

**EFFECTS OF ENERGY DRINKS ON THE CARDIOVASCULAR SYSTEM
IN STUDENTS OF UNIVERSITY OF BENIN**



BY

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BMS2202161

DEPARTMENT OF PHYSIOLOGY

SCHOOL OF BASIC MEDICAL SCIENCE

COLLEGE OF MEDICAL SCIENCE

UNIVERSITY OF BENIN

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OCTOBER, 2025

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BMS22O2161

**A PROJECT SUBMITTED TO THE DEPARTMENT OF PHYSIOLOGY,
SCHOOL OF BASIC MEDICAL SCIENCE, UNIVERSITY OF BENIN,
BENIN CITY, EDO STATE, NIGERIA.**

**IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE
AWARD OF BACHELOR OF SCIENCE (B.Sc.) DEGREE IN
PHYSIOLOGY, UNIVERSITY OF BENIN, BENIN CITY, EDO STATE.**

OCTOBER, 2025.

CERTIFICATION

This is to certify that this project work on "**EFFECT OF ENERGY DRINKS ON THE CARDIOVASCULAR SYSTEM IN STUDENTS OF THE UNIVERSITY OF BENIN**" was carried out by **ODIASE DIVINE**, with matriculation number **BMS2202161**, in partial fulfillment of the requirements on the award of Bachelor of science (B.Sc.) Degree in the Department of Physiology, School of Basic medical sciences, College of medical sciences, University of Benin, Benin city, Edo state. Nigeria.

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DEDICATION

I dedicate this project work to God and also to my family for their support and encouragement.

ACKNOWLEDGEMENT

I would like to express my sincere gratitude to my parents for their support and to my supervisor Dr.(Mrs) B.O. EIYA for her timely and constructive corrections made towards the completion of this work. And also to my colleagues, Mirabel, Benedict and Osas whom through their commitments, Dedication and Cooperation was I able to complete this work

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ABBREVIATIONS

ED- Energy Drinks

CVS- Cardiovascular system

ECG- Electrocardiogram

EDTA- Ethylenediaminetetraacetic acid

TMAO- trimethylamine N-oxide

RDA- Recommended Daily Allowance

LDL- Low density Lipoproteins

HDL- High density Lipoproteins

VLDC- Very low density lipoproteins

TC- Total cholesterol

SBP- Systolic blood pressure

DBP- Diastolic blood pressure

ABSTRACT

According to the Food and Drug Administration, energy drinks are defined as liquid products that typically contain caffeine, with or without additional ingredients. As energy drink consumption rates and popularity continue to rise, it's crucial to monitor their usage prevalence and investigate both the short-term and long-term effects of regular consumption to better understand their impact. The study aimed to investigate the effect of energy drinks (EDs) on liver function tests and hematological indices among young adult consumers in the University of Benin. 40, apparently young healthy adults between the ages of 18-25 years studying at the University of Benin, were divided into 2 groups of 20 each. Group 1 which is the control group comprised of participants who were not regular consumers of energy drinks and would not consume energy drink (Predator) during the study period. Group 2 comprised of adults with a history of energy drink consumption and would also consume energy drink (Predator) daily for 2 weeks. Blood samples were collected at baseline and after 2 weeks of consumption. Data were subjected to statistical analysis using GraphPad Prism version 8.1 statistical package and relevant statistical values were obtained. An unpaired Student t-test was used and data were presented as mean \pm standard error of the mean (SEM). Values of $P < 0.5$ were considered statistically significant. The statistical values obtained were presented graphically in the form of bar charts. The consumption of energy drinks among young adults did not cause significant alterations in the serum lipid profile for most parameters measured. Specifically, total cholesterol, triglycerides, HDL, and LDL levels in both experimental groups showed no significant differences when compared with the control group ($P > 0.05$). However, VLDL levels exhibited a significant decrease in both groups relative to the control ($P < 0.05$), suggesting that energy drink intake may have a slight lipid-lowering effect on VLDL concentration. The electrocardiographic (ECG) parameters — including heart rate, and blood pressure but Parameters like QRS complex becomes wider in Group 1 and increasingly wider in group 2 but normal in Control, ST interval elevation is observed in Group 1 and more elevated in Group 2 but flat in control group. No significant difference between the Axis of Group 1 and Control group but Group 2 axis progressively slants to the left

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND OF STUDY

The Food and Drug Administration (FDA) defines energy drinks (EDs) as “a class of products in liquid form that typically contains caffeine, with or without other added ingredients (Andrea *et al.*, 2023). The last decade has witnessed the greatest rise in the consumption of non-steroidal energy supplements for the specific purpose of boosting athletic performance, weight loss and augment training adaptations (Bishoy *et al.*, 2017). These products typically contain caffeine and a blend of additional ingredients such as taurine, guarana, amino acids, L-carnitine and B-vitamins which are purported to improve energy levels, metabolism, and exercise performance (Patrick *et al.*, 2020). These legal stimulants can increase alertness, attention, and energy, as well as increasing blood pressure, heart rate, and breathing (Andrea *et al.*, 2023). Prominent examples of energy drinks include Red Bull, Monster, NOS, Rockstar, Lucozade, Eastroc Super Drink, Bang Energy, Fearless, Predator and 5 Hour Energy. Moreover, energy drink consumption is associated with an unhealthy lifestyle (such as poor dietary habits, sedentary lifestyle) and is implicated as a trigger for substance use (such as tobacco, alcohol, drugs) and other risk behaviors (such as gambling, screen addiction, violence,

driving while intoxicated) (Jee-Seon and Jeong-mi, 2024). Possibly one of the most interesting areas for research regarding the consumption of energy drinks associated with athletic performance and the observed cardiovascular risks would be to study the genetic markers that indicate a greater predisposition to improve performance by consuming energy drinks and a protective effect against the damage they can cause to the cardiovascular system (Jorge and David, 2021).

Acute consumption can elevate sympathetic activity, leading to increased blood pressure, heart rate, and impairments in endothelial function even in healthy individuals. These physiological effects may be particularly harmful in individuals with underlying or genetic cardiac vulnerabilities, such as channelopathies, potentially triggering arrhythmias and, in rare cases, sudden cardiac arrest (Bohn and Fillipini, 2018)

1.2 PROBLEM STATEMENT

Although large-scale, long-term studies are limited, case reports and clinical trials have reported QT-interval prolongation, coronary artery spasm, ventricular arrhythmias, and myocardial infarction following energy drink intake. There remains a significant gap in understanding the long-term cardiovascular consequences of habitual energy drink consumption, especially in high-risk populations like adolescents, individuals with latent heart conditions, or those

combining energy drinks with exercise, alcohol, or medications (Bohn and Phillipini, 2018).

Acute adverse effects on endothelial function, blood pressure, heart rate, and arrhythmias in healthy individuals, Increased sympathetic drive and QT prolongation noted in autonomic and hemodynamic studies, Randomized controlled evidence (JAMA trial) showing increases in blood pressure and sympathetic activation following energy drink ingestion, Review summarizing cardiovascular risks including arrhythmias, myocardial infarction, QT-interval prolongation, aortic dissection, and death, Pediatric cardiovascular complications, such as arrhythmias, blood flow changes, and interactions with drugs or alcohol, Specific risks for patients with genetic cardiac conditions leading to arrhythmias or sudden cardiac arrest, Reviews highlighting the combined stimulatory effects of multiple ingredients and long-term unknowns, especially in youth, Case-based evidence and concerns raised in broader discussion (e.g., *The Conversation*), including death and regulatory responses (Shah *et al*, 2016).

1.3 AIM OF STUDY

To evaluate the effects of energy drink consumption on the cardiovascular system, including changes in heart rate, blood pressure, electrocardiographic parameters, and overall cardiac function in healthy and at-risk individuals.

1.4 JUSTIFICATION OF STUDY

Physiologic effects on the cardiovascular system

Evidence of reported energy drinks-related cardiovascular adverse effects has helped to further raise suspicion of these beverages. It is widely believed that caffeine, particularly at high doses, is associated with multiple cardiac comorbidities including palpitations and a number of arrhythmias such as atrial fibrillation and supraventricular and ventricular ectopy (Bishoy *et al.*, 2017). Energy drink consumption has been associated with cardiac arrest, myocardial infarction, spontaneous coronary dissection, and coronary vasospasm. This association is strengthened with studies showing increased platelet aggregation, increased systolic blood pressure (SBP), and QTc prolongation (Sachin *et al.*, 2019). However, inconsistencies in the current articles render it difficult to draw firm conclusions with regard to the effects of energy drinks on cardiovascular and cerebrovascular variables. These inconsistencies are due, in part, to differences in methodologies, volume of drink ingested, and duration of post-consumption measurements, as well as subject variables during the test (Erik *et al.*, 2016).

1.5 RESEARCH QUESTIONS:

Does energy drink consumption have any effect on the heart rate and blood pressure of Young adults (17-25)

Does regular consumption of energy drinks in young adults (17–25) contribute to early signs of cardiovascular dysfunction?

How does energy drink intake influence ECG changes, such as QT interval prolongation, in the 17–25 age group?

Could energy drink induced changes in lipid profile lead to long term cardiovascular effects?

1.6 SPECIFIC OBJECTIVES:

To determine the effects of energy drink consumption on heart rate and blood pressure

To determine the ways by which intake of energy drink could result in cardiac dysfunction

To determine the effects energy drink possess on ECG study

To determine energy changes in lipid profiles and the possible effects that could occur on the cardiovascular system

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 ENERGY DRINKS

Energy drinks are non-alcoholic functional beverages formulated to enhance alertness, reduce fatigue, and improve mental and physical performance. Their effects arise mainly from stimulant and metabolic ingredients, such as caffeine, taurine, guarana, ginseng, B-vitamins, sugars, and L-carnitine (Reissig *et al.*, 2009). Unlike sports drinks that focus on fluid and electrolyte replacement, energy drinks act primarily on the central nervous system (CNS) and cardiovascular system, inducing physiological changes that can impact cardiac function, vascular tone, and metabolism (Higgins *et al.*, 2010).

The modern concept of energy drinks originated in Japan during the early 1960s, with beverages like Lipovitan D developed by Taisho Pharmaceutical. These were marketed as “tonic” drinks to combat fatigue among workers (Seifert *et al.*, 2011). In the 1980s, Austrian entrepreneur Dietrich Mateschitz adapted the Thai energy drink Krating Daeng into Red Bull, which was launched in Austria in 1987. Red Bull’s success revolutionized the market, setting the template for modern energy drinks with its distinct slim can, high caffeine content, and aggressive marketing toward young adults. By the 1990s and 2000s, energy drinks spread globally across

Europe, North America, Asia, and Africa, with brands such as Monster, Rockstar, Burn, and Power Horse becoming household names (Zucconi *et al.*, 2013). Today, the global market exceeds \$80 billion annually, and energy drinks are among the fastest-growing segments of the functional beverage industry.

Caffeine which is the most important ingredient in a regular 50cl bottle is a 160–320 mg CNS stimulant which increases alertness and reduces fatigue, Stimulates cardiac contractility and heart rate, causes vasoconstriction and elevated blood pressure (Higgins *et al.*, 2010).

Taurine constituent in a 50cl bottle is about 1000–2000 mg. it is an Amino acid aiding muscle contraction, osmoregulation, and neurotransmission Modulates calcium handling in cardiac cells; stabilizes membranes and may protect against arrhythmia; can potentiate caffeine's inotropic effects i.e effects that changes the force of heart contraction (Seifert *et al.*, 2011).

Guarana Extract contains about 200–400 mg is a Natural caffeine source that prolongs stimulant effect, additive to caffeine load and may increase heart rate and blood pressure (Reissig *et al.*, 2009).

Ginseng Extract constituent in a 50cl bottle ranges from 100–200 mg. It is an Adaptogenic herb improving endurance and reducing stress. Mild vasodilatory

and cardioprotective effects through nitric oxide synthesis whereas in excess may cause hypertension or palpitations (Higgins *et al.*, 2010).

Sugars (Glucose, Sucrose) 50–60 g (~200–240 kcal).

Rapid carbohydrate energy which could result in acute hyperglycemia and transient sympathetic activation for increase in alertness; chronic intake linked to insulin resistance, obesity, and endothelial dysfunction (Zucconi *et al.*, 2013).

L-Carnitine contains about 100–500 mg in a 50cl bottle which is usually known for facilitation of fatty acid transport into mitochondria and may enhance cardiac energy metabolism; excessive intake may elevate trimethylamine N-oxide (TMAO) levels associated with atherosclerosis risk (Seifert *et al.*, 2011).

B-Vitamins (B2, B3, B5, B6, B12) 50–200 % RDA. They are cofactors in energy metabolism. They support cardiac muscle energy production. Normally excessive niacin (B3) may cause vasodilation and flushing and overuse may lead to palpitations.

Glucuronolactone about 600 mg in a 50cl bottle. It is a Carbohydrate derivative aiding detoxification. Limited cardiovascular data and may synergize with caffeine in stimulating metabolism.

Carbonated Water & Citric Acid (Vehicle and acidulant). No direct cardiac effects, though carbonation may increase gastric absorption rate of caffeine.

Though energy drinks may provide some short-term benefits in these areas, their long-term effects on health and performance remain unclear (Gutiérrez-Hellín and Varillas-Delgado, 2021). With the rising consumption of so-called energy drinks over the last few years, there has been a growing body of literature describing significant adverse health events after the ingestion of these beverages with the most common being the cardiovascular and neurological systems purported by ingredients in energy drinks such caffeine as well as potentiating effects of other stimulants in these drinks (Ali *et al.*, 2015). Consumption of energy drinks has been associated with a wide range of detrimental health effects, spanning from mild symptoms such as anxiety, gastrointestinal disturbances, dehydration, nervousness, and tachycardia, to more severe and potentially life-threatening conditions including rhabdomyolysis, acute kidney injury, ventricular fibrillation, seizures, acute mania, and stroke, with documented cases even linking energy drink consumption to fatalities (Costantino *et al.*, 2023). The regulation of energy drinks has been challenging and the absence of regulatory oversight has resulted in aggressive marketing of energy drinks targeted primarily toward young adults (Nadeem *et al.*, 2021). As energy drink consumption rates and popularity continue

to rise, it's crucial to monitor their usage prevalence and investigate both the short-term and long-term effects of regular consumption to better understand their impact (Jagim *et al.*, 2022). There are no conclusive studies to support beneficial effects of energy drinks, but instead there is enough evidence about the adverse effects of some of the most common components of these beverages and many more studies are needed to determine the safety of energy drinks, whichn has a considerable toxic potential not adequately informed to the consumer (Sanchez *et al.*, 2015).

2.2 THE CARDIOVASCULAR SYSTEM

The cardiovascular system which is also called the circulatory system is responsible for transporting blood, nutrients, oxygen, carbon dioxide, hormones, and metabolic waste products throughout the body. It consists of the heart, blood vessels (arteries, veins, and capillaries), and blood (Guyton & Hall, 2020). This system maintains homeostasis by regulating temperature, pH, and fluid balance, while ensuring efficient oxygen and nutrient delivery to tissues (Widmaier *et al.*, 2019). The heart is a muscular, cone-shaped organ located in the thoracic cavity, between the lungs, within the mediastinum. It weighs approximately 250–350 grams in adults and is roughly the size of a clenched fist (Marieb & Hoehn, 2018). The heart is enclosed in a pericardium, a double-layered sac consisting of the

fibrous pericardium and the serous pericardium, which protects the heart and reduces friction during contraction (Sherwood, 2016). The heart is a muscular organ divided into four chambers which are two atria and two ventricles. It pumps oxygen-poor blood to the lungs via the pulmonary circulation and oxygen-rich blood to the body via the systemic circulation (Marieb & Hoehn, 2018). The heart's rhythmic contractions are initiated by electrical impulses generated and conducted through a specialized system of autorhythmic cells (Hall & Hall, 2020):

Sinoatrial (SA) node – located in the right atrium; acts as the natural pacemaker, generating impulses at ~70–80 beats per minute.

Atrioventricular (AV) node – delays the impulse to allow complete atrial emptying before ventricular contraction.

Bundle of His (AV bundle) – conducts impulses through the interventricular septum. Right and left bundle branches – carry impulses to each ventricle.

Purkinje fibers – distribute impulses throughout the ventricles, ensuring coordinated contraction (Marieb & Hoehn, 2018).

This conduction sequence produces the characteristic ECG waves: the P wave (atrial depolarization), QRS complex (ventricular depolarization), and T wave (ventricular repolarization) (Goldberger, 2017). Arteries carry blood away from the

heart under high pressure, veins return blood to the heart under lower pressure, and capillaries facilitate exchange of gases and nutrients between blood and tissues (Hall, 2020). Blood is composed of plasma (fluid), red blood cells (oxygen transport), white blood cells (immune defense), and platelets (clotting) (Sherwood, 2016). The systemic circulation distributes oxygenated blood from the left ventricle to tissues and returns deoxygenated blood to the right atrium. The pulmonary circulation carries deoxygenated blood from the right ventricle to the lungs for oxygenation and returns oxygen-rich blood to the left atrium (Guyton & Hall, 2020).

Cardiac output (CO) is the product of heart rate and stroke volume. It determines how much blood the heart pumps per minute. Its regulation occurs through, Neural control (The autonomic nervous system adjusts heart rate and vessel diameter via sympathetic and parasympathetic pathways). Intrinsic control via the Frank–Starling mechanism, where increased venous return stretches myocardial fibers, increasing contraction strength. Extrinsic control through autonomic innervation: the sympathetic system increases HR and contractility, while the parasympathetic system (via the vagus nerve) decreases HR (Hall & Hall, 2020). Hormonal control (Adrenaline, noradrenaline, and renin-angiotensin-aldosterone system influence

blood pressure and cardiac activity). Local autoregulation (Tissues adjust blood flow according to metabolic needs) (Widmaier *et al.*, 2019).

The cardiovascular system ensures oxygen and nutrient delivery, waste removal, immune transport, and temperature regulation. Dysfunction can lead to disorders such as hypertension, atherosclerosis, heart failure, and myocardial infarction (Libby *et al.*, 2019).

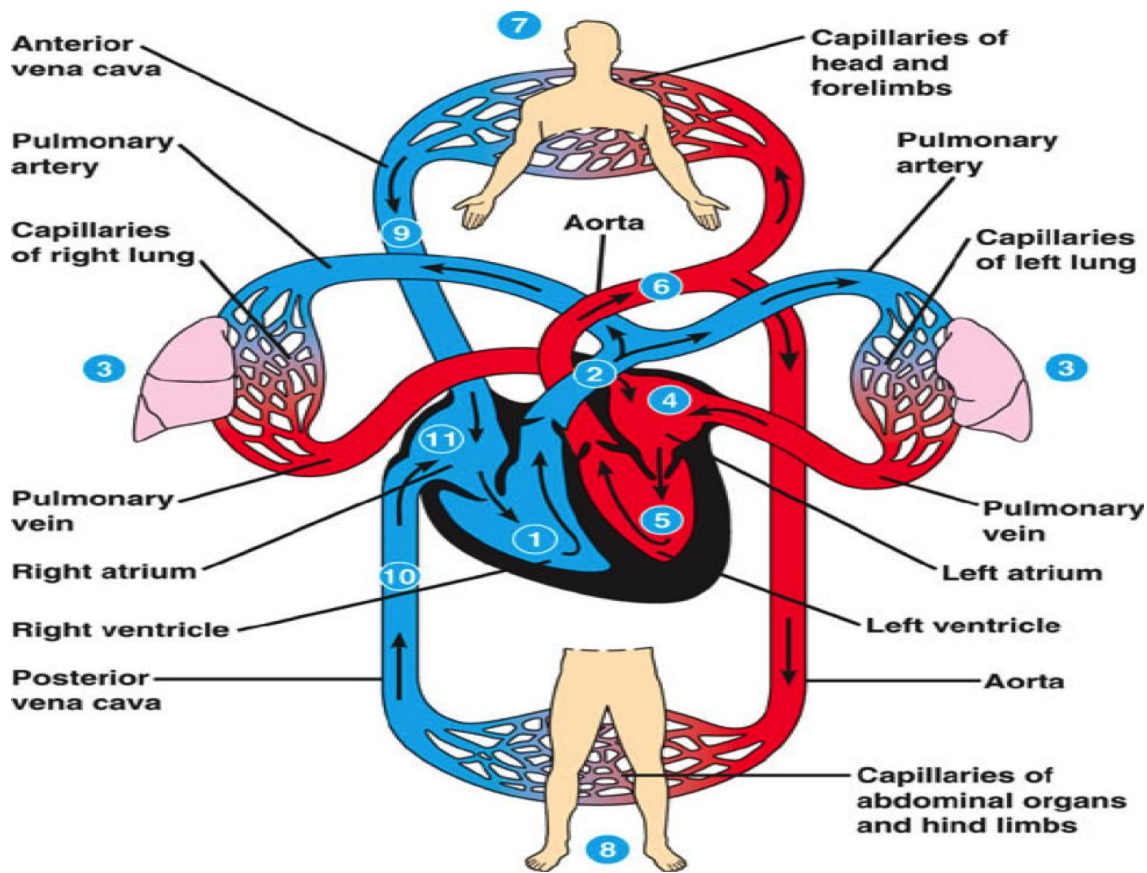


Fig 2.1 simplified illustration of the cardiovascular system

<https://share.google/x7RlySMO24bpMtU1q>

2.3 ELECTROCARDIOGRAM

An electrocardiogram (ECG) is a non-invasive diagnostic test that records the electrical activity of the heart over a period of time. It provides valuable information about cardiac rhythm, conduction abnormalities, and possible myocardial injury (Goldberger, 2017). The ECG is one of the most fundamental tools in cardiovascular medicine, used in both clinical and research settings to assess heart health (Bayés de Luna & Batchvarov, 2018). The heart's rhythmic contractions are initiated by electrical impulses generated by the sinoatrial (SA) node, which act as the natural pacemaker of the heart. These impulses spread through the atrioventricular (AV) node, bundle of His, bundle branches, and Purkinje fibers, producing depolarization and repolarization waves detectable on the skin surface (Hall & Hall, 2020). Electrodes placed on specific body sites detect voltage changes resulting from these electrical events, which are amplified and recorded as waves on the ECG tracing (Marieb & Hoehn, 2018).

Each ECG tracing consists of waves, intervals, and segments that correspond to different phases of the cardiac cycle:

P wave: Atrial depolarization (electrical activation of atria).

QRS complex: Ventricular depolarization (activation of ventricles).

T wave: Ventricular repolarization (recovery phase).

PR interval: Time between atrial and ventricular depolarization, reflecting AV nodal conduction.

ST segment: Early phase of ventricular repolarization, significant in ischemia detection.

QT interval: Total time for ventricular depolarization and repolarization (Macfarlane *et al.*, 2020).

ECG interpretation involves analyzing Heart rate and rhythm, Axis deviation, Presence of arrhythmias (e.g., atrial fibrillation, ventricular tachycardia), Evidence of ischemia or infarction, Conduction blocks (e.g., bundle branch block), It is essential for diagnosing myocardial infarction, electrolyte imbalances, drug toxicity, and conduction abnormalities (Goldberger, 2017).

2.4 EFFECTS OF ENERY DRINKS ON ECG READINGS

Energy drinks are beverages formulated to enhance alertness and physical performance, commonly containing caffeine, taurine, guarana, B-vitamins, ginseng, and sugars. While often marketed as performance enhancers, increasing research shows that they can influence cardiac electrophysiology, detectable through electrocardiogram (ECG) changes (Shah *et al.*, 2019; Cao *et al.*, 2021). These ECG

effects are primarily linked to the stimulatory actions of caffeine and other additives on the cardiovascular system.

One of the most documented ECG effects of energy drink consumption is QTc interval prolongation, which represents delayed ventricular repolarization. Prolongation of the QTc interval is clinically significant because it increases the risk of torsades de pointes and other potentially fatal ventricular arrhythmias (Shah *et al.*, 2019). In a randomized controlled trial by Shah *et al.* (2019), healthy young adults who consumed 946 mL of a commercial energy drink demonstrated a mean QTc prolongation of 10–20 ms compared to placebo, accompanied by an increase in systolic blood pressure. Similarly, Cao *et al.* (2021) in a systematic review found consistent evidence of QTc lengthening and elevated heart rate following acute consumption of energy drinks across several human studies.

Beyond QTc changes, sinus tachycardia and palpitations are frequently observed. These are reflected on ECG as increased heart rate or premature atrial and ventricular contractions (Hanif *et al.*, 2020). Case reports have described energy drink-induced arrhythmias, including supraventricular tachycardia and atrial fibrillation in otherwise healthy young individuals (Hanif *et al.*, 2020; Markon *et al.*, 2019). Ellermann *et al.* (2022) further demonstrated experimentally that combinations of caffeine and taurine altered ventricular action potential duration

and increased the likelihood of ventricular arrhythmias in isolated heart preparations. The physiological mechanisms underlying these ECG alterations involve sympathomimetic stimulation due to caffeine, which raises circulating catecholamines and enhances myocardial excitability and automaticity (Hall & Hall, 2020). While caffeine alone is known to increase heart rate and blood pressure, the synergistic combination with taurine, guarana, and other ingredients can modulate cardiac ion channels and repolarization currents, potentially amplifying QTc changes (Ellermann *et al.*, 2022). Excess sugar and ginseng content may further influence electrolyte balance and autonomic tone, indirectly affecting ECG readings (Cao *et al.*, 2021).

Clinically, these findings highlight the importance of monitoring ECG parameters in individuals who consume large volumes of energy drinks, particularly those with underlying cardiac disease, electrolyte abnormalities, or who are using QT-prolonging medications. A QTc increase of more than 30–60 ms after consumption, or a QTc exceeding 450 ms in men and 470 ms in women, should prompt further evaluation (Shah *et al.*, 2019). Physicians are encouraged to ask patients presenting with palpitations, syncope, or arrhythmias about their energy-drink intake (Cao *et al.*, 2021).

Although moderate consumption may be tolerated by healthy individuals, evidence suggests that excessive or high-volume intake especially over short periods can provoke transient or clinically relevant ECG changes (Ellermann *et al.*, 2022). Therefore, caution is advised, particularly among adolescents and young adults who often combine these drinks with alcohol or other stimulants.

2.5 LIPID PROFILE TESTS

A lipid profile, also known as a lipid panel, is a group of blood tests that measure the concentration of various lipids (fats) in the bloodstream. It is a fundamental diagnostic tool for assessing an individual's risk of developing cardiovascular diseases (CVDs) such as atherosclerosis, coronary artery disease, and stroke (Rifai *et al.*, 2018). The lipid profile evaluates the metabolism of fats in the body by quantifying lipids that are transported in the plasma as lipoproteins. Since lipids such as cholesterol and triglycerides are insoluble in water, they are carried by proteins in the form of lipoprotein complexes (Hall & Hall, 2020). Abnormalities in these components may indicate metabolic disorders like dyslipidemia or secondary causes such as diabetes, obesity, hypothyroidism, or liver disease (Grundy *et al.*, 2019).

A standard lipid profile includes the following measurements:

Total Cholesterol (TC): Represents the overall amount of cholesterol in the blood, including LDL, HDL, and VLDL fractions. Elevated levels (>200 mg/dL or >5.2 mmol/L) are associated with increased CVD risk (Rifai *et al.*, 2018).

Low-Density Lipoprotein Cholesterol (LDL-C): Often termed “bad cholesterol,” LDL carries cholesterol to tissues. High LDL-C promotes atheroma formation and arterial plaque buildup (Grundy *et al.*, 2019). Optimal values are typically <100 mg/dL.

High-Density Lipoprotein Cholesterol (HDL-C): Known as “good cholesterol,” HDL transports cholesterol from peripheral tissues back to the liver for excretion. Higher HDL-C (>60 mg/dL) is protective, whereas lower levels (<40 mg/dL in men and <50 mg/dL in women) increase CVD risk (Mach *et al.*, 2019).

Triglycerides (TG): The main form of stored fat in the body. Elevated triglycerides (>150 mg/dL) may indicate insulin resistance, obesity, or metabolic syndrome (Toth *et al.*, 2013).

Very Low-Density Lipoprotein (VLDL): Synthesized in the liver to transport endogenous triglycerides. It is often estimated as $VLDL = TG/5$ (in mg/dL) in fasting samples (Rifai *et al.*, 2018).

The lipid profile is vital in Screening for dyslipidemia and metabolic syndrome, Monitoring lipid-lowering therapy (e.g., statins), Assessing cardiovascular risk in both primary and secondary prevention (Mach *et al.*, 2019), Evaluating hereditary lipid disorders such as familial hypercholesterolemia. Lifestyle factors like diet, physical activity, smoking, and alcohol intake significantly affect lipid levels, and management includes lifestyle modification along with pharmacotherapy when necessary (Grundy *et al.*, 2019).

Parameter	Desirable/Optimal range	Boaderline high	Low risk
Total cholesterol	<200mg/dL (5.2 mmol/L)	200–239 mg/dL	≥240 mg/dL
LDL	<100mg/dL (2.6mmol/L)	130–159 mg/dL	≥160 mg/dL
HDL	≥60mg/L (1.6 mmol/L)	40–59 mg/dL	<40 mg/dL
Triglycerides	<150mg/dL (1.7 mmol/L)	150–199 mg/dL	≥200 mg/dL

Adapted from: (Grundy *et al.*, 2019).

2.6 ENERGY DRINK EFFECTS ON LIPID PROFILE TESTS

The lipid profile comprises total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Energy drinks can influence these parameters through several physiological mechanisms:

Most EDs contain 10–13 g of sugar per 100 mL. A standard 500 mL can may thus supply more than 50 g of sugar, mainly in the form of sucrose or glucose-fructose syrup (Reissig *et al.*, 2009). Excess sugar intake increases hepatic lipogenesis, resulting in elevated triglyceride and VLDL-cholesterol levels, while potentially reducing HDL-C (Te Morenga *et al.*, 2013). Caffeine stimulates sympathetic nervous activity, increasing circulating catecholamines (epinephrine and norepinephrine), which promote lipolysis and raise free fatty acids (FFAs) in plasma (Higgins *et al.*, 2018). Chronic elevations in FFAs can increase hepatic triglyceride synthesis and LDL production (Grasser *et al.*, 2014). Taurine, an amino sulfonic acid found in many EDs (about 1000 mg/100 mL), has been shown to have lipid-modulating properties, potentially reducing serum cholesterol and triglyceride levels (Zhang *et al.*, 2004). However, its effect may be overshadowed by the high sugar and caffeine content when consumed excessively. Niacin can beneficially increase HDL-C and decrease LDL-C and triglycerides, but the low

doses present in EDs (typically 8–20 mg per serving) are unlikely to exert significant lipid-lowering effects (Kujawa *et al.*, 2020).

Several studies have evaluated the effects of energy drinks on lipid profiles in both humans and animals:

Single-dose consumption of caffeinated EDs has minimal immediate effect on serum lipids, though transient increases in plasma FFAs and blood pressure may occur (Worthley *et al.*, 2010). Repeated intake over weeks or months is associated with elevated triglycerides, increased LDL-C, and reduced HDL-C, particularly when beverages are high in sugar (Basrai *et al.*, 2019). In a study on healthy adults consuming EDs daily for two weeks, serum triglycerides and total cholesterol increased significantly, while HDL-C decreased (Basrai *et al.*, 2019).

Long-term energy drink exposure in rats led to hyperlipidemia and hepatic fat accumulation, suggesting oxidative stress and hepatic dysfunction as underlying mechanisms (Elitok *et al.*, 2015; Al Dera *et al.*, 2017). Alterations in lipid profile due to chronic ED consumption may predispose individuals to atherosclerosis, coronary artery disease, and metabolic syndrome. Increased LDL-C and triglycerides, coupled with reduced HDL-C, accelerate lipid deposition in arterial walls and impair endothelial function (Mach *et al.*, 2019). Individuals with pre-existing dyslipidemia or insulin resistance may therefore face heightened

cardiovascular risk when consuming energy drinks regularly (Alsunni, 2015; Higgins *et al.*, 2018).

While occasional consumption of energy drinks may not significantly alter lipid metabolism, chronic or high-frequency intake particularly of sugar-sweetened formulations—can adversely affect serum lipid profiles. These effects arise from increased hepatic lipogenesis, catecholamine-induced lipid mobilization, and oxidative stress. Therefore, moderation and consumer awareness are critical to preventing lipid abnormalities and long-term cardiovascular complications.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 MATERIALS

Some of this materials include:

Energy Drink (Predator)

Lab coat

Hand gloves

Test tube/ test tube racks

EDTA

Syringes and needles

Sphygmomanometer

Weighing scale

Stadiometer

Lipid profiling (measurement of triglycerides, HDL and LDL)

Electrocardiogram

Ultrasound gel

Questionnaire

3.2 STUDY DESIGN

This study will involve human subject and be divided into two groups of young adults between the age of 17 to 25 studying at the School of Basic Medical Sciences that takes energy drinks frequently.

Group 1 will be the control group; that is the group that have not taken energy drink at all while The Test group will comprises young adults that takes energy drinks frequently and they will be given energy drinks (predator) to take for one week. The test group would comprise of 2 groups also.

Group 2 would be subjected to 1 bottle per day and group 3 to 2 bottles per day following ethical guidelines

Then blood samples would be taken at baseline after the two weeks of consumption

3.2.1 Energy drink content (predator)

NUTRITIONAL INFORMATION

	Per 100ml	Per 400ml
Energy	238KJ (56kcal)	653KJ
Carbohydrates	14g	56g
Of which sugar	13.5g	64g
Protein	0.04g	0.2g

Fats ,saturates, protein — negligible amount.

3.2.2 COMPOSITION INFORMATION

	Per 100ml	400ml
Vitamin B3	0.3mg (21%)	128mg (85%)
Vitamin B6	0.3mg (23%)	12mg (92%)
Caffine	30mg	120mg

3.3 INCLUSION CRITERIA

Must be within the ages of 17 to 25

Must be a student of University of Benin, Basic medical science

Regular consumer of Energy Drinks

Must not be underweight

3.4 EXCLUSION CRITERIA

Hypertension and Hypotension patients

Diabetic patients

Chronic alcoholic

People on Medications

3.5 ETHICAL CONSIDERATIONS

Prior to the commencement of the study, a well-structured questionnaire will be administered to each participant to gather essential health information, demographic data, and lifestyle patterns. Participants will be fully informed about the study's objectives, procedures, potential risks, and benefits. Informed consent will be obtained from all participants, indicating their voluntary agreement to take

part in the study. Confidentiality and anonymity of all responses will be strictly upheld throughout the research process.

3.6 ETHICAL APPROVAL.

Ethical approval for the study will be sought and obtained from the Ethics Committee of the College of Medical Sciences, University of Benin. This study would be carried out at the Physiology Laboratory in the University of Benin.

3.7 SAMPLE COLLECTION

For the lipid profile tests, After cleaning the overlying skin with cotton wool soaked in methylated spirit, blood samples of about 10mls were obtained with minimal trauma via the cubital fossa of the participants using a sterile disposable syringe and needle and placed into lithium heparin bottles after which the samples were centrifuged to obtain the plasma. The obtained sample were then taken to the laboratory after being labeled for the determination of Total Cholesterol levels, Triglycerides, HDL and LDL levels.

Before the commencement of ECG recording, the participant's skin at electrode placement sites was cleaned with cotton wool soaked in methylated spirit to reduce skin resistance Then ultrasound gel was applied. Disposable adhesive electrodes were then attached to the standard anatomical positions, one on each wrist and

ankle (limb leads), and six precordial electrodes on the chest (V₁–V₆). The electrodes were connected to the ECG machine using sterile lead wires. Participants were instructed to relax and remain still during the procedure to minimize movement artifacts. A resting 12-lead ECG was recorded for each participant while lying in a supine position. The recorded ECG tracings were then labeled with each participant's code and stored for further analysis of heart rate, rhythm, and electrical axis.

3.8 SAMPLE ANALYSIS

3.8.1 Lipid Profile Analysis

Biochemical analysis of lipid parameters was conducted using standard enzymatic colorimetric methods.

Total Cholesterol (TC): Determined using the cholesterol oxidase–peroxidase (CHOD–PAP) method.

Triglycerides (TG): Determined using the glycerol phosphate oxidase–peroxidase (GPO–PAP) method.

High-Density Lipoprotein (HDL): Measured after precipitation of low- and very-low-density lipoproteins using phosphotungstic acid.

Low-Density Lipoprotein (LDL): Calculated using the Friedewald formula:

$$\text{LDL} = \text{TC} - (\text{TG}/5 + \text{HDL})$$

Results were expressed in mg/dL and interpreted according to the World Health Organization (WHO, 2020) reference ranges.

3.8.2 Electrocardiogram (ECG) Analysis

The recorded ECG tracings were analyzed for rate, rhythm, and morphology of waveforms. Parameters such as P-wave, PR interval, QRS complex, QT interval, and T-wave were examined. The mean electrical axis of the heart was also determined from the limb leads.

Abnormal findings such as arrhythmias, conduction blocks, axis deviation, or ST-T segment changes were noted and interpreted according to standard clinical ECG criteria (as described by Guyton & Hall, 2021; Goldberger *et al.*, 2018).

3.9 STATISTICAL ANALYSIS

All the data obtained from the experiments were expressed as mean \pm Standard Error of Mean(SEM). Statistical analysis was performed by one-way analysis of variance (ANOVA) followed by a posthoc test for assessing differences amongst the means using Graph pad Prism 10.2.2 statistical software (Graphpad, San Diego, CA). 95% confidence limit was used to consider all significant values.

CHAPTER FOUR

4.0 RESULTS

4.1 RESULTS OF STATISTICAL ANALYSIS

4.1.1 For the lipid profile;

Lipid profile Parameters	Control group	Group 1	Group 2
TC	156.3	148.8	162
TG	110.7	103.7	108.7
HDL	46.8	44.7	48.7
LDL	92.4	83.8	91.5
VLDL	23.1	16.7	18.1

Table 4.1: Comparing the mean values of Total Cholesterol, Triglycerides, High density lipoprotein, Low density lipoprotein and very low density lipoprotein of Young adults in the University of Benin following regulated energy drink intake

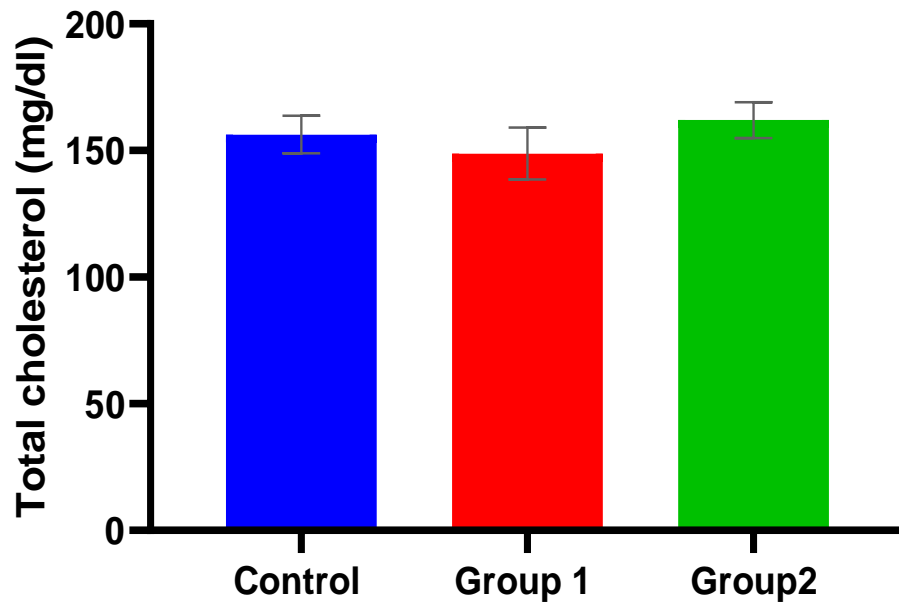


Figure 4.1: Showed total cholesterol concentration in young adults following consumption energy drink

There were no significant in group 1 and group 2 relative to control

*P < 0.05 indicates significant difference relative to control

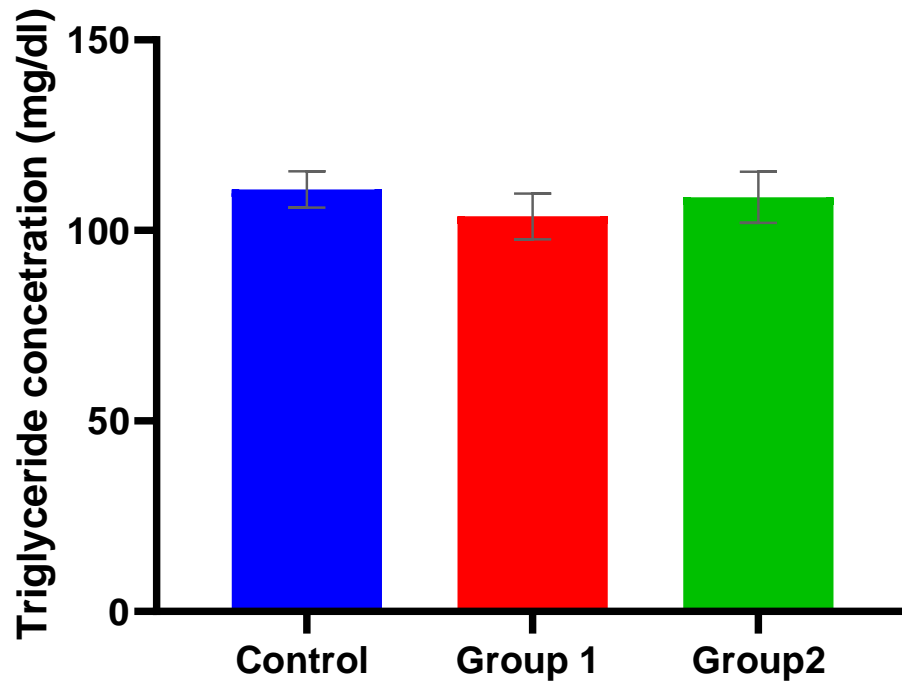


Figure 4.2: Showed triglyceride concentration in young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

*P < 0.05 indicates significant difference relative to control

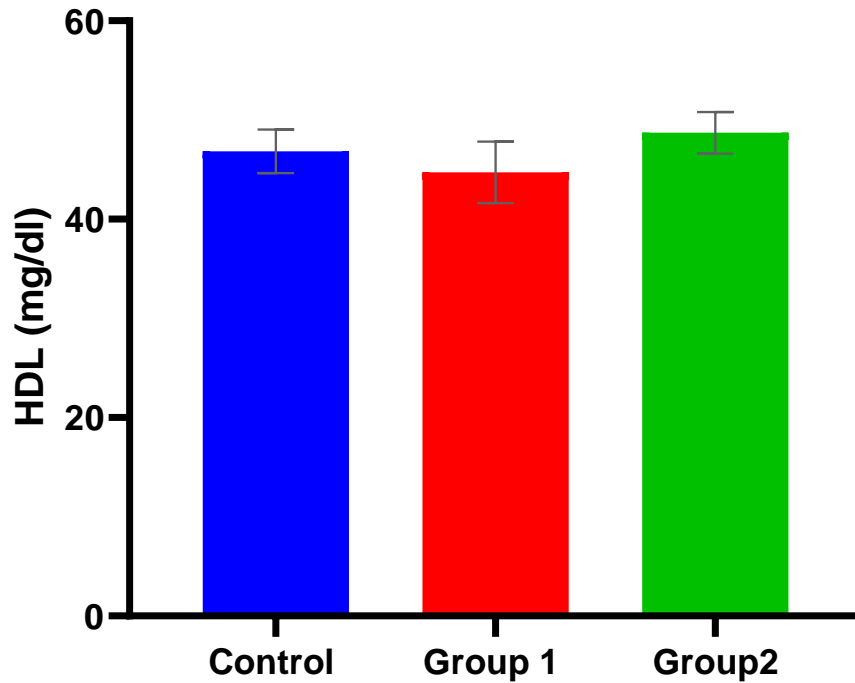


Figure 4.3: Showed HDL concentration in young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

*P < 0.05 indicates significant difference relative to control

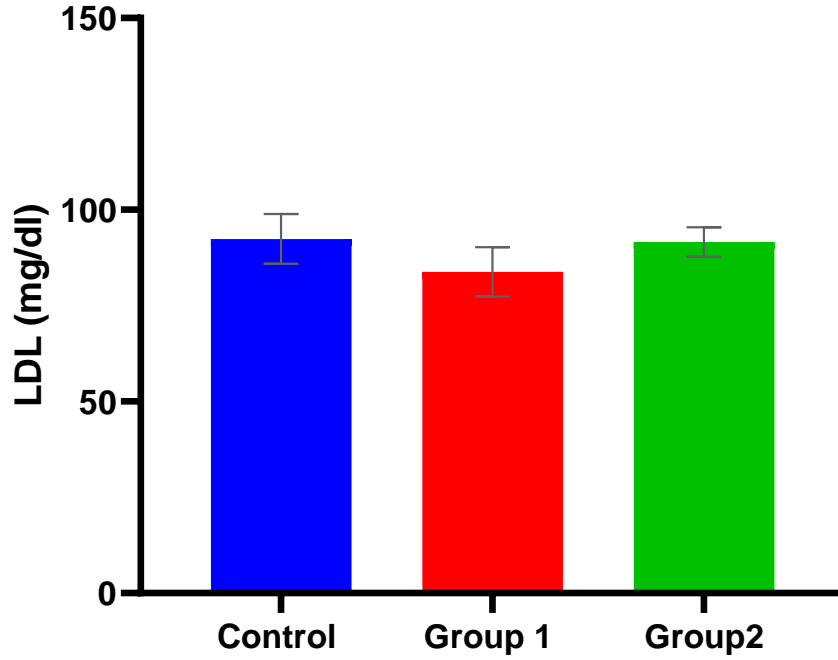


Figure 4.4: Showed LDL concentration in young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

*P < 0.05 indicates significant difference relative to control

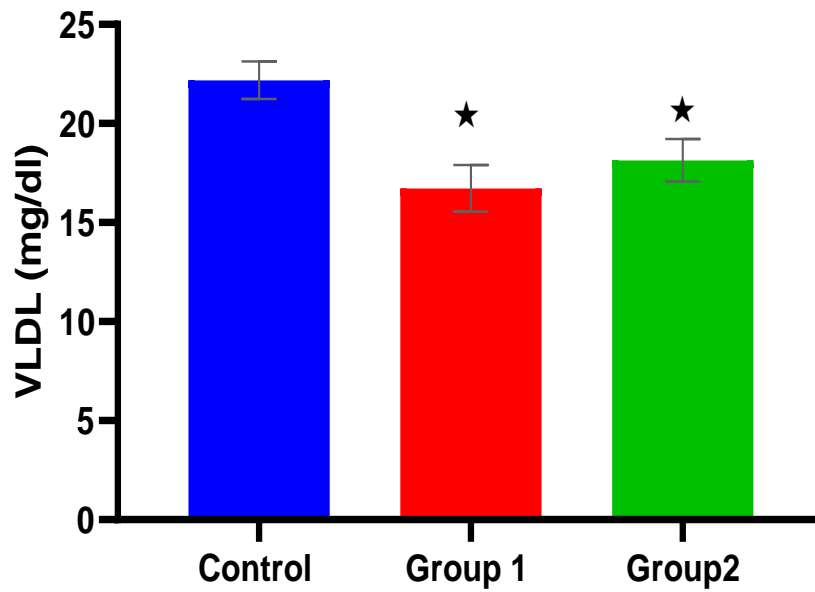


Figure 4.5: Showed VLDL concentration in young adults following consumption energy drink

There were significant decreases in group 1 and group 2 relative to control

*P < 0.05 indicates significant difference relative to control

4.1.2 For the ECG Parameters;

Parameters	Control Group	Group 1	Group 2
PR	151.0	156.0	162.0
QRS	83.4	85.6	92.4
QT	357.4	357.4	364.2
QTc	387.0	388.8	385.4
HR	77.4	81.2	89.2

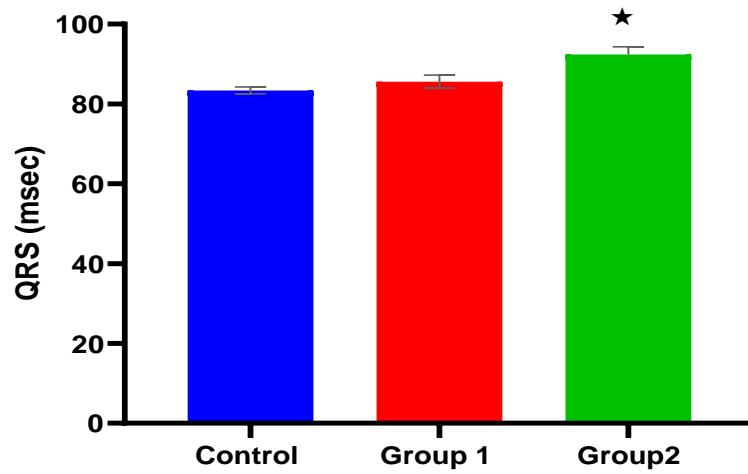


Figure 4.6: Showed QRS co in ECG reading in young adults following consumption energy drink

There was a significant increase in group 2 compared with control, but there was no significant difference in group 1 relative to control

**P < 0.05 indicates significant difference relative to control*

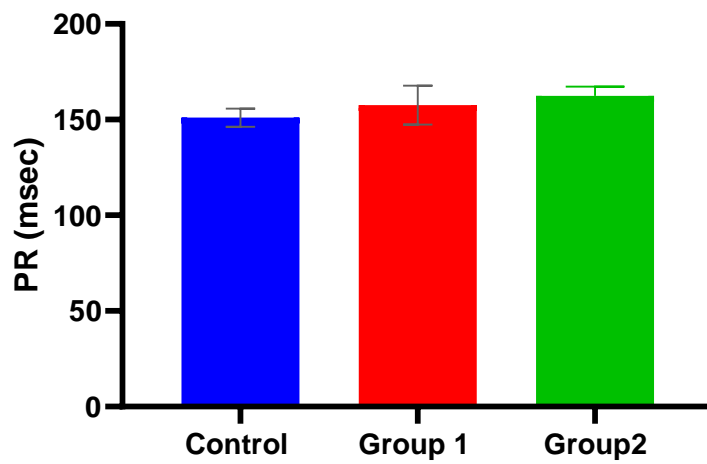


Figure : Showed PR interval in ECG reading of young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

**P < 0.05 indicates significant difference relative to control*

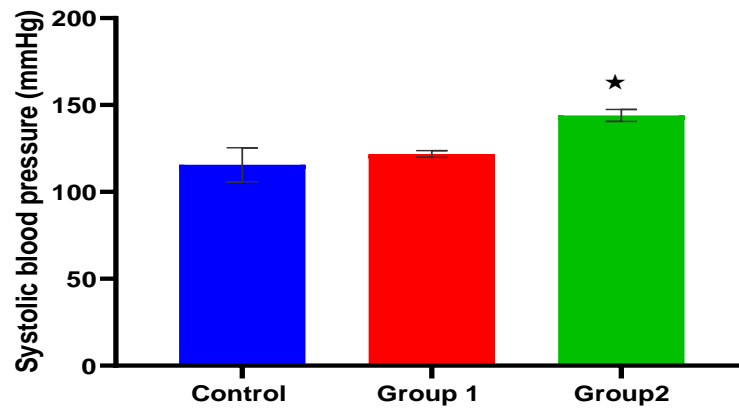


Figure : Showed SBP in young adults following consumption energy drink

There was a significant increase in group 2 compared with control, but there was no significant difference in group 1 relative to control

**P < 0.05 indicates significant difference relative to control*

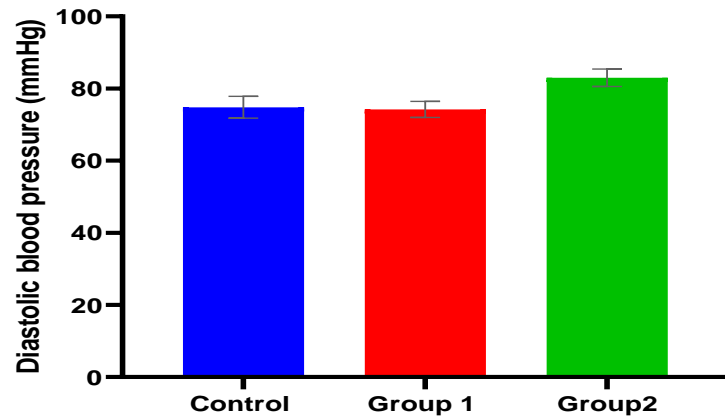


Figure : Showed DBP of young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

**P < 0.05 indicates significant difference relative to control*

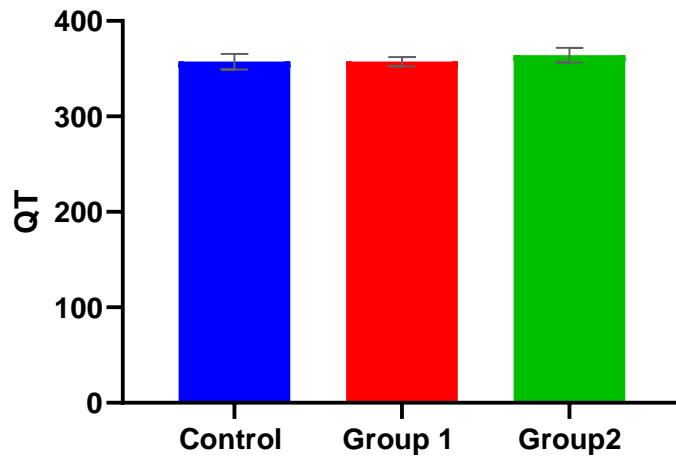


Figure : Showed QT interval in ECG reading of young adults following consumption energy drink

There were no significant differences in group 1 and group 2 relative to control

**P < 0.05 indicates significant difference relative to control*

CHAPTER FIVE

5.0 DISCUSSION AND CONCLUSION

5.1 DISCUSSION

Because of P values obtained were higher than the P values regarded as significant, the results obtained from this study were not statistically significant (Less than 0.05 was considered as statistically significant).

The findings indicate that short-term or moderate energy drink intake does not markedly alter lipid metabolism in young adults. This aligns with the work of Alhadi et al. (2023), who found that caffeine consumption which is a major component of most energy drinks does not significantly affect total cholesterol, HDL-C, or LDL-C levels in healthy individuals. Caffeine acts as a metabolic stimulant through adenosine receptor antagonism, enhancing catecholamine release and promoting lipolysis and fatty acid oxidation (Farias-Pereira *et al.*, 2019). While this can elevate free fatty acids in circulation, it does not necessarily translate into significant changes in serum lipid fractions under short-term exposure. The observed significant decrease in VLDL-C suggests a possible enhanced utilization of triglyceride-rich lipoproteins or reduced hepatic synthesis of VLDL. Taurine—another bioactive compound commonly found in energy drinks—has been shown to modulate lipid metabolism by increasing bile acid

conjugation and improving hepatic lipid clearance (Chen *et al.*, 2016). Experimental studies in animals have demonstrated that taurine supplementation can reduce hepatic triglyceride and cholesterol accumulation, which could contribute to the decline in VLDL-C (Park *et al.*, 1998). Therefore, the reduction in VLDL-C observed in this study may reflect a beneficial metabolic adaptation rather than an adverse lipid disturbance. The absence of changes in other lipid components reinforces that short-term exposure may not significantly impact lipoprotein balance in healthy young adults.

For the ECG, The findings from this study reveal that the consumption of energy drinks produced notable cardiovascular effects among young adults. Specifically, there was a significant increase in QRS complex duration and systolic blood pressure (SBP) in Group 2, while Group 1 showed no significant difference compared with the control. The PR and QT intervals, as well as diastolic blood pressure (DBP), remained statistically unchanged across groups. The significant prolongation of the QRS complex in Group 2 suggests delayed ventricular depolarization, which may be attributed to increased sympathetic stimulation caused by high caffeine and taurine levels in energy drinks (Higgins *et al.*, 2018). This finding aligns with previous reports that excessive caffeine intake can alter cardiac conduction and increase the risk of arrhythmogenic effects (Shah *et al.*,

2016). Similarly, the elevated SBP in Group 2 corroborates earlier studies indicating that energy drinks acutely raise blood pressure through heightened catecholamine release, increased myocardial contractility, and vascular resistance (Grasser *et al.*, 2015; Worthley *et al.*, 2010). Although DBP did not significantly change, the rise in SBP suggests a transient hypertensive effect, consistent with the sympathetic activation observed after caffeine ingestion (Steinke *et al.*, 2009). The absence of significant differences in PR and QT intervals implies that moderate consumption may not exert adverse effects on atrioventricular conduction or ventricular repolarization. However, higher doses, as seen in Group 2, might predispose individuals to conduction abnormalities or arrhythmias with prolonged use (Higgins *et al.*, 2018).

5.2 CONCLUSION

This study demonstrates that energy drink consumption, particularly at higher doses, can significantly increase QRS duration and systolic blood pressure in young adults, indicating a potential cardiovascular risk when consumed excessively. Moderate intake did not produce significant ECG or blood pressure alterations, suggesting a dose-dependent effect. It is therefore recommended that young adults limit energy drink consumption, especially before physical activity or in individuals with underlying cardiovascular risk factors. Energy drink

consumption among young adults, at the tested quantities, does not significantly alter serum lipid parameters or blood pressure, except for a notable reduction in VLDL-C. These results suggest that short-term consumption is metabolically tolerable, though long-term studies are required to determine potential cumulative effects, especially in individuals with metabolic or cardiovascular risk factors.

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