Effect of Energy Drinks (3 Brands) Consumption on Oxidative Stress Parameters in Wistar Rats Exposed to Stress

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DOI: https://doi.org/10.51244/IJRSI.2026.1308000037

Received: 16 August 2026; Accepted: 22 August 2026; Published: 29 August 2026

ABSTRACT

Energy drinks (EDs) are widely consumed beverages containing high doses of caffeine and metabolic stimulants. This study investigated the effects of three brands of energy drinks on oxidative stress parameters in Wistar rats exposed to stress. Thirty adult Wistar rats were divided into five groups of six animals each. Groups 1: (control group) was sub-divided into two groups of three animals each (positive and negative); the positive control were given food and distilled water and exposed to heat stress, negative control received food and distilled water only. Group II, III and IV: 3 ml/kg of body weight dosage of the energy drinks was administered orally (Climax, Power Horse, and Monster) respectively, two times daily for three weeks. Group V: 0.1 ml/kg of the epinephrine (standard drug) was administered intraperitoneally, twice daily for three weeks. All groups except the negative control were exposed to heat stress daily for 10 minutes for a period of 3 weeks (one group after the other). Oxidative stress markers including malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), and glutathione reductase (GSH) were analyzed using standard spectrophotometric methods. Significant decreases were observed in MDA levels across heat-exposed, Climax-treated, Monster-treated, and epinephrine-treated groups ($p<0.05$). CAT activity significantly decreased in all treatment groups compared to controls ($p<0.05$). Power Horse treatment significantly increased SOD levels (66.66±10.39 U/mL) compared to controls (43.33±2.72 U/mL; $p<0.05$). GSH levels significantly increased across all treatment groups ($p<0.05$). The most pronounced alterations occurred in the Power Horse-treated group. Energy drink consumption induces significant alterations in oxidative stress parameters, particularly elevating SOD levels. Further investigation should be carried out to highlight potential health risks associated with chronic energy drink consumption.

Keywords: Energy drinks, oxidative stress, malondialdehyde, superoxide dismutase, catalase, glutathione reductase, Wistar rats.

INTRODUCTION

Energy drinks (EDs) are beverages that contain large amounts of caffeine and other stimulants such as taurine, carbohydrates, glucuronolactone, and B-complex vitamins (Attila and Çakır, 2011). The energy-boosting effect comes mainly from sugar, while the stimulating effect comes from caffeine, which is comparable to one cup of coffee containing at least 80 mg of caffeine (Miller, 2008). Caffeine is one of the most commonly consumed alkaloids worldwide and can also be found in coffee and tea. However, consuming high doses of caffeinated EDs has been shown to cause abnormal stimulation of the nervous system (Dworzariski, Opielak and Burdan, 2009) as well as adverse effects in the cardiovascular, hematologic, and gastrointestinal systems (Seifert, Schaechter and Hershorin, 2011).

Energy drinks have also been reported to enhance physical performance, reduce mental exhaustion, and improve mood (Gheith, 2017; Kassab et al., 2018). These effects are attributed to ingredients such as carbohydrates, vitamins, caffeine, L-carnitine, glucuronolactone, and herbal supplements (Munteanu et al.,

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VIII August 2026

2014). Previous studies have confirmed the harmful effects of energy drinks on several organs and tissues, including the heart, kidney, and liver (Khayyat et al., 2012; Bukhar et al., 2012; Taiwo and Adesokan, 2017). Munteanu et al. (2014) also showed that energy drinks affect certain blood parameters and are a risk factor for metabolic diseases. Researchers have raised concerns about combining alcohol, which has known toxic effects, with energy drinks (Rengin et al., 2017; Díaz et al., 2016; Ugwuja, 2014).

Studies have revealed that EDs contain mainly taurine, glucuronolactone, caffeine, ginseng, and guarana (Seifert et al., 2011). These substances, most of which act as stimulants, are not regulated by the US Food and Drug Administration (FDA). The levels of these stimulants vary among different brands of EDs and are often higher than allowable values (Tanne, 2012). Research has shown that caffeine levels in EDs range from 50 to 505 mg per can, much higher than the caffeine content of one can of Coke (34 mg) (Burrows et al., 2013).

Reports of significant health problems due to energy drink consumption have increased in recent years. In 2013, ED-associated emergency visits in the US doubled from 10,068 in 2007 to over 20,000 in 2011 (Ali et al., 2015). A major problem in understanding the link between energy drinks and their adverse effects is that very little is known about the toxicity of the various compounds in them. However, based on reported cases of ED-associated health problems and the well-established effects of their active ingredients, it is very likely that the observed adverse effects are linked to their composition (Seifert et al., 2011).

Studies elsewhere have raised concerns about limited safety data on caffeinated EDs (Howland et al., 2010). In addition, there is not enough information about the effects of energy drinks on oxidative stress and lipid peroxidation. Oxidative stress is linked to several diseases such as diabetes, cancer, and cardiovascular disorders (Reis et al., 2017). However, reactive oxygen species (ROS) can also have beneficial functions in cell signaling and fighting microorganisms. Increased ROS production or depletion of the antioxidant defense system can cause an imbalance between oxidants and antioxidants (Ilaiyaraja and Khanum, 2011). Excess ROS damages lipids, proteins, and DNA (Bloomer et al., 2013). To prevent this damage, cells have a defense mechanism consisting of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione reductase (GSH) (Ilaiyaraja and Khanum, 2011).

Statement of the Problem

Not many studies have assessed the effects of energy drinks on the antioxidant system. Research has shown that energy drinks with caffeine, taurine, and guarana reduced the activity of SOD and CAT in vitro, but no changes in GSH activity were observed (Zeidán-Chuliá et al., 2013).

Oxidative stress occurs when there is an imbalance between the production of oxidant compounds and antioxidant defense systems in the body (Halliwell, 1996). Free radicals and ROS are constantly produced at low levels as part of normal biological processes (Valko et al., 2007). When produced in excess, they can cause cell injury, such as damage to lipids, proteins, and nucleic acids (Valko et al., 2007), or even cell death. The brain is particularly vulnerable to damage from free radicals due to its high oxygen consumption, abundant lipid content, and insufficient antioxidant enzymes compared to other tissues (Halliwell and Gutteridge, 1997).

Considering that energy drink consumption among people is high and that it has shown reducing effects on antioxidant markers and toxicity to vital systems like the central nervous system, it is necessary to evaluate the effects of energy drinks and their constituents on oxidative stress parameters in Wistar rats.

Aim and Objectives

Aim: To determine the effect of energy drinks (3 brands) consumption on oxidative stress parameters in Wistar rats exposed to stress.

Specific Objectives to:

i. determine the effect of energy drinks on Glutathione reductase (GSH) in Wistar rats.

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ii. determine the effect of energy drinks on Catalase (CAT) in Wistar rats.

iii. determine the effect of energy drinks on Superoxide dismutase (SOD) in Wistar rats.

iv. determine the effect of energy drinks on Malondialdehyde (MDA) in Wistar rats.

Significance of the Study

This study is important because it will add to existing knowledge on the effect of energy drink consumption on oxidative stress parameters, especially the risks of chronic consumption. It will also serve as a basis for further research on the effects of energy drinks and their constituents on antioxidant markers.

Scope of the Study

This study was carried out to determine the effect of three brands of energy drinks on oxidative stress parameters in albino Wistar rats. The study used biochemical assays with a spectrophotometer to determine the concentration and activity of various antioxidant markers (GSH, CAT, SOD, and MDA) in samples obtained from Wistar rats exposed to stress.

MATERIALS AND METHODS

Experimental Animals and Housing

Thirty adult Wistar rats were divided into five groups of six animals per group. The animals were kept in well-aerated cages (6 per cage) in a climate-controlled room (21 to 24°C) with a 12-hour light/dark cycle. The animals had free access to water and were fed balanced food for laboratory rats (daily intake 20 g per animal). The animal house was cleaned daily, and waste was removed for animal comfort (Carillon et al., 2014).

Experimental Design

All 30 Wistar rats were exposed to heat stress to induce hyperthermic stress for 10 minutes daily for 3 weeks (one group after the other), except for the negative control. After daily heat stress exposure, the following procedures were carried out:

Group 1 (Control group): The control group was sub-divided into two groups of three animals each (positive and negative control).

Positive control was given food, distilled water orally, and exposed to heat stress daily.

Negative control was given only food and distilled water daily for three weeks.

Group 2 (Climax-treated): 3 ml per kg body weight of energy drink one (Climax) was given orally, twice daily for three weeks.

Group 3 (Monster-treated): 3 ml per kg body weight of energy drink two (Monster) was given orally, twice daily for three weeks.

Group 4 (Power Horse-treated): 3 ml per kg body weight of energy drink three (Power Horse) was given orally, twice daily for three weeks.

Group 5 (Epinephrine-treated): 0.1 ml of the standard drug (epinephrine) per kg body weight was given intraperitoneally, twice daily for three weeks.

Basis for the 3mL/kg Dosage and Administration Protocol

1. **Dose-Equivalent Rationale (Interspecies Allometric Scaling):** The selection of 3mL/kg body weight administered twice daily (6mL/kg/day total) is based on body surface area (BSA) normalization according

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to standard FDA interspecies conversion principles (Reagan-Shaw et al., 2008). For an average human adult weighing 70kg, a standard moderate-to-high daily consumption of energy drinks (e.g., two 250mL cans or one 500ml can) equates to approximately 7.1mL/kg/day. Applying the animal-to-human scaling factor (K_m ratio of 37 / 6 = 6.2 for rats):

$$\text{Human Equivalent Dose (HED)} = \text{Rat Dose} \times \left(\frac{(K_m \text{ (rat)}}{K_m \text{ (human)}} \right)$$

The 6mL/kg/day administration in Wistar rats models a clinically relevant, sub-chronic human intake regime rather than an acute toxicological overdose. Dividing the volume into two daily doses (3mL/kg twice daily) mimics human episodic consumption patterns while preventing gastric overload in rats.

2. **Compositional Basis across Brands:** Although the volumetric dose (3mL/kg) remains uniform across experimental groups, each brand delivers a distinct pharmacological load of active stimulants:

   i. **Caffeine:** Acts as an adenosine receptor antagonist that upregulates central and peripheral sympathetic tone, elevating baseline cellular metabolic rates.

   ii. **Taurine:** Functions as an osmoregulator and neuromodulator that can interact with cellular calcium handling and endogenous antioxidant mechanisms.

   iii. **Glucuronolactone & B-Vitamins:** Act as metabolic intermediates in hepatic detoxification pathways and oxidative phosphorylation, influencing mitochondrial electron flux.

   iv. **Carbohydrates (Glucose/Sucrose):** Provide rapid glycemic input, shifting energetic turnover and reactive oxygen species (ROS) generation during cellular respiration.

Role of Epinephrine and Justification as a Positive Control

i. **Physiological Role**

Epinephrine (adrenaline) is a non-selective α- and β adrenergic receptor agonist. It serves as the primary endogenous hormone involved in the systemic "fight-or-flight" response, driving sympathetic activation, glycogenolysis, cardiac output, and peripheral vascular constriction.

ii. **Justification as a Positive Control (Stress Model Validation)**

In physiological research, epinephrine is used as a standard reference drug to establish a validated baseline of neuroendocrine-mediated stress. Exogenous administration of epinephrine elevates circulating catecholamines, stimulating membrane-bound NADPH oxidases and autoxidation pathways that produce systemic oxidative stress.

iii. **Comparative Reference Standard:** By comparing the energy drink-treated group against the epinephrine group, this study evaluates whether commercial energy drink formulations (which contain central nervous system stimulants like caffeine) induce biochemical shifts and enzymatic redox alterations via pathways similar to classical adrenergic activation.

Sample Collection

A total of 5 mL blood samples were collected from each group by cardiac puncture and stored in sample bottles with lithium heparin as an anticoagulant.

Analysis of Oxidative Stress Parameters

Oxidative stress parameters were determined in the Biochemistry Research Laboratory of the Department of Biochemistry, University of Port Harcourt. Oxidative stress was quantified by measuring the concentration of

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malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and glutathione reductase (GSH) using a Jenway spectrophotometer. The concentration was expressed as nmol/mL.

Determination of MDA Concentration

Plasma malondialdehyde (MDA) concentration was assayed according to Ohkawa et al. (1979) as modified by Tanko et al. (2014). Briefly, 2.4 mL of 1/12 H_2SO_4 and 0.3 mL of 10% sodium tungstate (Na_2WO_4) were added to 0.3 mL of plasma and centrifuged at 2054 × g for 10 minutes. Then 0.5 mL of distilled water, 3 mL of 0.05 N HCl, and 1.0 mL of 1% thiobarbituric acid (TBA) were added and made up to 5 mL with distilled water. The mixture was kept in a water bath at 95°C for 60 minutes. After cooling, the mixture was centrifuged again at 2054 × g, and the supernatant was collected and its absorbance measured in a visible spectrophotometer (OPTIZEN 1412V, Korea) at 532 nm. A standard control was prepared by adding 0.5 mL of distilled water, 3 mL of 0.05 N HCl, and 1 mL of 1% TBA to 0.3 mL of 10 nmol/L TEP (tetraethoxipropane). Absorbance was measured as described. MDA concentration was expressed as nmol/mL of plasma.

Determination of GSH Level

Glutathione reductase (GSH) level was quantified as described by Hu (cited in Berar et al., 2015). Plasma was mixed with 10% tricarboxylic acid (TCA), and after 10 minutes, it was centrifuged. The supernatant was separated, and 1.7 mL phosphate buffer (pH 8) and 1 mL o-phthalaldehyde were added. After 15 minutes, emission intensity at 420 nm was measured at an excitation of 350 nm.

Determination of Catalase (CAT) Activity

Catalase activity was assayed using the method of Aebi (1984) in erythrocyte hemolysate. Decomposition of H_2O_2 was followed directly by monitoring the reduction of absorbance at 240 nm. Enzyme activity was calculated as catalytic content of a sample. Data were expressed as k/g Hb.

Determination of Superoxide Dismutase (SOD) Activity

Superoxide dismutase (SOD) activity was measured as described by Beutler (1984) and modified by Tanko et al. (2014). Briefly, 500 µL of hemolysate was added to 1.5 mL of ice-cold distilled water, followed by 0.5 mL ethanol and 0.3 mL chloroform. The solution was mixed after each addition, vortexed for 30 seconds, and allowed to stand for 5 minutes for complete precipitation. The mixture was centrifuged at 2683 × g for 5 minutes. Then 3.9 mL of buffer was added to 70 µL of the hemolysate extract, vortexed, and equilibrated with air for 10 minutes. This was followed by addition of 40 µL of 20 mM pyrogallol solution. The blank was prepared by replacing the 70 µL of hemolysate with 20% (v/v) ethanol. The spectrophotometer was zeroed at 420 nm with deionized water, and the absorbance of the blank and samples was read in 1 minute.

Method of Data Analysis

Results were presented as Mean ± SEM in tables and charts. Mean values between groups were compared using analysis of variance (ANOVA). Differences were considered statistically significant at $p<0.05$.

RESULTS

Table 1: Pattern of response of oxidative markers after 21-day treatment with various brands of energy drinks

Group	Malondialdehyde (nmol/mL ± SEM)	Catalase (U/mL ± SEM)	Superoxide Dismutase (U/mL ± SEM)	Glutathione Reductase (U/mL ± SEM)
Control group	2.16 ± 0.18	43.92 ± 6.14	43.33 ± 2.72	3.56 ± 0.04
Heat-exposed group	1.64 ± 0.05*	8.47 ± 1.89*	52.22 ± 1.36	4.67 ± 0.01*

Climax-treated group	1.62 ± 0.11*	6.74 ± 3.17*	44.44 ± 2.48	4.05 ± 0.06*
Monster-treated group	1.60 ± 0.03*	14.44 ± 9.92*	35.55 ± 5.44	4.30 ± 0.04*
Power Horse-treated group	1.98 ± 0.08	8.38 ± 1.41*	66.66 ± 10.39*	4.28 ± 0.07*
Epinephrine-treated group	1.70 ± 0.09*	37.57 ± 11.38	46.66 ± 5.15	4.16 ± 0.07*

Values are presented as mean ± SEM. N=5. indicates values are statistically significant compared to control values ($p<0.05$).

Malondialdehyde (MDA)

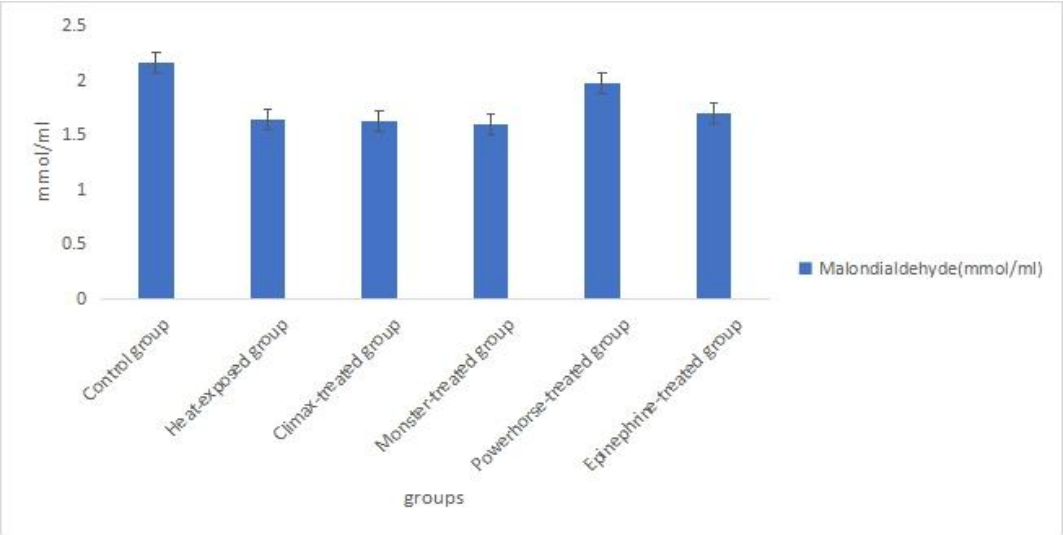

Figure 1: Pattern of response of Malondialdeyde after 21 day-treatment with various brands of energy drinks

Figure 1 shows the pattern of response of MDA after 21-day treatment. There was a statistically significant decrease in mean MDA levels in the heat-exposed group (1.64 ± 0.05), Climax-treated group (1.62 ± 0.11), Monster-treated group (1.60 ± 0.03), and epinephrine-treated group (1.70 ± 0.09) compared to the control group (2.16 ± 0.18) at $p<0.05$. The Power Horse-treated group (1.98 ± 0.08) showed a decrease that was not statistically significant.

Superoxide Dismutase (SOD)

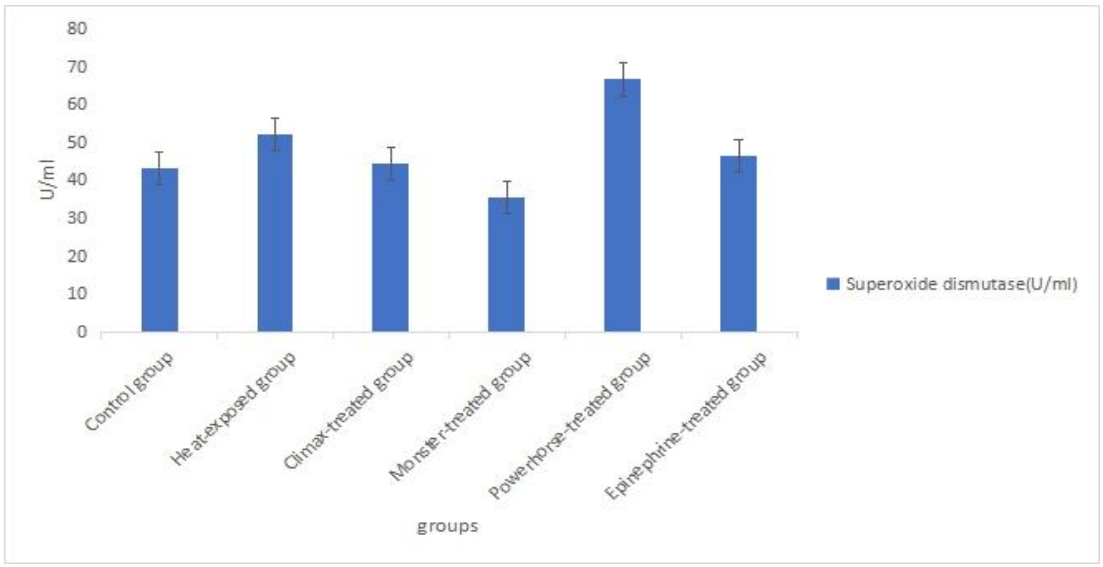

Figure 2: Pattern of response of Superoxide dismutase after 21 day-treatment with various brands of energy drinks

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VIII August 2026

Figure 2 shows that there was a statistically significant increase in the mean SOD level in the Power Horse-treated group (66.66 ± 10.39 U/mL) compared to the control group (43.33 ± 2.72 U/mL) at $p<0.05$. Other groups showed non-significant changes.

Glutathione Reductase (GSH)

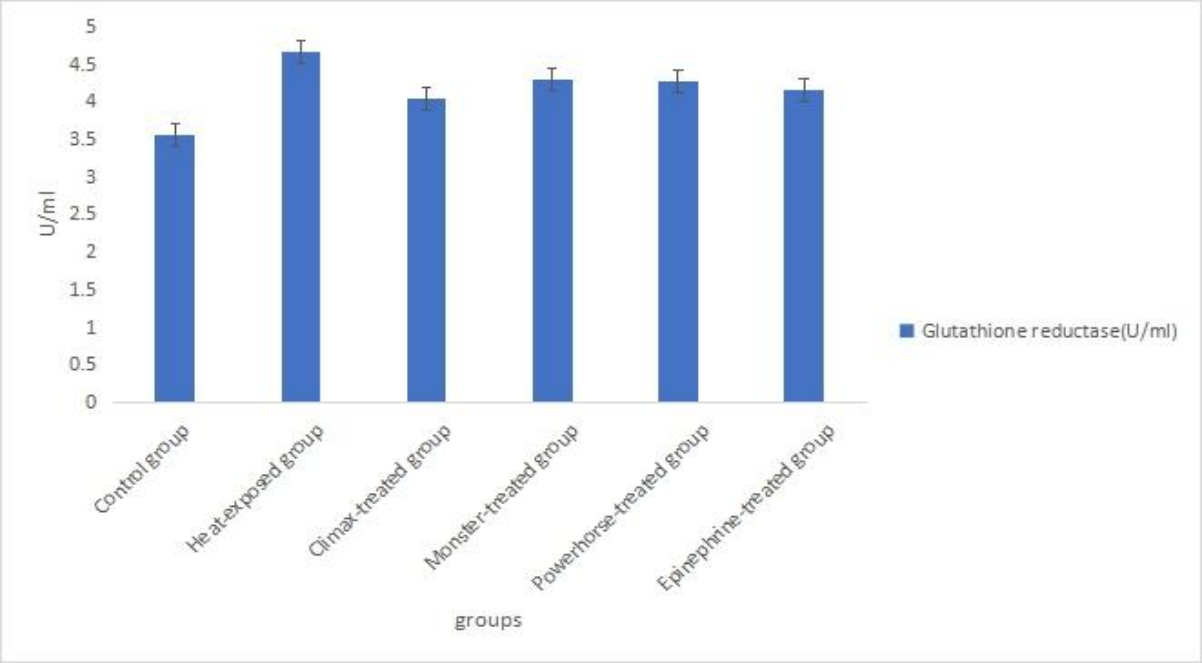

Figure 3: Pattern of response of Glutathione reductase after 21 day-treatment with various brands of energy drinks

Figure 3 shows that GSH levels were statistically significantly increased in all treatment groups: heat-exposed group (4.67 ± 0.01 U/mL), Climax-treated group (4.05 ± 0.06), Monster-treated group (4.30 ± 0.04), Power Horse-treated group (4.28 ± 0.07), and epinephrine-treated group (4.16 ± 0.07) compared to the control group (3.56 ± 0.04) at $p<0.05$.

Catalase (CAT)

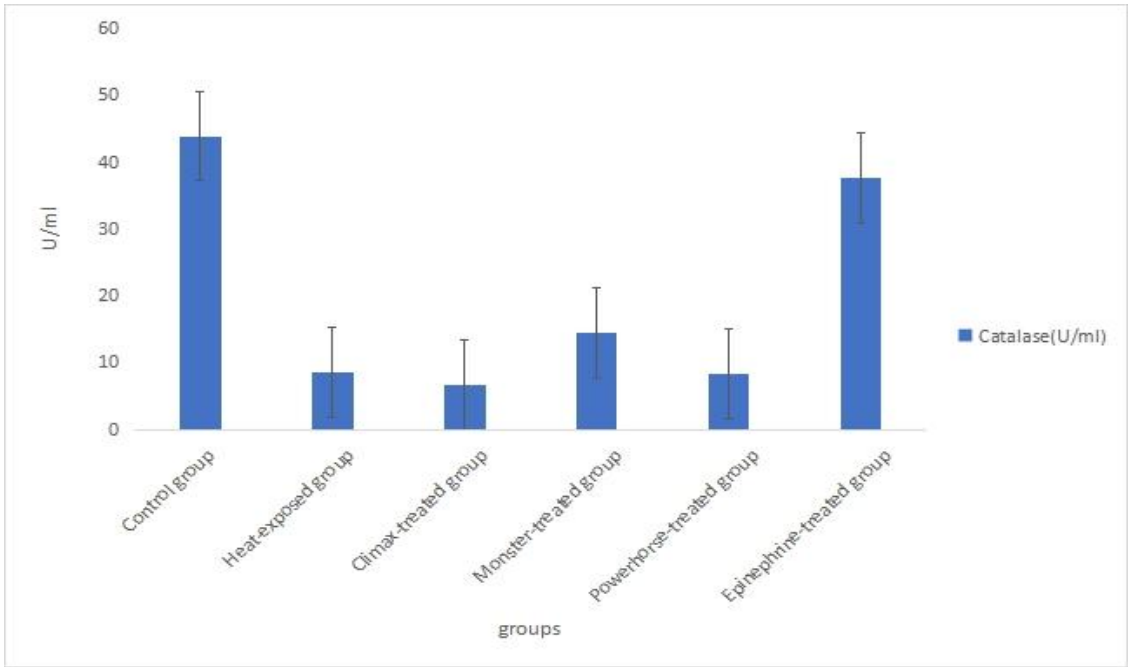

Figure 4: Pattern of response of Catalase after 21 day-treatment with various brands of energy drinks

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XIII Issue VIII August 2026

Figure 4 shows that CAT activity statistically significantly decreased in the heat-exposed group (8.47 ± 1.89 U/mL), Climax-treated group (6.74 ± 3.17), Monster-treated group (14.44 ± 9.92), and Power Horse-treated group (8.38 ± 1.41) compared to the control group (43.92 ± 6.14) at $p<0.05$. The epinephrine-treated group (37.57 ± 11.38) showed a decrease that was not statistically significant.

DISCUSSION

Malondialdehyde (MDA)

This study revealed a statistically significant decrease in mean MDA levels in the heat-exposed, Climax-treated, Monster-treated, and epinephrine-treated groups compared to the control group. This finding is contrary to the study of Aleryani (2018), whose research indicated that energy drinks increased MDA levels. The decrease in MDA observed in our study may be due to the presence of antioxidant compounds in energy drinks, such as taurine and certain B-vitamins, which may enhance free radical scavenging (Higgins et al., 2010). Additionally, the three-week exposure period may have induced adaptive responses not seen in shorter-term studies.

Catalase (CAT)

The study showed that CAT significantly decreased in the heat-exposed group, Climax-treated group, Monster-treated group, and Power Horse-treated group compared to the control group. Catalase is an important antioxidant enzyme that works with the non-enzymatic antioxidant system to protect cells from oxidative damage by free radicals (Mansy et al., 2018). Indeed, antioxidant enzymes are the first line of defense that protects cells from oxidative stress-induced damage. The significant reductions in blood levels of CAT due to sub-chronic administration of energy drinks may overwhelm the neutralizing capacities of the antioxidant enzymes.

This catalase reduction may be due to the high caffeine content of EDs, which may directly inhibit CAT activity (Zeidán-Chuliá et al., 2013). Additionally, the oxidative burden from ED metabolism may overwhelm the antioxidant system, leading to enzyme inactivation (Goc et al., 2019). The lesser reduction in CAT activity in the epinephrine-treated group suggests that ED ingredients, rather than stress alone, are mainly responsible for this effect.

Superoxide Dismutase (SOD)

In the present study, a statistically significant elevation in mean SOD levels was observed exclusively in the Power Horse-treated group compared to the control group. This differential response suggests that specific active ingredients or formulations within particular energy drink brands may uniquely modulate systemic antioxidant defense mechanisms. Superoxide dismutase serves as a primary enzymatic antioxidant that catalyzes the dismutation of superoxide radicals (O_2) into hydrogen peroxide (H_2O_2) and molecular oxygen, thereby acting as a critical front-line buffer against endogenous oxidative stress. The upregulation of SOD activity in this group likely reflects an adaptive physiological response or a compensatory upregulation of cellular redox machinery to mitigate an altered oxidative environment.

The selective alteration in SOD activity highlights the variable impact of complex commercial formulations, where interactive or synergistic effects among constituent ingredients such as caffeine, taurine, and B-complex vitamins can influence enzymatic kinetics (Poulos and Pasch, 2015; Reis et al., 2017). Notably, while SOD induction functions as a protective dismutation mechanism, efficient redox homeostasis requires a proportional elevation in downstream hydrogen peroxide-clearing enzymes, such as catalase (CAT) or glutathione peroxidase. In the absence of a corresponding upregulation in CAT, robust SOD activity can lead to an accumulation of intermediate H_2O_2 flux, altering the steady-state equilibrium of reactive oxygen species within the vascular and cellular compartments (Memudu et al., 2020). Consequently, this localized elevation in SOD activity illustrates a complex pattern of antioxidant response characterized by shifted enzymatic ratios rather than uniform radical scavenging capacity.

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Glutathione Reductase (GSH)

GSH was significantly increased in all treatment groups: heat-exposed, Climax-treated, Monster-treated, Power Horse-treated, and epinephrine-treated compared to the control group. Increased GSH levels will maintain the supply of reduced glutathione. This is in line with the studies of Mansy et al. (2018) and Aleryani (2018), whose research indicated that energy drinks significantly decreased glutathione levels. The universal GSH increase across all treatment groups suggests that all three ED brands stimulate the glutathione antioxidant system, possibly through activation of antioxidant response pathways (Birben et al., 2012). This compensatory response may partially offset the harmful effects of CAT reduction and SOD elevation.

Brand-Specific Effects

The distinct responses observed across the evaluated energy drink brands highlight the importance of formulation-specific assessments when examining cellular redox regulation. Power Horse administration induced the most prominent alterations in the antioxidant profile marked by a selective elevation in SOD activity whereas Climax and Monster treatments produced more moderate biochemical shifts. These observed variations are likely attributable to differences in key active constituents, such as caffeine, taurine, and supplementary stimulant concentrations, which vary significantly across commercial brands (Reid et al., 2017).

The concomitant reduction in CAT activity alongside altered SOD and GSH levels reflects a complex modulation of the enzymatic antioxidant system. Rather than acting as a uniform stimulus, chronic energy drink consumption appears to disrupt the functional balance between primary antioxidant enzymes, shifting the steady-state equilibrium of reactive oxygen species (Reuter et al., 2010). In particular, the disproportionate elevation of SOD relative to catalase creates a kinetic mismatch, where superoxide dismutation proceeds without a corresponding increase in downstream hydrogen peroxide clearance. Consequently, these formulation-dependent alterations in SOD, CAT, and GSH profiles illustrate distinct patterns of antioxidant enzyme modulation that alter overall redox homeostasis.

CONCLUSION AND RECOMMENDATIONS

Summary of Finding

This study determined the effect of three brands of energy drinks on oxidative stress parameters (GSH, CAT, SOD, and MDA) in Wistar rats. Thirty adult Wistar rats were divided into five groups of six animals each for the treatment groups, with the control group subdivided into positive and negative control groups of three animals each. Treatment substances were administered using standard laboratory procedures. Blood tests were performed after the treatment period, and the results were analyzed.

In summary, this study demonstrated that sub-chronic exposure to various energy drink formulations significantly alters the functional pattern of enzymatic and non-enzymatic antioxidant markers:

- **Malondialdehyde (MDA):** A statistically significant decrease in mean MDA levels was observed in the Climax-treated, Monster-treated, and epinephrine-treated groups compared to the control group.
- **Catalase (CAT):** CAT activity was significantly reduced in the Climax-treated, Monster-treated, and Power Horse-treated groups relative to controls.
- **Superoxide Dismutase (SOD):** A statistically significant elevation in mean SOD levels occurred uniquely in the Power Horse-treated group compared to the control group, reflecting formulation-specific modulation of enzymatic dismutation pathways.
- **Glutathione Reductase (GSH):** GSH levels were significantly increased across all experimental cohorts including the heat-exposed, Climax-treated, Monster-treated, Power Horse-treated, and epinephrine-treated groups compared to the control group.

Conclusion

This study demonstrated that energy drink consumption alters systemic redox homeostasis, as evidenced by significant brand-dependent shifts in enzymatic and non-enzymatic antioxidant parameters. Overall, the findings indicate that chronic exposure to commercial energy drinks is associated with distinct modulations of cellular oxidative stress responses. In view of the complex biochemical shifts observed across different formulations, these beverages should be consumed with caution.

The combination of elevated SOD activity and suppressed CAT capacity reflects an enzymatic kinetic imbalance, where increased superoxide dismutation yields intermediate H_2O_2 flux that exceeds downstream catalase clearance. Rather than acting as simple cellular stimulants, these findings suggest that chronic energy drink consumption disrupts the steady-state equilibrium of the antioxidant defense network, leading to altered regulation of cellular oxidative processes.

Recommendations

Based on the findings of this study, the following recommendations are made:

1. Consumers should limit their intake of energy drinks, especially chronic consumption.
2. Regulatory authorities should require better labeling of energy drink ingredients and their potential health risks.
3. Further research is needed to identify the specific ingredients responsible for the observed effects.
4. Public health campaigns should educate people, especially young adults, about the potential risks of energy drink consumption.
5. Long-term studies on the effects of energy drink consumption on human health are urgently needed.

Contribution to Knowledge

Consequent to the completion of this investigation, the following contributions are made to the body of scientific knowledge:

1. Energy drink consumption can cause significant changes in some oxidative stress parameters.
2. The findings of this study have added to existing literature on the effect of energy drink consumption on oxidative stress parameters.
3. This study provides evidence that different brands of energy drinks may have different effects on the body, highlighting the need for brand-specific safety assessments.

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