0.05 mg/dL), which may reflect a non-linear dose-response relationship where low doses induce specific stress responses that are altered at higher doses. These biochemical findings are broadly consistent with studies reporting increases in renal biomarkers in response to energy drink consumption (Aninweze et al., 2025; Miller, 2008). The non-significant changes in potassium levels differ from findings in studies where energy drinks caused significant electrolyte imbalances (Murt, 2025), a discrepancy that may be attributed to differences in energy drink formulation, species used, or study duration.

The histological findings, as illustrated in Plates 1-6, revealed progressive, dose-dependent structural changes in both the liver and kidneys. The control liver (Plate 1) displayed normal hepatic architecture, with a central vein surrounded by radiating cords of hepatocytes and an intact portal triad containing bile ducts, consistent with normal liver histology in untreated albino rats (Petterino & Argentino-Storino, 2006; Thoolen et al., 2010). The low-dose liver (Plate 2) showed disruption of normal architecture, with multiple central veins and hepatocyte necrosis, indicating early treatment-related hepatocellular injury. These changes correlate with the significant elevations in AST, ALT, and ALP, and the reduction in total protein observed in the same group, and are considered adverse at this dose level (Boone et al., 2005; Thoolen et al., 2010). The high-dose liver (Plate 3) demonstrated more severe pathological changes, including a prominently engorged central vein containing blood and extensive hepatocyte necrosis. These findings corroborate the highly significant elevations in AST, ALT, ALP, and direct bilirubin, along with the pronounced decrease in total protein recorded in the high-dose group, representing clear evidence of dose-dependent hepatotoxicity (Greaves, 2011; Thoolen et al., 2010).

Regarding the kidney histology, the control group (Plate 4) exhibited intact glomerular tufts enclosed within Bowman's capsule and well-defined medullary tubules, with only mild baseline inflammatory changes noted. This mild baseline inflammation, observed in the

absence of any treatment, may reflect an underlying condition among the weaned albino rats and represents a confounding factor that should be considered when interpreting treatment group comparisons, though it does not negate the observed treatment effects (Murt, 2025). The low-dose kidney (Plate 5) showed notable histopathological changes, including preserved but affected glomerular tufts, diffuse polymorphonuclear inflammatory cell infiltration, distortion of normal tissue architecture, and vascular congestion, suggesting that even low doses of the energy drink can induce notable kidney damage (Kutia et al., 2019). The high-dose kidney (Plate 6) displayed the most severe alterations, characterized by glomerular tufts with markedly widened Bowman's capsule spaces and renal tubule necrosis accompanied by inflammatory infiltration, indicating that higher doses cause more profound renal damage (Qassim et al., 2022).

Overall, the biochemical and histopathological findings of this study demonstrate that consumption of the "Fearless" energy drink induces dose-dependent hepatotoxicity and nephrotoxicity in weaned albino rats, with higher doses producing more severe and widespread tissue injury. These results are consistent with documented evidence of organ damage associated with energy drink consumption (Bano et al., 2020; Greene et al., 2014) and underscore the importance of dose regulation in assessing the safety of such beverages.

4. Conclusion

This study highlights the significant histopathologic and biochemical changes in the liver and kidney of albino rats treated with the branded energy drink "Fearless." The results indicate that both low and high doses of the energy drink can cause notable liver and kidney damage, characterized by structural alterations and inflammation. Inflammation noted in the control group complicates interpretation and warrants further investigation.

Based on these findings, long-term consumption of energy drinks should be discouraged due to the adverse effects observed in animal studies. Therefore, it is crucial to exercise caution in the consumption of energy drinks. Further research is needed to understand the mechanisms of liver and kidney damage and to explore protective strategies to mitigate the adverse impacts of energy drinks on liver and kidney health.

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