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Research Article

Impact of Commercial Energy Drinks (Red Bull and Monster) on Cardiac Enzymes, Electrolyte Ions, and Oxidative Stress Markers in Immature Male Rats

Manal Hameed Raheem*, Hanaa Enayaa Mahood

Department of Biology, College of Education, University of Al-Qadisiyah, Iraq

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MHR conducted the experimental work, data analysis and manuscript drafting. HEM supervised the study and revised the manuscript.

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Cardiotoxicity, Electrolyte imbalance, Energy drinks, GSH, MDA, Troponin

Abstract | This study aimed to investigate the physiological and biochemical impacts of two commercially available energy drinks Red Bull and Monster on immature male rats. The analysis focused on cardiac enzyme activity, electrolyte ion levels, and oxidative stress markers. Forty male albino rats were randomly divided into five groups (n = 8 per group): one control group and four treatment groups receiving either a standard or double oral dose of Red Bull or Monster (3.57 mL/kg and 7.14 mL/kg body weight) for 45 consecutive days. Blood samples were collected to measure serum levels of troponin, lactate dehydrogenase (LDH), creatine kinase (CK), sodium (Na^+), potassium (K^+), malondialdehyde (MDA), and glutathione (GSH). The results showed statistically significant increase in ($P < 0.05$) in troponin, LDH, CK, Na^+, K^+, and MDA levels across all treatment groups, particularly in the high-dose Monster group (T4), which recorded the highest values for troponin (130.70 ± 1.78 pg/mL), LDH (506.41 ± 2.52 U/L), CK (243.83 ± 1.05 U/L), Na^+ (188.11 ± 0.89 mmol/L), K^+ (6.96 ± 0.05 mmol/L), and MDA (8.71 ± 0.035 µmol/L). Conversely, GSH levels were significantly reduced, with the lowest concentration observed in the T4 group (10.20 ± 0.247 µmol/L) compared to the control (24.39 ± 0.251 µmol/L). In conclusion, the chronic intake of Red Bull and Monster especially at high doses induces dose-dependent cardiotoxicity, disrupts electrolyte homeostasis, and exacerbates oxidative stress in developing male rats. These findings raise concerns regarding the unregulated consumption of energy drinks among adolescents.

Novelty Statement | This study compares the effects of Red Bull and Monster energy drinks on cardiovascular biochemical markers in immature male rats in Iraq. The results reveal distinct toxicological profiles and potential cardiovascular risks during early development.

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Introduction

Energy drinks (EDs) use has become more and more popular among adolescents and young adults, because of the claimed effects to ameliorate physical stamina, mental performance, and cognitive reaction. They are usually high in caffeine and they also contain other active substances, such as taurine, sugar, B vitamins, ginseng and other herbal extracts (Vargiu *et al.*, 2021; Bunch *et al.*, 2023). Despite very aggressive marketing of these products, which emphasize their energy enhancing effects, recent data have facilitated significant reservations regarding the possible physiological and toxicological risks evidenced by abuse of these sort of 'medicines' especially among

Corresponding author: Manal Hameed Raheem
edu.bio.posta126@qu.edu.iq

young people. Cardiotoxic effects are one of the major health hazards associated with ED consumption. High consumption of energy drinks has been associated with negative cardiovascular consequences, such as: arrhythmias, changes in the blood pressure, QTc interval prolongation, myocardial infarction and in the most severe case with a sudden death (Costa *et al*., 2023; Costantino *et al*., 2023). The risks of which may be increased in adolescents because of their immature cardiovascular systems, making them more vulnerable to caffeine-induced toxicity (Mota-Rojas *et al*., 2023).

Energy drinks are also associated with alterations in cardiac electrophysiology, mainly mediated by caffeine and taurine. Caffeine is an adenosine receptor antagonist, which causes increased sympathetic nervous system activity and elevation of intracellular calcium level, which could result in myocardial challenge (Chami and Di Primio, 2024). These effects could be amplified at the cellular level via synergistic alterations of cardiac contractility and excitability when combined with taurine, possibly resulting in arrhythmogenic outcomes (indicative reference: Mai-Lippold *et al*., 2021). Troponin I (cTnI), lactate dehydrogenase (LDH), creatine kinase (CK-MB) also are the important markers reflecting myocardial injury. In previous experimental models, consumption of energy drink has been shown to increase the enzymes levels, implying cardiomyocytes disruption or necrosis (Motamed *et al*., 2023; Jagim et al., 2014). These biochemical alterations often include changes in electrolyte balance, in particular sodium (Na^+) and potassium (K^+) ions. These ions are critical for conducting a cardiac action potential and electrophysiologic stability, and disturbances may result in the development of arrhythmias (Mandilaras *et al*., 2022).

In addition to the direct cardiac effects of energy drinks, they have also been linked to the induction of oxidative stress which is one of the most important contributors to the pathophysiology of cardiovascular disease. It is evidenced that an increase in ROS generation and resultant lipid peroxidation are indicated by the increased level of malondialdehyde (MDA) and often by the depletion of antioxidant compounds such as glutathione (GSH) (Jomova *et al*., 2023; Patani *et al*., 2023). This redox dyshomeostasis is regarded as an early signature of endothelial dysfunction and of myocardial injury, aggravating the cardiovascular overload SC consumption may exert some impacts (Zhao *et al*., 2023; Trofin *et al*., 2025). Despite several researches focusing on the individual physiological effects of energy drinks, insufficient comprehensive research studies have been conducted in immature organisms concerning the effect of these stimulants on the cardiac enzymes, oxidative stress, as well as electrolyte balance. Thus, this study was carried out to assess the interaction of dietary Red Bull in combination with Monster on developing male rats by measuring serum levels of cTnI, LDH, CK-MB, Na^+, K^+, MDA, and GSH. Results of the study are intended to better inform potential health risks of energy drinks when consumed on a chronic (i.e., daily) basis during adolescence and provide evidence-based public health recommendations.

Materials and Methods

Experimental animals

The study involved 40 immature male albino rats aged 5–6 weeks and weighing 120–140 g. Animals were housed in plastic cages under controlled conditions (temperature: 20–25°C, 12-h light/dark cycle) and provided with standard pellet diet and water ad libitum. The experiment was conducted in the animal house of the College of Science, University of Al-Qadisiyah, between October 1 and December 27, 2024. Ethical handling and housing conditions were in accordance with institutional guidelines.

Energy drink administration and experimental design

Commercially available Red Bull (Rauch, Germany) and Monster (Monster Beverage Corp.) were administered orally via gavage at a dose of 3.57 mL/kg body weight, as determined from previous studies (Bano *et al*., 2020). Rats were randomly assigned into five groups (n=8 per group) for a duration of 45 days: Group C (Control): Received no treatment (standard feed and water only). Group T1: Received Red Bull at 3.57 mL/kg/day. Group T2: Received double the Red Bull dose (7.14 mL/kg/day). Group T3: Received Monster at 3.57 mL/kg/day. Group T4: Received double the Monster dose (7.14 mL/kg/day).

Sample collection

At the end of the experimental period, animals were anesthetized using chloroform. Blood was drawn via cardiac puncture using sterile 5 mL syringes. Samples were allowed to clot, centrifuged at 3000 rpm for 15 min, and serum was separated and stored at -20°C for biochemical assays. Hearts were harvested, rinsed in saline, and preserved in 10% formalin for histological analysis.

Biochemical assays

Troponin I was Quantified using competitive ELISA technique (Bakker and Mucke, 2007). The assay used anti-cTnI antibodies and HRP-conjugated antigen. Absorbance was read at 450 nm. Lactate Dehydrogenase (LDH) was measured via enzymatic reduction of pyruvate to lactate using NADH as a coenzyme, monitored at 340 nm (Young, 1997). Creatine Kinase (CKP) was determined by the UV kinetic method, using substrate-buffer mixtures and coenzymes, following Neumeier *et al*. (1976). Electrolytes (Na^+ and K^+) was assessed enzymatically using Mindray kits based on modified Kaplan and Tietz protocols (Kaplan and Pesce, 2007; Tietz, 2006). Na^+ was measured at 405

nm and K^+ at 578 nm using colorimetric methods.

Oxidative stress markers analysis

Glutathione (GSH) was estimated using Ellman's reagent DTNB as per Sedlak and Lindsay (1968). The absorbance of the yellow-colored product was measured at 412 nm. Malondialdehyde (MDA) was used as a marker for lipid peroxidation which was determined via thiobarbituric acid reaction forming a pink adduct, which was read at 532 nm (Guidet and Shah, 1989).

Statistical analysis

Data were analyzed using SPSS software (version 25). One-way ANOVA was used to determine significant differences between groups. Differences were considered statistically significant at $P \leq 0.05$. Post hoc analysis was performed using the Least Significant Difference (LSD) test (Moder, 2010).

Results

Cardiac biomarkers (Troponin, LDH, CKP)

The results presented in Table 1 indicate a statistically significant ($P < 0.05$) elevation in the levels of troponin, lactate dehydrogenase (LDH), and creatine kinase (CKP) in all treatment groups compared to the control. The highest values were recorded in group T4 (double dose of Monster), with troponin at 243.83±1.05 pg/mL, LDH at 506.41±2.52 U/L, and CKP at 130.70±1.78U/L. These were considerably elevated compared to the control group values of 120.90±0.65 pg/mL, 266.91±1.52 U/L, and 35.08±0.599 U/L, respectively. A clear dose-dependent relationship was observed, where higher doses resulted in more pronounced enzyme elevations.

Table 1: Effect of red bull and monster on cardiac biomarkers in immature male rats.

Group	Troponin (pg/mL)	LDH (U/L)	CKP (U/L)
Control	120.90±0.652^{E}	266.91±1.523^{E}	35.08±0.599^{E}
T1 (Red Bull)	182.63±1.152^{D}	344.20±1.859^{D}	60.76±0.863^{D}
T2 (Red Bull x^2)	225.59±1.452^{B}	450.39±1.985^{B}	102.36±1.844^{B}
T3 (Monster)	197.78±0.965^{C}	415.75±2.124^{C}	74.12±1.242^{C}
T4 (Monster x^2)	243.83±1.056^{A}	506.41±2.521^{A}	130.70±1.785^{A}
LSD	9.532	14.526	8.563

Different letters indicates statistical difference at $P < 0.05$

Electrolyte ion levels (Na^+, K^+)

Table 2 demonstrates a significant increase in sodium and potassium ion levels in all treatment groups compared to the control. Group T4 exhibited the highest elevations with 188.11 ± 0.89 mmol/L of K^+ and 6.96 ± 0.05 mmol/L of Na^+, versus control values of 140.77 ± 0.58 mmol/L and 4.28 ± 0.02 mmol/L, respectively. These findings suggest electrolyte imbalance, likely resulting from the combined effects of caffeine, sugar, and other stimulant compounds in the energy drinks.

Table 2: Effect of red bull and monster on electrolyte ion levels in immature male rats.

Group	Sodium (mmol/L)	Potassium (mmol/L)
Control	4.28±0.024 ^{E}	140.77±0.586 ^{E}
T1 (Red Bull)	5.17±0.015 ^{D}	150.15±0.748 ^{D}
T2 (Red Bull x2)	6.14±0.035 ^{B}	174.36±0.847 ^{B}
T3 (Monster)	5.72±0.024 ^{C}	162.69±0.456 ^{C}
T4 (Monster x2)	6.96±0.052 ^{A}	188.11±0.896 ^{A}
LSD	0.251	4.563

Different letters indicates statistical difference at $P < 0.05$

Oxidative stress markers (MDA, GSH)

As shown in Table 3, malondialdehyde (MDA) levels significantly increased in all treatment groups compared to the control, indicating elevated lipid peroxidation and oxidative stress. The highest MDA level was seen in group T4 (10.20 ± 0.247 µmol/L) versus the control (24.39 ± 0.251 µmol/L). Conversely, reduced glutathione (GSH) levels significantly decreased, with the lowest concentration found in T4 (8.71 ± 0.035 µmol/L) compared to the control (3.001 ± 0.012 µmol/L). These data confirm that chronic energy drink intake leads to oxidative imbalance by increasing ROS and depleting antioxidant defenses.

Table 3: Effect of red bull and monster on oxidative stress markers in immature male rats.

Group	MDA (µmol/L)	GSH (µmol/L)
Control	24.39±0.251A	3.001±0.012^{E}
T1 (Red Bull)	16.67±0.124^{B}	5.39±0.022^{D}
T2 (Red Bull x2)	12.24±0.253^{D}	7.28±0.05^{B}
T3 (Monster)	14.05±0.362^{C}	6.23±0.019^{C}
T4 (Monster x2)	10.20±0.247^{E}	8.71±0.035^{A}
LSD	1.002	0.456

Different letters indicates statistical difference at $P < 0.05$

Discussion

Effect of energy drinks on cardiac biomarkers (Troponin, LDH, CKP)

The significant elevation in serum troponin, LDH, and CKP in all energy drink-treated groups, particularly T4 (double dose of Monster), suggests early myocardial damage. These enzymes are reliable markers of cardiomyocyte injury, often released following membrane disruption or ischemia (Chimezie, 2013; Ahmad *et al.*, 2022; Gualberto *et al.*, 2024). Troponin I is highly specific to cardiac tissue and its elevation in rats following chronic ED exposure parallels human clinical findings indicating myocardial stress even in the absence of infarction (El-Rahman *et al.*, 2025). The rise in CKP and LDH, which are involved in cellular energy metabolism, further supports

the hypothesis that cardiac tissue undergoes metabolic strain under the influence of excessive stimulant intake (Sreekumar *et al*., 2023).

Mechanistically, the presence of high doses of caffeine up to 80 mg per can and its synergism with taurine can trigger excessive β-adrenergic stimulation, leading to calcium overload and increased mitochondrial workload (Martins *et al*., 2024). This overstimulation can cause cellular injury, enzyme leakage, and even necrosis in prolonged exposure scenarios (Chami and Di Primio, 2024). Animal studies and human case reports have documented arrhythmias, QT prolongation, and ECG changes following ED use, confirming the cardiotoxic nature of these beverages (Shah *et al*., 2019). The damage may be subclinical initially but can progress with sustained use, especially in young or developing cardiac systems.

Impact on electrolyte balance (Sodium and Potassium)

Our results demonstrated a clear and significant elevation in both sodium and potassium levels across treatment groups, with the highest values in T4. These ions are crucial for maintaining normal cardiac rhythm, action potential duration, and overall electrochemical stability. Caffeine and taurine influence renal tubular reabsorption and sodium-potassium ATPase activity, possibly explaining the observed dysregulation in electrolyte homeostasis (Ali, 2023; Mandilaras *et al*., 2022). Additionally, these components can indirectly stimulate the renin-angiotensin-aldosterone system (RAAS), enhancing sodium retention and altering potassium excretion.

Such ionic disturbances are known risk factors for ventricular arrhythmias and conduction abnormalities. Hyperkalemia, in particular, shortens repolarization time, increases cell excitability, and may trigger fatal events in susceptible individuals (Chami and Di Primio, 2024). Furthermore, taurine though considered cardioprotective in isolation may paradoxically increase membrane permeability in combination with caffeine, exacerbating potassium loss from intracellular stores and contributing to the net serum increase (Babu *et al*., 2021).

Oxidative stress and antioxidant status (MDA, GSH)

MDA, a product of lipid peroxidation, was significantly elevated in all treatment groups, while GSH a primary intracellular antioxidant was markedly depleted, confirming redox imbalance. These changes indicate the onset of oxidative stress, likely mediated by increased reactive oxygen species (ROS) production due to excessive mitochondrial activity (Jomova *et al*., 2023; Trofin *et al*., 2025). The liver and heart are especially vulnerable to oxidative stress due to their high oxygen demand. ED-induced mitochondrial disruption impairs electron transport chain function, resulting in electron leakage and formation of superoxide radicals (Patani *et al*., 2023). These radicals attack polyunsaturated fatty acids in membrane phospholipids, producing MDA.

GSH depletion, on the other hand, compromises cellular detoxification processes. Reduced GSH levels were observed across all ED-treated groups, suggesting consumption of antioxidant reserves in response to accumulating ROS (El- Rahman *et al*., 2025). This antioxidant exhaustion may predispose tissues to chronic injury and apoptosis. Moreover, synthetic additives, colorants, and sugars in energy drinks may amplify oxidative burden, especially in immature organisms where antioxidant systems are underdeveloped (Trofin *et al*., 2025; Jomova *et al*., 2023).

Combined cardiotoxic impact and public health concerns

Energy drinks are often marketed to adolescents, athletes, and university students for enhanced energy and focus. However, the accumulating evidence from this study and global reports supports serious concerns over their cardiotoxicity, particularly when consumed chronically or in large amounts (Costa *et al*., 2023; Higgins *et al*., 2017). Several clinical reports have linked energy drink overuse to sudden cardiac death, myocardial infarction, and even cardiac arrest in young individuals with or without pre-existing conditions. The lack of strict regulation, especially in developing regions, exacerbates the risk (Chen *et al*., 2019). This study underscores the importance of public awareness campaigns, regulatory policies limiting caffeine content, and the necessity of warning labels targeting youth. Moreover, integrating cardiac biomarker screening and ECG analysis in frequent users may be a preventive approach in high-risk populations.

Conclusion

This study demonstrated that chronic administration of Red Bull and Monster energy drinks, particularly at high doses, leads to significant biochemical and physiological disturbances in immature male rats. Notably, the consumption of these beverages resulted in dose-dependent elevations in cardiac biomarkers (troponin I, LDH, CKP), electrolyte imbalances (increased Na^+ and K^+), and pronounced oxidative stress, as evidenced by increased MDA and reduced GSH levels. These alterations collectively suggest that commercial energy drinks have cardiotoxic, electrolytic, and oxidative consequences, even in the absence of overt histological damage. The effects were more severe with Monster compared to Red Bull, and more prominent in double-dose groups, highlighting the importance of dose in mediating toxicity. Given the widespread consumption of energy drinks among adolescents and young adults, the findings raise serious concerns about their long-term safety, especially during developmental stages. Regulatory measures, public awareness, and further mechanistic studies are warranted to mitigate potential health risks associated with excessive energy drink consumption.

Declarations

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IRB approval

This study was approved by the Institutional Review Board (IRB) of the University of Al-Qadisiyah, College of Education, under Reference No. 11571, dated 15/10/2024.

Ethical statement

This research was approved by the Ethical Approval Committee (Reference No. 11571, dated 15/10/2024). The study also received institutional approval from the University of Al-Qadisiyah, College of Education. All procedures involving animals were performed in accordance with institutional and national ethical guidelines.

Declaration of generative AI and AI-assisted technologies in the writing process

No Generative AI and AI-assisted technologies wer used in the writing process.

Conflict of interest

The authors have declared no conflict of interest.

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