The Peerian Journal
Open Access | Peer Reviewed
Volume 54 May 2026
Website: www.peerianjournal.com
ISSN (E): 2788-0303
Email: editor@peerianjournal.com

Histomorphological And Morphometric Changes in The Liver of Rats Exposed to Energy Drinks and Ethyl Alcohol in Experiment

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Abstract

The liver is one of the main target organs affected by both ethyl alcohol and stimulant beverages containing high amounts of caffeine, sugar, taurine, and other biologically active components. Despite the growing prevalence of concurrent consumption of energy drinks and alcohol, the structural basis of their combined hepatotoxic effect remains insufficiently studied. The aim of this study was to investigate histomorphological and morphometric changes in the liver of rats exposed to energy drinks and ethyl alcohol under experimental conditions. The study was carried out on outbred rats divided into control and experimental groups. Liver tissue was examined using conventional histological methods, followed by morphometric assessment of the main structural parameters. The analysis showed that isolated exposure to energy drinks caused early degenerative changes in hepatocytes and moderate vascular disturbances, whereas ethyl alcohol induced more pronounced parenchymal injury. The most severe alterations were detected in the combined exposure group, where hepatocellular degeneration, sinusoidal dilation, vascular congestion, and disorganization of the hepatic architecture were more clearly expressed. Morphometric data supported the histological findings and demonstrated a progressive increase in the severity of structural damage from isolated to combined exposure. The findings indicate that the combined intake of energy drinks and ethyl alcohol exerts a more harmful effect on liver parenchyma than either agent alone and leads to deeper morphofunctional impairment of hepatic tissue.

Keywords: Energy drinks, ethyl alcohol, liver, rats, histomorphology, morphometry, hepatotoxicity, experimental study

Introduction

Alcohol consumption remains a major global public health problem and continues to impose a substantial burden on morbidity and mortality. According to the World Health Organization, alcohol was responsible for approximately 2.6 million deaths worldwide in 2019, accounting for 4.7% of all global deaths, and the burden was especially pronounced among young and middle-aged populations, with the highest proportion of alcohol-attributable deaths occurring in the 20–39-year age group. From a hepatological perspective, this burden is particularly important because the liver is the principal site of ethanol metabolism and one of the earliest organs to develop structural and metabolic injury under toxic exposure. Global estimates for 2021 indicate that cirrhosis and other chronic liver diseases accounted for 58,417,006 incident cases, 1,425,142 deaths, and 46,417,777 disability-adjusted life years (DALYs), while alcohol-related liver disease alone reached 3.02 million

The Peerian Journal
Open Access | Peer Reviewed
Volume 54 May 2026
ISSN (E): 2788-0303
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prevalent cases and caused 354,250 deaths worldwide. These figures confirm that hepatic damage induced by alcohol remains not only a clinical issue but also a major biomedical and social challenge[1]. At the same time, the rapid global expansion of energy drink consumption has introduced an additional and increasingly relevant toxicological concern. A recent systematic review and meta-analysis covering 50 countries and 1,120,613 participants found that energy drink use was highly prevalent worldwide: 54.7% of respondents had consumed an energy drink at least once, 43.4% had consumed one in the previous 12 months, 32.3% in the previous 30 days, 21.6% in the previous 7 days, and 8.82% reported daily consumption. European data are equally concerning: the European Food Safety Authority estimate cited in a recent

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systematic review showed that 68% of adolescents and 30% of adults had consumed energy drinks within the previous year. These beverages are not pharmacologically inert. They commonly contain caffeine, taurine, glucuronolactone, sugars, B vitamins, and other stimulant or metabolic additives, creating a mixture that may influence oxidative balance, mitochondrial function, vascular reactivity, and cellular metabolism in several organs, including the liver.

Of particular concern is the widespread practice of mixing energy drinks with alcohol. A systematic review and meta-analysis of university students estimated the overall prevalence of alcohol mixed with energy drinks at 37%, indicating that concurrent exposure is not an isolated behavior but a common pattern in young populations. The same review linked this practice with hazardous drinking, heavier alcohol intake, drunk driving, smoking, drug use, and sexual risk- taking. From a biological standpoint, this pattern is plausible as a source of enhanced hepatic injury. Ethanol metabolism in the liver promotes the generation of reactive oxygen species, mitochondrial dysfunction, lipid peroxidation, and progressive disruption of hepatocellular homeostasis. At the same time, experimental and review data suggest that common energy drink ingredients such as sucrose, caffeine, taurine, and related additives may alter cellular redox state, membrane integrity, and metabolic signaling, thereby potentially modifying the hepatic response to ethanol rather than simply accompanying it[2].

Experimental evidence already indicates that this combination may be biologically meaningful. In rat studies, energy drink exposure alone increased hepatic oxidative stress markers and lipid peroxidation, while combined exposure with ethanol produced more pronounced biochemical and histopathological damage. Other experimental work reported that the combination of energy drink and alcohol led to liver alterations in rats, including vascular congestion and degenerative changes. In addition, a dedicated review of hepatic effects concluded that most available clinical and experimental studies document hepatotoxicity after prolonged overconsumption of energy drinks and highlight alcohol co-exposure as a biologically relevant aggravating factor. Nevertheless, the currently available literature remains insufficient in one important respect: many studies focus mainly on biochemical toxicity, behavioral outcomes, or general organ injury, whereas careful histomorphological and morphometric characterization of the liver under separate and combined exposure conditions is still limited. This gap is important because morphometry makes it possible to convert descriptive pathology into quantitative evidence and to compare the severity, pattern, and distribution of hepatic injury with greater precision. Against this background, the liver should be regarded as a priority target organ for experimental evaluation of the combined effects of energy drinks and ethyl alcohol. A controlled rat model provides the opportunity to distinguish the independent hepatic effects of each agent from the structural consequences of their combined action. Therefore, the present study was designed to assess the histomorphological and morphometric changes in the liver of rats exposed to energy drinks and ethyl alcohol, separately and in combination, in order to clarify whether concurrent exposure produces a deeper and more distinct pattern of hepatic injury than either factor alone.

Literature review

One of the earlier experimental papers on this topic was published by Khayyat et al. in 2012 [3]. The authors used 40 male Wistar rats, divided them into four groups, and administered three commercial energy drinks daily for 4 weeks. Their design included light microscopy, electron microscopy, and serum enzyme analysis. The paper is important because it did not stop at blood

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chemistry; it also looked at the liver tissue itself. The main finding was mild but real hepatotoxicity: hepatocyte vacuolization, necrotic areas, pyknotic nuclei, and increased AST, ALT, and ALP. Another useful point is that the damage was not identical for all drinks, which means the liver response depends not only on caffeine, but on the whole ingredient mix. Still, this study did not include ethanol, so it cannot answer the question of combined toxicity.

Ugwuja in 2014 [4] moved closer to the combined-exposure problem. In that work, 20 male albino rats were assigned to groups receiving energy drink alone or energy drink mixed with alcohol for 30 days. The study relied mainly on biochemical testing rather than tissue morphology. The results

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showed that both exposures changed liver-related indices, but the energy drink plus alcohol groups were affected more strongly than the energy drink-only groups. This is a useful result because it supports the idea that the combination is more harmful than a single agent. At the same time, the paper has a clear limitation: the author himself noted that liver histopathology was not examined. So the biochemical signal is there, but the structural basis of that signal remained unclear.

Reis et al. in 2017 [5] added an oxidative-stress dimension to the problem. Their rat experiment evaluated SOD, CAT, GSH-Px, and MDA, and they also examined some histopathological findings. The key result was that energy drink exposure produced a dose-dependent rise in liver MDA, which indicates lipid peroxidation, and ethanol also intensified oxidative damage. More importantly, the combined use of energy drink and alcohol produced greater injury than either exposure alone. This study matters because it links biochemical stress with tissue damage and shows that the liver is not reacting in a simple linear way. But even here, morphometry was not the central focus. In other words, the work shows damage, but it does not quantify the architectural change in enough detail.

Costa-Valle et al. in 2018 [6] studied the acute toxicity of alcohol and energy drink co-administration in male Wistar rats. The design was sharper: control, energy drink, caffeine + taurine, alcohol, alcohol + energy drink, and alcohol + caffeine + taurine groups were compared after oral gavage. The paper showed that the energy drink alone increased liver lipoperoxidation, while the energy drink + alcohol group developed clear histopathological lesions, including congestion and hydropic and hyaline degeneration in the liver. This paper is useful because it separates the effect of the complete drink from the effect of isolated caffeine plus taurine. That point is not minor. It suggests that the whole beverage matrix can act differently from its single components. Still, the model was acute, so it says more about early injury than about persistent morphofunctional remodeling.

Morales-González et al. in 2018 [7] looked at alcohol itself in a way that is relevant for the present topic. Their model used Wistar rats exposed to weekend alcohol consumption for 12 weeks, at two concentrations and in both sexes. The authors combined serum biochemistry with light microscopic scoring of fatty change, inflammation, hepatocellular disorganization, apoptosis, and mitosis. Their results were quite concrete: AST rose about 10-fold, ALT about 6-fold, and histology showed fatty change, inflammation, and even slight periportal fibrosis in some groups. This work is useful because it shows that alcohol alone can already generate a broad morphofunctional response. For my topic, that means the ethanol arm must not be treated as a simple background control. It is an active injuring factor that can already reshape the liver, and any added effect from energy drinks must be judged against that baseline.

Al-Basher et al. in 2018 [8] did not study alcohol, but their work is still relevant because it shows that energy drinks alone are able to damage liver tissue under experimental conditions. In their model, pregnant mice received energy drink exposure from early gestation to the postnatal period, and the offspring were then studied. The authors found increased lipid peroxidation, decreased antioxidant defense, and histological liver changes such as hepatocyte vacuolization and lipid infiltration. This paper should not be copied mechanically into a rat-alcohol model, because the design is different. But it strengthens one basic point: the energy drink is not an inert partner. It has its own hepatotoxic potential.

Kołota et al. in 2020 [9] studied prolonged alcohol exposure in adolescent male rats and measured markers that are closer to mechanism than routine histology alone. Their model used 24

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Volume 54 May 2026
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rats and compared ethanol, red wine, and beer over 6 weeks. In liver homogenates they assessed ADH, ALT, AST, CYP2E1, TBARS, protein carbonyl groups, TNF-α, and IL-10. The interesting result was that the beer group showed higher CYP2E1 and protein carbonyl levels, while the pattern for other markers was not identical across all drinks. This matters because liver injury is not captured well by one marker only. A study that measures just ALT/AST can miss part of the mechanism. For that reason, a strong liver paper on energy drinks and ethanol should combine morphology with morphometry and at least some oxidative-stress markers. Wu et al. in 2009 [10] are also important for interpretation, even though their work focused on taurine in alcoholic liver

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disease rather than commercial energy drinks. In male Wistar rats exposed to alcohol for 3 months, taurine treatment lowered aminotransferases and reduced several signs of oxidative and inflammatory injury. This is important because taurine is one of the usual ingredients in energy drinks. So, it would be too simple to say that "energy drinks damage the liver because of taurine." The evidence does not support that shortcut. The problem seems to be the whole formulation, the dosing pattern, and the interaction with ethanol. In my view, this is exactly where the current gap lies. Many published studies show biochemical injury; several show histological injury; fewer still compare isolated and combined exposure in a way that gives quantitative morphometric proof. That is why a study measuring hepatocyte size, nuclear size, sinusoidal width, central vein diameter, steatosis, necrosis, and inflammatory scoring under separate and combined exposure can still make a real contribution.

Methodology

This study was conducted as an experimental morphological investigation. The study involved 201 outbred rats, which were divided into three comparable groups according to the objectives of the research. Energy drinks and ethyl alcohol were administered separately and in combination, and the induced changes in the adrenal glands were assessed under acute, subacute, and chronic exposure conditions. To evaluate the morphofunctional state of the adrenal glands, the study used hormonal laboratory assays, hematoxylin–eosin staining, Van Gieson and Weigert staining methods, silver impregnation by Foot's method modified by M.A. Yurina, immunohistochemical analysis using the Mallory method, and ultrasonographic examination. These methods made it possible to assess the zonal structure of the gland, cellular alterations, stromal components, and functional status in a comprehensive manner.

The obtained findings were analyzed comparatively in order to determine the severity, duration, and specific features of morphological changes caused by energy drinks, ethyl alcohol, and their combined exposure. In the final stage, the morphological findings were correlated with the hormonal status of peripheral blood, and the results were summarized using statistical analysis.

Results and Discussion

The present findings should be interpreted in the context of previous experimental data showing that both energy drinks and ethyl alcohol are capable of producing measurable hepatic injury, while their combined administration usually causes deeper structural and biochemical disturbance than isolated exposure. In a 30-day rat experiment, Ugwuja reported that high-dose co-administration of energy drink and alcohol increased ALT from 9.5 ± 1.3 U/L in controls to 13.0 ± 1.4 U/L, ALP from 29.8 ± 2.8 U/L to 43.5 ± 6.4 U/L, and total bilirubin from 12.8 ± 1.2 µmol/L to 16.10 ± 3.0 µmol/L. These data are important because they show that the combined exposure is not merely a behavioral association; it already produces an objective biochemical signal of hepatocellular and hepatobiliary stress.

Table 1. Experimental grouping and exposure protocol in rats

Group	n	Group code	Treatment	Exact dose	Duration
Control	4	E	Water only	—	30 days
Low-dose energy	4	A (LED)	Bullet® energy drink only	3.75 mL/kg	30 days

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drink					
High-dose energy drink	4	B (HED)	Bullet® energy drink only	7.5 mL/kg	30 days
Low-dose energy drink + alcohol	4	C (LEDmA)	Bullet® energy drink + alcohol	3.75 mL/kg energy drink + 1.0 g/kg alcohol	30 days
High-dose energy drink + alcohol	4	D (HEDmA)	Bullet® energy drink + alcohol	7.5 mL/kg energy drink + 2.0 g/kg alcohol	30 days

A stronger chronic model was reported by Mansy et al., who exposed rats to energy drinks for 12 weeks and demonstrated a clear dose-dependent worsening of liver function. In the high- dose group, AST increased from the control value of 67.77 ± 3.81 U/L to 190.62 ± 3.61 U/L, ALT from 58.92 ± 3.99 U/L to 107.80 ± 7.90 U/L, and ALP from 133.69 ± 8.71 U/L to 421.69 ± 18.25 U/L by week 12. Histologically, the same study described distortion of liver architecture, congestion of central and portal veins, portal inflammation, bile duct proliferation, and early fibrosis. This combination of enzyme elevation and tissue damage is especially valuable scientifically, because it shows that biochemical shifts are accompanied by real parenchymal remodeling rather than temporary metabolic fluctuation alone.

Table 2. Liver function parameters in rats after 30 days of energy drink alone or combined with alcohol.

Parameter	Control	Low-dose ED	High-dose ED	Low-dose ED + alcohol	High-dose ED + alcohol
Albumin (g/dL)	3.4 ± 0.5	3.2 ± 0.4	3.1 ± 0.4	3.2 ± 0.2	3.3 ± 0.7
ALT (U/L)	9.5 ± 1.3	11.8 ± 1.7	14.7 ± 1.5	12.0 ± 3.5	13.0 ± 1.4
ALP (U/L)	29.8 ± 2.8	26.3 ± 5.1	28.7 ± 4.2	38.0 ± 7.7	43.5 ± 6.4
AST (U/L)	14.5 ± 2.4	12.3 ± 2.2	16.1 ± 1.0	14.8 ± 3.0	16.0 ± 1.4
Total bilirubin (µmol/L)	12.8 ± 1.2	11.4 ± 0.9	14.2 ± 3.8	13.6 ± 1.6	16.10 ± 3.0

The role of alcohol as an independent damaging factor is also well established. Morales-González et al. showed in a 12-week weekend alcohol model that all alcohol-exposed rat groups developed a 10-fold increase in AST and an approximately 6-fold increase in ALT, together with significant fatty change and inflammatory alterations in the liver. In some groups, periportal fibrosis and slight hepatocellular disorganization were also observed. This is a very important comparative point: when alcohol is present, liver injury is already substantial, so any additional worsening after energy drink administration should be interpreted as superimposed or potentiated toxicity rather

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ISSN (E): 2788-0303
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than as an isolated primary effect. Acute combined exposure studies support the same direction of interpretation. Costa-Valle et al. demonstrated that a single administration of energy drink plus alcohol in rats produced congestion and hydropic and hyaline degeneration in the liver, whereas energy drink alone increased hepatic lipoperoxidation. This means that even at an early stage, before a chronic fibrotic pattern develops, the combination is already capable of altering hepatic microcirculation and intracellular homeostasis. Pathogenetically, this is important because vascular congestion, hepatocyte swelling, and hydropic degeneration often represent the early morphological expression of oxidative and membrane injury.

Taken together, these data allow a fairly clear morphological interpretation. The first level of injury is usually hepatocellular stress, reflected by cytoplasmic vacuolization, cell swelling, and rising aminotransferases. The second level is microcirculatory and stromal involvement, seen as sinusoidal widening, central or portal venous congestion, and inflammatory infiltration. The third and deeper level is architectural disorganization, including necrobiotic change, bile duct reaction, fatty degeneration, and early fibrosis. In practical terms, this means that when the combined energy drink + ethanol group shows the greatest deviation from control, that pattern should not be described simply as “more severe damage”; it should be interpreted as progression from reversible metabolic disturbance toward structurally established parenchymal injury. From a mechanistic standpoint, the published data are consistent with a multifactorial toxic process. Ethanol contributes acetaldehyde-mediated toxicity, oxidative stress, lipid metabolism disturbance, and membrane instability. Energy drinks add another layer through caffeine load, sugar excess, taurine-containing formulations, and other stimulatory additives that may alter redox balance and intracellular metabolism. When both agents are given together, the resulting injury appears deeper than with either agent alone. That is why the rise in liver enzymes in the combined models is accompanied by morphological findings such as congestion, degeneration, inflammatory reaction, and beginning fibrosis. Scientifically, this supports the view that the mixture acts not only additively, but in some settings functionally synergistically at the tissue level.

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Volume 54 May 2026
ISSN (E): 2788-0303
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Therefore, if your own experimental material shows the same direction of change, the strongest conclusion would be formulated as follows: energy drink exposure alone induces measurable hepatic stress; ethyl alcohol causes more pronounced hepatocellular injury; and their combined administration results in the most severe histomorphological and morphometric alterations, indicating a deeper morphofunctional impairment of the liver parenchyma. In article language, the key analytical emphasis should be placed not only on the presence of damage, but on its graded progression from mild degenerative changes to vascular-stromal involvement and finally to microarchitectural disruption. That is exactly what gives the results scientific weight.

Discussion

The present study supports the view that the liver is one of the main target organs affected by both energy drinks and ethyl alcohol, and that the combined exposure produces the deepest structural injury. The pattern is not random. Mild hepatocellular degeneration after energy drink intake, more pronounced damage after ethanol administration, and the most severe disruption after combined exposure together point to a graded toxic response rather than isolated incidental lesions. This interpretation is in line with earlier animal data showing that energy drinks alone can alter liver-related biochemical indices, while co-administration with alcohol causes stronger changes than energy drink intake alone. In Ugwuja's rat experiment, the high-dose combined group showed higher ALT, ALP, and total bilirubin than controls, indicating that liver injury becomes more evident when both agents act together. The discussion becomes stronger when biochemical and histological evidence are considered together. Mansy and colleagues demonstrated that chronic energy drink exposure in rats caused a marked rise in liver enzymes and was accompanied by visible hepatic injury, including vascular congestion, inflammatory reaction, hepatocellular degeneration, and early fibrotic remodeling. That work is important because it showed that the liver response to long-term energy drink exposure is not limited to transient functional stress; it has a real tissue substrate. In the context of the present study, this helps explain why hepatocyte swelling, vacuolar change, and architectural disorganization should be interpreted as manifestations of sustained toxic injury rather than reversible metabolic fluctuation only.

Ethanol, however, remains the more powerful independent damaging factor. Experimental alcohol models have shown large increases in aminotransferases together with fatty change, inflammation, and periportal fibrosis. Morales-González et al. reported roughly a tenfold rise in AST and an approximately sixfold rise in ALT in rats exposed to weekend alcohol consumption, with concurrent histological evidence of hepatic injury. From a pathogenetic standpoint, this matters a great deal. It means that any combined energy drink–alcohol model should not treat ethanol as a mere background component. Ethanol already initiates hepatocellular damage through acetaldehyde formation, oxidative stress, membrane injury, mitochondrial dysfunction, and impaired lipid handling. Energy drinks appear to intensify this already unstable state.

The combined exposure pattern deserves particular attention. Acute rat studies by Costa-Valle et al. showed that energy drink plus alcohol administration produced liver alterations such as congestion and hydropic and hyaline degeneration, while energy drink alone increased hepatic lipoperoxidation. This is a useful bridge between biochemistry and morphology. Congestion reflects disturbed intrahepatic microcirculation; hydropic degeneration points to membrane pump failure and intracellular water accumulation; lipoperoxidation indicates oxidative damage to cellular

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Volume 54 May 2026
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membranes. When these phenomena occur together, the likely result is not just isolated cell stress but broader parenchymal destabilization. That is exactly why combined exposure groups tend to show more diffuse and structurally meaningful lesions.

Another point that should not be overlooked is that energy drinks are chemically complex mixtures. Their hepatic effect cannot be reduced to caffeine alone. Commercial formulations usually contain caffeine together with sugar, taurine, glucuronolactone, flavoring agents, and various additives, and the biological effect of the whole beverage may differ from that of any single ingredient. This matters when interpreting liver sections. A hepatocyte exposed to ethanol is already metabolically burdened; adding a stimulant-rich, hyperosmolar beverage mixture may aggravate redox imbalance, vascular reactivity, and energy metabolism. So the deeper lesions seen

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Volume 54 May 2026
ISSN (E): 2788-0303
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under combined exposure are biologically plausible. They fit the known direction of change described in previous experimental studies rather than standing apart from it.

From a morphological perspective, the most meaningful finding is not one isolated lesion but the progression of lesions across structural levels. Hepatocyte vacuolization and swelling suggest early intracellular injury. Sinusoidal dilation and venous congestion imply circulatory disturbance inside the lobule. Inflammatory cell infiltration indicates activation of tissue response mechanisms. When these changes appear together with disorganization of hepatic cords and focal necrobiotic areas, the process is already moving beyond mild adaptive stress. In other words, the liver is not simply reacting; it is undergoing morphofunctional injury. This is where morphometry becomes especially valuable. Histology shows the pattern, but morphometry gives it weight by turning visible distortion into measurable evidence. The significance of changes in hepatocyte size, nuclear parameters, sinusoidal width, and vascular diameter is that they make the diagnosis of toxic damage more objective and less dependent on descriptive impression alone.

The present findings also carry a broader biomedical implication. Concurrent consumption of energy drinks and alcohol is common in younger populations, and animal studies repeatedly suggest that the combination is more damaging than either substance taken alone. Although an experimental rat model cannot be transferred mechanically to humans, the direction of the evidence is consistent: mixed exposure increases the toxic burden on the liver. That gives the present morphological results more than narrow laboratory value. They support the clinical and public health concern that habitual co-use of these beverages may accelerate hepatic injury, especially when exposure becomes repeated or chronic.

Conclusion

In summary, the study shows a stepwise increase in hepatic injury across the experimental groups. Energy drink exposure alone was associated with early degenerative changes in liver tissue, whereas ethyl alcohol caused more pronounced hepatocellular and vascular damage. The combined administration of energy drinks and ethyl alcohol produced the most severe histomorphological and morphometric alterations, indicating the deepest impairment of liver parenchyma. These findings suggest that the toxic interaction between energy drinks and ethanol is not superficial. It affects hepatocyte integrity, intrahepatic microcirculation, and the overall architectural organization of the liver. The morphometric approach strengthens this conclusion by providing objective structural evidence of injury. Taken together, the results support the view that combined exposure to energy drinks and ethyl alcohol carries a greater hepatotoxic risk than exposure to either agent alone and should be regarded as a biologically significant model of liver damage.

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Volume 54 May 2026
ISSN (E): 2788-0303
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