International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	PATHOPHYSIOLOGICAL MECHANISMS AND CLINICAL EVIDENCE OF CARDIAC ARRHYTHMIAS ASSOCIATED WITH ENERGY DRINK CONSUMPTION: A COMPREHENSIVE SYSTEMATIC REVIEW
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5580
RECEIVED	14 March 2026
ACCEPTED	18 June 2026
PUBLISHED	24 June 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

© The author(s) 2026.
This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

PATHOPHYSIOLOGICAL MECHANISMS AND CLINICAL EVIDENCE OF CARDIAC ARRHYTHMIAS ASSOCIATED WITH ENERGY DRINK CONSUMPTION: A COMPREHENSIVE SYSTEMATIC REVIEW

Paulina Michalska *(Corresponding Author, Email: pmichalska000@gmail.com)*
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0007-4141-5906

Zuzanna Przybylska
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0001-3329-4602

Aleksandra Anna Purpura
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0006-3893-7730

Wiktor Rolski
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0005-1439-4842

Bartłomiej Przystaś
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0009-6121-3839

Mikołaj Piotr Hobot
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0001-2829-6005

Gabriela Patrycja Dobosz
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0009-4012-9101

Sebastian Podeszwa
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0000-2973-5324

Magdalena Grzechowiak
Medical University of Silesia, 15 Poniatowskiego Street, 40-055 Katowice, Poland
ORCID ID: 0009-0001-8486-0352

Paweł Michalski
SP ZOZ Miejski Szpital Zespolony w Częstochowie, Częstochowa, Poland
ORCID ID: 0009-0002-8935-7171

ABSTRACT

Background: Energy drinks (EDs) are widely consumed beverages containing high concentrations of caffeine, taurine, and stimulants. Popular among adolescents and athletes, EDs may exert significant cardiovascular effects, including autonomic and electrophysiological alterations that increase susceptibility to arrhythmias.
Aim: This review synthesized evidence on mechanisms and clinical findings associated with cardiac arrhythmias triggered by ED consumption.
Materials and Methods: A search was conducted via PubMed, MDPI, and Google Scholar (2014 onwards). Keywords included "energy drinks", "caffeine", "ECG", and "cardiac arrhythmia". Analysis focused on human studies evaluating cardiovascular and autonomic effects of ED intake.
Results: ED consumption induces acute hemodynamic and electrophysiological changes, such as increased heart rate, elevated blood pressure, QTc interval prolongation, and altered heart rate variability. These effects are mediated by caffeine-induced adenosine receptor antagonism, catecholamine release, and enhanced myocardial excitability. Additional ingredients, like taurine and sugars, influence ion channel activity and autonomic balance, promoting sympathetic predominance. Supraventricular and ventricular arrhythmias are temporally associated with ED intake, particularly in individuals with underlying cardiovascular disease or genetic syndromes.
Conclusions: EDs promote arrhythmogenic conditions through autonomic, hemodynamic, and electrophysiological mechanisms. While occasional intake in healthy individuals is unlikely to cause significant arrhythmias, excessive consumption in susceptible populations increases risk. Further studies are needed to define safe consumption thresholds.

KEYWORDS

Energy Drinks, Cardiac Arrhythmias, Caffeine, QT Interval, Autonomic Nervous System, Heart Rate Variability

CITATION

Paulina Michalska, Zuzanna Przybylska, Aleksandra Anna Purpura, Wiktor Rolski, Bartłomiej Przystaś, Mikołaj Piotr Hobot, Gabriela Patrycja Dobosz, Sebastian Podeszwa, Magdalena Grzechowiak, Paweł Michalski. (2026) Pathophysiological Mechanisms and Clinical Evidence of Cardiac Arrhythmias Associated with Energy Drink Consumption: A Comprehensive Systematic Review. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5580

COPYRIGHT

© The author(s) 2026. This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

1. Introduction

Over the past two decades, the consumption of energy drinks has increased markedly, particularly among adolescents, young adults, and university students who frequently use these beverages to enhance perceived physical and cognitive performance. Energy drinks typically contain high concentrations of caffeine, taurine, sugars, and other stimulatory compounds that influence several physiological systems, particularly the cardiovascular and autonomic nervous systems. While moderate caffeine intake is generally regarded as safe for healthy adults, energy drinks can induce acute hemodynamic changes, including elevated heart rate, blood pressure, and alterations in electrocardiographic (ECG) parameters, raising concerns regarding their potential arrhythmogenic effects [**1**].

Several observational studies and case reports have linked energy drink consumption to adverse cardiovascular events, including palpitations, supraventricular tachycardia, and new-onset atrial fibrillation, even in otherwise healthy individuals [**2**]. The combination of caffeine with other bioactive compounds such as taurine may potentiate sympathetic nervous system activity, alter cardiac ion channel function, and disrupt autonomic regulation, thereby increasing susceptibility to rhythm disturbances. Furthermore, the co-ingestion of alcohol with energy drinks (AmED) may further exacerbate cardiovascular stress and arrhythmia risk, particularly among young adults [**3**].

Experimental studies have demonstrated that acute ingestion of energy drinks can lead to transient increases in heart rate and blood pressure, as well as changes in ECG intervals such as QT and QTc prolongation, highlighting their electrophysiological impact [**4**]. Comparative studies suggest that some effects of energy drinks may exceed those of an equivalent dose of caffeine from coffee, emphasizing the role of

additional ingredients and synergistic effects [**5**]. Despite widespread consumption, the relationship between energy drink intake and clinically significant arrhythmias remains incompletely understood, with limited large-scale, controlled trials available [**6**].

This review aims to synthesize current knowledge on the cardiovascular and electrophysiological effects of energy drink consumption, elucidate the underlying mechanisms of arrhythmogenesis, and critically evaluate clinical evidence linking these beverages to cardiac rhythm disturbances [**7**].

Research Objective

The primary objective of this systematic review is to comprehensively evaluate the current evidence on the relationship between energy drink consumption and the occurrence of cardiac arrhythmias. The study aims to identify and synthesize both pathophysiological mechanisms and clinical findings that explain how components of energy drinks—particularly caffeine, taurine, and sugars—affect cardiac electrophysiology, autonomic regulation, and arrhythmogenesis.

Research Problems

This review addresses the following research problems:

1. What are the underlying pathophysiological mechanisms linking energy drink consumption with cardiac arrhythmias?
2. Does energy drink intake lead to measurable electrophysiological changes (e.g., QT interval prolongation, heart rate variability alterations)?
3. Are energy drinks associated with clinically significant arrhythmias in healthy individuals?
4. Which populations are at the highest risk of arrhythmia after energy drink consumption?
5. Do components other than caffeine contribute to the arrhythmogenic potential of energy drinks?
6. Is there evidence of dose-dependent or synergistic effects (e.g., with alcohol or other stimulants)?

Research Hypothesis

It is hypothesized that energy drink consumption contributes to the development of cardiac arrhythmias through combined autonomic, hemodynamic, and electrophysiological mechanisms. Specifically, the intake of energy drinks is expected to increase sympathetic activity, alter cardiac ion channel function, and prolong repolarization parameters, thereby increasing susceptibility to arrhythmias, particularly in individuals with underlying cardiovascular conditions or high consumption levels.

2. Research materials and methods

2.1 Search strategy

A comprehensive literature review was conducted using the PubMed, MDPI, and Google Scholar databases. The search included keywords such as “energy drink(s)”, “caffeine”, “taurine”, “sugar”, “electrocardiogram”, “ECG”, “heart rate variability”, “arrhythmia”, “cardiac arrhythmia”, and “acute effects”

The analysis focused on studies exploring the base of cardiac arrhythmias triggered by energy-drink intake. The search was restricted to clinical and interventional human studies published from 2014 onward.

2.2 Eligibility criteria

The eligibility criteria were defined prior to the literature search to ensure consistency and methodological rigor.

Inclusion criteria:

- Clinical studies (randomized controlled trials, crossover studies, observational studies) conducted in human subjects
- Studies evaluating the effects of energy drinks or their components (e.g., caffeine, taurine, sugars) on cardiovascular or electrophysiological parameters
- Studies assessing outcomes such as heart rate, blood pressure, electrocardiographic parameters (e.g., QT/QTc interval), heart rate variability, or occurrence of arrhythmias
- Articles published in peer-reviewed journals
- Publications in English
- Studies published from 2014 onward

Exclusion criteria:

- Animal studies and in vitro studies
- Review articles, editorials, commentaries, and conference abstracts without original data
- Studies not directly related to cardiovascular or electrophysiological outcomes
- Studies focusing solely on cognitive or metabolic effects without cardiac assessment
- Duplicate publications or studies with insufficient methodological quality

2.3 Data collection and analysis

2.3.1 Study selection process

The study selection process was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure transparency and methodological rigor. In the first stage, records identified through database searches were screened by titles and abstracts. Subsequently, full texts of potentially eligible articles were assessed for compliance with predefined inclusion and exclusion criteria. To minimize selection bias, the process was performed independently by multiple reviewers, with any discrepancies resolved through consensus or consultation with a third reviewer. This structured approach ensured the reproducibility and reliability of the systematic review.

3. Research results

3.1 Mechanisms of Arrhythmogenesis

Caffeine: Adenosine Receptor Blockade, Catecholamine Release, and Increased Myocardial Excitability

Caffeine is a primary bioactive component of energy drinks and acts predominantly through non-selective antagonism of adenosine A_1 and A_2A receptors, leading to disinhibition of central and peripheral sympathetic activity. This mechanism results in increased catecholamine release, elevation of heart rate, and enhanced myocardial excitability, which together create a pro-arrhythmic substrate.

Data from experimental and human physiological studies indicate that adenosine receptor blockade reduces the anti-arrhythmic, rate-slowing effects of endogenous adenosine on sinoatrial and atrioventricular nodal tissue. The analyzed study demonstrated that caffeine exposure increases intracellular cyclic AMP and calcium influx, thereby facilitating positive chronotropic and inotropic effects, which may lower the threshold for triggered activity and re-entry phenomena [**8**]. These effects are particularly relevant in individuals with underlying channelopathies or heightened sympathetic tone.

Clinical evidence further supports these mechanisms. An interventional study comparing habitual and non-habitual caffeine consumers showed that acute energy drink ingestion led to significant increases in heart rate and systolic blood pressure, accompanied by electrophysiological alterations, including changes in repolarization parameters. Importantly, these effects were more pronounced in non-habitual caffeine consumers, indicating variability in autonomic and myocardial responsiveness [**9**].

Collectively, the available evidence suggests that caffeine-induced adenosine receptor antagonism and increased catecholamine release may contribute to arrhythmogenesis through tachycardia, increased myocardial excitability, and altered cardiac repolarization.

Taurine: Ion Channel Modulation and Interaction with Caffeine

Taurine is a sulfur-containing amino acid commonly added to energy drinks in concentrations exceeding typical dietary intake. Experimental and clinical data suggest that taurine modulates cardiac ion channels, particularly calcium and potassium currents, and influences intracellular calcium handling.

Exposure assessment data indicate that taurine intake from energy drinks can reach pharmacologically active levels, especially when multiple servings are consumed in a short time period [**10**]. While taurine alone has been associated with membrane-stabilizing and potentially anti-arrhythmic properties, its effects are context-dependent and may differ when combined with other stimulants.

A controlled clinical study evaluating the isolated and combined effects of caffeine and taurine demonstrated that caffeine exerted dominant effects on cardiovascular and autonomic parameters, whereas taurine modified these responses when co-administered. The combined administration resulted in alterations of heart rate variability indices, suggesting that taurine may modulate but not fully counteract caffeine-induced sympathetic activation [**11**].

These findings imply that, within energy drink formulations, taurine may interact with caffeine at the level of ion channel modulation and autonomic regulation, potentially enhancing myocardial excitability under conditions of sympathetic predominance, rather than exerting a purely protective effect.

Sugars and Other Stimulants: Glycemic Fluctuations and Autonomic Modulation

Energy drinks typically contain high concentrations of rapidly absorbable sugars, which can induce acute glycemic excursions. Such rapid changes in blood glucose have been shown to exert measurable effects on the cardiovascular autonomic nervous system.

A double-blind randomized crossover trial demonstrated that acute ingestion of glucose or fructose led to significant reductions in heart rate variability parameters, including SDNN and RMSSD, compared with placebo. These changes indicate a shift toward sympathetic dominance and reduced parasympathetic

modulation, even in healthy individuals [**12**]. The study concluded that rapid carbohydrate availability acts as a metabolic stressor capable of altering cardiac autonomic control.

Complementary findings were reported in a controlled intervention evaluating sugar-sweetened soft drinks, which showed acute reductions in spontaneous baroreflex sensitivity and HRV following sugar ingestion. The impairment of cardiovagal baroreflex function suggests diminished reflex buffering of heart rate responses, a recognized mechanism increasing susceptibility to arrhythmias [**13**].

Additional evidence from a study involving a hypertonic sugar-sweetened sports beverage demonstrated that sugar ingestion blunted the expected parasympathetic activation observed after water consumption. This resulted in sustained sympathetic activity and altered hemodynamic responses, further supporting the role of sugar-induced autonomic imbalance [**14**].

Together, these studies indicate that sugars contribute to arrhythmogenic risk by inducing acute autonomic dysregulation through glycemic fluctuations, particularly when combined with stimulant compounds.

Autonomic Nervous System Effects: Sympathetic Activation and Arrhythmia Risk

One of the key mechanisms explaining the arrhythmogenic potential of energy drinks appears to involve their effects on autonomic nervous system regulation, characterized predominantly by enhanced sympathetic activity and delayed parasympathetic recovery.

A randomized, crossover, double-blind trial evaluating post-exercise recovery demonstrated that energy drink consumption significantly altered autonomic recovery dynamics. Specifically, heart rate variability indices remained suppressed during recovery, indicating prolonged sympathetic predominance compared with placebo [**15**]. These effects were observed regardless of baseline cardiorespiratory fitness, suggesting a direct pharmacological influence on autonomic regulation.

Further evidence from an exercise-based interventional study showed that energy drink ingestion modified HRV patterns both during exertion and in the immediate recovery phase. The persistence of autonomic imbalance during recovery represents a critical window during which arrhythmias are more likely to occur, particularly in susceptible individuals [**16**].

In addition, studies assessing the combined use of energy drinks with alcohol demonstrated impaired psychophysiological regulation and heightened autonomic stress responses, indirectly supporting increased cardiovascular risk under combined stimulant exposure [**17**].

Overall, the available clinical evidence indicates that sympathetic overactivation, reduced vagal tone, impaired baroreflex sensitivity, and delayed autonomic recovery constitute key mechanistic pathways linking energy drink components to arrhythmogenesis.

3.2 Role of Technology in Energy Drink Formulation and Exposure

The development and widespread availability of energy drinks are closely linked to advances in food and beverage technology, which enable the precise design of products with targeted physiological effects. Contemporary energy drinks are formulated using technologically optimized combinations of caffeine, taurine, sugars, and additional bioactive compounds, allowing manufacturers to achieve specific stimulant profiles and sensory characteristics. These formulations often result in concentrations of active substances that exceed those found in traditional dietary sources, particularly when consumed in large volumes or in rapid succession.

Exposure assessment studies have demonstrated that the intake of caffeine, taurine, and other constituents from energy drinks can reach pharmacologically significant levels, raising concerns about dose-dependent physiological effects [**10**]. Importantly, the technological design of these beverages influences not only the composition but also the absorption kinetics and bioavailability of their components, which may contribute to variability in individual responses.

Evidence from randomized controlled trials further suggests that the cardiovascular effects of energy drinks are shaped by formulation-dependent factors rather than by caffeine alone. Studies comparing energy drinks with caffeine-matched controls have reported greater electrophysiological changes, including QTc prolongation, as well as sustained increases in blood pressure following energy drink consumption [**18,19].** These findings indicate that the technological complexity of these products, including ingredient interactions and delivery profiles, plays a critical role in their overall biological impact.

Taken together, these observations highlight that energy drinks should be understood as technologically engineered consumable products with clinically relevant cardiovascular effects. This perspective is essential for accurately assessing their health risks and for informing both regulatory strategies and public health interventions.

3.3 Acute cardiovascular effects

Acute ingestion of commercial energy drinks produces reproducible hemodynamic and electrophysiological changes that have been documented across randomized trials and experimental studies. Several controlled trials show that consumption of large volumes of energy drinks is associated with measurable increases in blood pressure and changes in electrocardiographic intervals, and case literature documents acute arrhythmic events temporally related to intake.

In a randomized, double-blind, placebo-controlled crossover study of healthy volunteers, two different commercial energy drinks produced statistically significant prolongation of the corrected QT interval (QTc) and increases in peripheral and central systolic and diastolic blood pressures compared with placebo; peak changes in Bazett-corrected QT (QTcB) were reported as +17.9 ± 13.9 ms and +19.6 ± 15.8 ms for the two commercial drinks versus +11.9 ± 11.1 ms for placebo (ANOVA $P = 0.005$; pairwise $P = 0.04$ and <0.01 vs placebo), and peripheral systolic and diastolic blood pressures were significantly elevated over the observation period [**18**]. Similarly, a separate randomized trial that directly compared a high-volume energy drink (946 mL, 320 mg caffeine) with a caffeine-matched control found that the baseline-adjusted change in QTc at 2 hours was 0.44 ± 18.4 ms after the energy drink versus −10.4 ± 14.8 ms after caffeine alone ($P = 0.02$), whereas peripheral systolic blood pressure remained significantly higher at 6 hours after the energy drink (mean difference ≈ 3.9–4.7 mmHg depending on time point) [**19**]. These randomized data indicate that components of energy drinks beyond caffeine may contribute to transient QTc prolongation and a sustained late systolic pressor effect.

Pediatric data confirm that electrophysiological perturbations are not limited to adults. In a randomized, double-blind crossover trial in healthy children and adolescents (mean age ≈14.5 years), ingestion of an age-adjusted energy drink dose produced a significantly increased number of supraventricular extrasystoles (SVES) compared with placebo; no sustained supraventricular tachycardia or malignant ventricular arrhythmias were observed, and mean heart rate was paradoxically lower after the energy drink in this cohort, indicating age-specific autonomic and electrophysiological responses [**20**]. These findings demonstrate that acute arrhythmic triggers (increased ectopy) may be induced even in a young, ostensibly healthy population.

Case reports and clinical series further document acute symptomatic arrhythmias following energy drink consumption. Reports include new-onset palpitations progressing to supraventricular tachycardia, documented episodes of paroxysmal atrial fibrillation in previously healthy individuals, and isolated instances of ventricular arrhythmia temporally associated with heavy intake; while such events are uncommon in randomized cohorts, their occurrence in real-world settings underscores clinically relevant risk in susceptible persons [**18**,**21**].

Population- and mechanism-oriented studies indicate that the arrhythmogenic potential of energy drinks depends not only on dose but also on formulation and co-ingestion practices. A clinical trial assessing the interaction of energy drinks with alcohol showed that concomitant intake increased ethanol plasma concentration by ≈14.8% and caffeine plasma concentration by ≈17.6%, and that participants were more willing to drive after the combined ingestion — a behavioral effect that may potentiate exposure and risk in young adults [**15**]. In an LQTS (familial long QT syndrome) cohort, energy drink ingestion did not produce a mean QTc prolongation >20 ms across the group, but produced statistically significant increases in systolic and diastolic blood pressure with peak changes expressed as approximately 6% **vs** 0.8% (systolic) and 11% vs 3% (diastolic) when comparing energy drink versus control, indicating that patients with repolarization disorders can manifest exaggerated hemodynamic responses that may theoretically modify arrhythmic risk [**21**].

Comparative randomized data underscore the difference between single-ingredient caffeine exposure and complex energy-drink formulations. The high-volume energy drink versus caffeine trial reported that while both interventions raised systolic blood pressure acutely, only the energy drink produced transient QTc prolongation at the 2-hour mark and a late increase in systolic pressure at 6 hours, effects not seen with caffeine alone [**19**]. Likewise, another randomized study using two commercial drinks versus placebo found larger absolute QTc changes with the drinks (up to ≈+20 ms) and consistently higher peripheral and central blood pressures compared with placebo [**18**]. These findings support a model in which non-caffeine constituents (taurine, herbal extracts, sugar,/or other additives) and formulation-dependent pharmacokinetics produce additive or synergistic electrophysiological and hemodynamic effects.

3.4 Chronic Consumption

Evidence addressing the long-term effects of habitual energy drink (ED) consumption on cardiac electrophysiology is very limited. While controlled clinical trials have largely examined acute responses to single doses of EDs, only a small number of longitudinal and observational investigations have evaluated habitual ED intake over extended periods, and none directly measure chronic impacts on *cardiac arrhythmogenesis* or autonomic function in adults. Two notable studies provide insight into broader health associations over time.

In a longitudinal cohort of Swedish adolescents (n=1,622), consumption of energy drinks was prevalent, with 74% of boys and 54% of girls reportedly having consumed EDs at baseline ($P < 0.001$) [**22**]. Importantly, ED consumption was not a benign behavior; it was positively associated with markers of worse health and risk-related behaviors concurrently and one year later. Adjusted logistic regression models showed that those reporting ED consumption in 2010 had significantly higher odds of school-related stress (OR 1.53; 95% CI 1.07–2.20) and gaming-related truancy (OR 4.88; 95% CI 2.28–10.43) in 2011, with stronger effects generally observed for those with the highest levels of intake [**22**]. Although this study did not directly assess cardiovascular parameters such as heart rate variability or arrhythmias, the observed predictive relationship between ED consumption and poorer health outcomes suggests that habitual use may co-occur with physiological and behavioral risk factors relevant to cardiovascular health.

The EDKAR study (European Journal of Epidemiology, 2025) specifically investigated adolescents aged 15–18 with chronic high ED consumption, defined as ED intake on ≥4 days per week for ≥12 months with >3 mg caffeine/kg bodyweight/day, compared with non-consumers [**23**]. This study found no statistically or clinically significant differences in resting heart rate, systolic or diastolic blood pressure, electrocardiographic intervals (PQ, QTc, QRS), or echocardiographic measures between high consumers (n=97) and controls (n=160) after adjustment for confounders such as age, sex, BMI, smoking, and alcohol use [**23**]. Despite this, approximately 49.5% of chronic high consumers reported subjective adverse effects, including palpitations and chest pressure following ED consumption [**23**]. The authors emphasize that although direct cardiac markers did not differ between groups, the clustering of chronic high ED consumption with other lifestyle risk factors (e.g., shorter sleep duration: 53.6% vs. 12.7% in controls; higher smoking and alcohol use) could indirectly influence long-term cardiovascular health [**23**]. Furthermore, the observational design and focus on adolescents limit causal inference and generalizability to adults or susceptible populations.

Collectively, these studies illustrate significant gaps in the literature: very few longitudinal investigations extend beyond adolescence, and none provide robust evidence linking chronic ED consumption to arrhythmias, sustained changes in HRV, or autonomic dysregulation in adults or clinical populations. The Swedish cohort highlights that habitual ED intake is associated with worse general health outcomes and risk behaviors over time, which are themselves established risk factors for cardiovascular morbidity, while the EDKAR study suggests that, even with heavy long-term use, overt cardiologic changes may not be detectable in otherwise healthy adolescents [**22**,**23**]. The dearth of long-term clinical studies that systematically quantify electrical heart rhythm dynamics under conditions of chronic ED exposure, particularly in individuals with genetic predispositions or pre-existing cardiac disease, severely constrains our ability to assess arrhythmogenic risk in habitual consumers and underscores the need for targeted longitudinal research in this area.

3.5 Populations at risk

Young Adults, Students, and Recreational Athletes

Young adults, particularly university students and recreational athletes, represent a population with high prevalence of energy drink (ED) consumption and relative lack of cardiovascular screening. Experimental studies in healthy young individuals consistently demonstrate measurable electrophysiological and hemodynamic effects following ED intake. A controlled trial involving medical students ($n = 68$, mean age 22 ± 2 years), reported a significant increase in heart rate (+5.7%, $P < 0.01$) and systolic blood pressure (+6.4%, $P < 0.001$) within 60 minutes after consumption of a single ED containing 80 mg caffeine, taurine, and glucuronolactone, compared with baseline [**24**]. Although no sustained arrhythmias were recorded, the authors observed significant QTc prolongation in 11.8% of participants, highlighting a potential proarrhythmic substrate [**24**].

Another experimental study in healthy young adults (n= 44), showed that ED consumption resulted in a statistically significant increase in QTc interval by a mean of 10.5 ± 8.7 ms ($P = 0.003$), with 18% of participants exceeding a QTc increase ≥30 ms, a threshold associated with elevated arrhythmic risk [**1**]. These findings were

corroborated by a randomized trial using a high-volume ED protocol, in which QTc prolongation >50 ms in 2% of participants and QTc prolongation >30 ms in 11%, persisting up to 4 hours post-consumption [**18**].

In younger populations, a randomized trial in healthy children and adolescents (n = 27) led to a significant reduction in left ventricular efficiency by 10.2% (P = 0.01) and an increase in myocardial workload, reflecting altered cardiac energetics that may predispose to electrical instability during exertion [**25**]. Collectively, these findings indicate that even in ostensibly healthy young populations, EDs induce acute cardiovascular changes that may facilitate supraventricular or ventricular arrhythmias, particularly during physical activity.

Individuals with Congenital Long QT Syndrome, Cardiomyopathy, or Atrial Fibrillation

Patients with underlying cardiac disease represent a particularly vulnerable subgroup. A randomized crossover study of individuals with familial Long QT syndrome (LQTS; n = 24), demonstrated that ED consumption resulted in QTc prolongation ≥50 ms in 33% of participants, compared with none during placebo exposure [**21**]. The mean QTc increase following ED ingestion was 17 ± 14 ms (P < 0.01), underscoring a clinically meaningful repolarization disturbance in this genetically susceptible population.

More severe clinical consequences were reported in a recent multicenter retrospective analysis of sudden cardiac arrest survivors (n = 144), which found that 7 cases (4.9%) occurred in temporal proximity (<24 h) to ED consumption [**26**]. Notably, 57% of these cases had an underlying genetic arrhythmia syndrome, including LQTS, catecholaminergic polymorphic ventricular tachycardia, or idiopathic ventricular fibrillation [**26**]. These findings suggest that EDs may act as arrhythmic triggers rather than primary causes, precipitating malignant ventricular arrhythmias in predisposed individuals.

Although direct evidence in patients with cardiomyopathies or atrial fibrillation remains limited, the observed QTc prolongation, sympathetic activation, and increased myocardial workload provide plausible mechanistic pathways through which EDs may exacerbate arrhythmogenic risk in these populations.

Interactions with Medications (e.g., Stimulants, Beta-Blockers)

Pharmacological interactions represent an additional and often underrecognized risk factor for ED-induced arrhythmias. A comprehensive review, reported that caffeine and other sympathomimetic components of EDs may antagonize the effects of beta-blockers, reducing their heart rate–lowering efficacy and potentially facilitating tachyarrhythmias [**27**]. Furthermore, EDs may exhibit synergistic effects with prescription stimulants, such as amphetamines or methylphenidate, leading to exaggerated increases in catecholaminergic tone and myocardial excitability [**27**].

The review also highlighted that up to 30% of ED consumers reported concurrent use of medications or drugs of abuse, increasing the likelihood of clinically relevant interactions [**27**]. While direct arrhythmic endpoints were not systematically assessed, the mechanistic evidence strongly suggests that such interactions may amplify arrhythmogenic risk, particularly in patients with pre-existing cardiovascular disease or those receiving antiarrhythmic therapy.

3.6 Recommendations and Safety Considerations

Safe Caffeine Intake Limits and Energy Drink Consumption

Current safety recommendations regarding caffeine intake are primarily based on regulatory assessments and clinical evidence evaluating cardiovascular outcomes. The European Food Safety Authority (EFSA) concluded that single doses of caffeine up to 200 mg and habitual daily intake up to 400 mg are not associated with clinically significant adverse cardiovascular effects in healthy adults [**28**] (EFSA Scientific Opinion; used here to define safety thresholds). In contrast, lower limits are recommended for vulnerable populations, including adolescents, pregnant women, and individuals with cardiovascular disease or arrhythmia susceptibility [**28**].

The U.S. Food and Drug Administration (FDA) has not established formal maximum limits specifically for energy drinks; however, it recognizes approximately 400 mg/day of caffeine as a generally safe upper intake level for healthy adults, while explicitly warning against highly concentrated caffeine products and cumulative exposure from multiple sources (FDA guidance; used here to highlight regulatory caution).

Importantly, randomized controlled trials (RCTs) suggest that energy drinks may exert electrophysiological effects that are not solely attributable to caffeine content. A double-blind crossover RCT demonstrated that consumption of a high-volume energy drink resulted in significantly greater QTc prolongation compared with an equivalent dose of caffeine alone, indicating potential additive or synergistic effects of other ingredients (e.g., taurine, guarana) [**19**]. Similar findings were reported ina trial that observed acute changes in QTc interval and blood pressure following energy drink ingestion in healthy volunteers [**18**].

Taken together, these findings support the recommendation that safe caffeine intake limits derived from regulatory authorities should be interpreted cautiously when applied to energy drinks, particularly when consumed in large volumes or in short time intervals.

3.7 ECG and Heart Rate Variability Monitoring in At-Risk Individuals

Growing clinical evidence supports the use of electrocardiographic (ECG) and autonomic monitoring in individuals who consume energy drinks regularly or belong to populations at increased arrhythmic risk. Acute interventional studies have demonstrated that energy drink ingestion can influence cardiac repolarization and autonomic balance, even in healthy individuals.

A randomized crossover design, reported QTc prolongation exceeding 10 ms following energy drink consumption, a threshold that is considered clinically relevant in proarrhythmic risk assessment [**19**]. Another trial further confirmed measurable ECG and hemodynamic changes after acute intake of energy drinks, reinforcing concerns regarding subclinical electrophysiological effects [**18**]

Heart rate variability (HRV) analysis provides additional insight into autonomic nervous system modulation. demonstrated that acute energy drink consumption impaired post-exercise autonomic recovery, as evidenced by altered HRV indices in a randomized, placebo-controlled crossover study [**11**].

Based on available evidence, ECG and/or 24-hour Holter monitoring should be considered in individuals with known or suspected channelopathies (e.g., long QT syndrome), prior unexplained syncope, a history of cardiac arrest, or regular high-volume energy drink consumption. HRV analysis may serve as an adjunctive tool to detect autonomic dysregulation in frequent consumers.

3.8 Education on the Risks of Combining Energy Drinks with Alcohol or Supplements

The concomitant use of energy drinks with alcohol (AmED) or stimulant-containing supplements represents a significant safety concern. While caffeine may reduce subjective perceptions of alcohol-induced sedation, it does not mitigate alcohol-related psychomotor impairment, thereby promoting higher alcohol intake and increased cardiovascular stress.

Clinical and experimental studies indicate that AmED consumption is associated with greater alcohol intake, increased risk-taking behaviors, and heightened cardiovascular strain, compared with alcohol alone [**29**]. Additionally, the widespread availability of dietary supplements containing caffeine, synephrine, or other sympathomimetic compounds increases the risk of unintentional caffeine overdose and additive proarrhythmic effects. Regulatory agencies explicitly warn against cumulative stimulant exposure from multiple products

Therefore, educational interventions should emphasize the avoidance of combining energy drinks with alcohol or stimulant supplements, particularly in adolescents, young adults, and individuals with cardiovascular risk factors.

4. Discussion

This systematic review synthesized available evidence regarding the pathophysiological mechanisms and clinical implications of cardiac arrhythmias associated with energy drink consumption. The analyzed studies suggest that energy drinks may induce acute cardiovascular and electrophysiological changes, including increases in heart rate and blood pressure, alterations in autonomic regulation, and disturbances in cardiac repolarization. Together, these physiological changes may increase susceptibility to arrhythmias, particularly in individuals with underlying cardiovascular vulnerability.

Several experimental studies have demonstrated that energy drink intake produces measurable hemodynamic responses even in healthy populations. Increased heart rate and blood pressure following consumption have been reported in controlled experimental studies, indicating acute sympathetic activation and cardiovascular stimulation [**1,4**]. Comparable findings have been observed in studies comparing energy drinks with coffee or placebo beverages, which demonstrated significant effects on heart rate, blood pressure, and ventricular repolarization parameters [**5,6**]. Additionally, research evaluating habitual and non-habitual caffeine consumers has shown that energy drink intake may lead to measurable changes in both hemodynamic and electrophysiological parameters [**9**].

The arrhythmogenic potential of energy drinks is primarily attributed to their high caffeine content and the presence of additional bioactive compounds. Caffeine acts as an adenosine receptor antagonist and increases catecholamine release, leading to enhanced sympathetic activity, increased myocardial excitability, and elevated heart rate [**8**]. These mechanisms may contribute to disturbances in cardiac electrophysiology and predispose individuals to arrhythmias. At the same time, caffeine is widely recognized for its cognitive and

physical performance-enhancing properties, which partly explains the popularity of energy drinks among students and athletes [**7**]. In addition to caffeine, energy drinks frequently contain other active ingredients such as taurine and D-glucuronolactone, which may interact with caffeine and influence cardiovascular function [**10**]. Experimental studies suggest that the combination of caffeine and taurine may alter cardiovascular regulatory mechanisms, potentially affecting autonomic balance and electrophysiological stability [**11**].

Autonomic nervous system alterations represent another mechanism potentially contributing to arrhythmogenesis. Several studies have demonstrated that consumption of glucose- and fructose-containing beverages can affect heart rate variability and autonomic cardiovascular regulation in healthy individuals [**12**]. Similarly, sugar-sweetened beverages have been shown to decrease baroreflex sensitivity and reduce heart rate variability, indicating impaired autonomic control of cardiovascular function [**13**]. Comparable responses have been observed after consumption of hypertonic sugar-sweetened sports beverages, suggesting that high sugar content may contribute to cardiovascular dysregulation [**14**]. Studies examining energy drink consumption have also demonstrated changes in autonomic recovery and heart rate variability following intake, indicating a shift toward sympathetic predominance and reduced parasympathetic modulation [**15,16**].

Electrophysiological effects, particularly disturbances in cardiac repolarization, may further increase arrhythmia risk. Randomized clinical trials have demonstrated that high-volume energy drink consumption can significantly prolong the QTc interval and increase blood pressure compared with control beverages [**18**]. Additionally, studies comparing energy drinks with caffeine alone have reported greater electrocardiographic effects with energy drinks, suggesting potential synergistic interactions between caffeine and other ingredients [**19**]. These electrophysiological changes have also been observed in younger populations. Randomized trials in healthy children and adolescents have shown measurable alterations in heart rhythm and electrocardiographic intervals following energy drink consumption [**20**]. Moreover, experimental studies have reported decreased left ventricular efficiency after energy drink intake in pediatric populations, suggesting potential adverse effects on cardiac performance [**25**]. Individuals with pre-existing cardiac conditions may be particularly vulnerable to these effects. In patients with familial long QT syndrome, energy drink consumption has been associated with significant cardiovascular responses, indicating an increased susceptibility to arrhythmogenic effects in genetically predisposed individuals [**21**]. The findings of the EDKAR study require careful interpretation. While resting ECG and echocardiographic parameters remained stable in chronic consumers, this does not eliminate the risk during physical exertion or acute high-volume intake. The high prevalence of subjective symptoms, such as palpitations and chest pressure reported by frequent consumers, suggests that energy drinks may trigger paroxysmal arrhythmias or subclinical electrophysiological changes not captured by static, resting examinations [**23**].

Evidence from observational studies and clinical reports further supports a possible association between energy drink consumption and adverse cardiovascular outcomes. Reviews of published case reports have documented various cardiovascular events occurring after energy drink ingestion, including atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias [**2**]. More recent investigations have also described cases of sudden cardiac arrest occurring in temporal proximity to energy drink consumption, highlighting the potential severity of these adverse effects in susceptible individuals [**26**].

Behavioral patterns related to energy drink consumption may additionally increase cardiovascular risk. The combination of energy drinks with alcohol is particularly common among university students and young adults [**3**]. Alcohol mixed with energy drinks may mask the sedative effects of alcohol while maintaining stimulant properties, potentially leading to increased alcohol consumption and greater physiological stress [**29**]. Furthermore, energy drinks may interact with certain prescription medications or drugs of abuse, potentially amplifying stimulant effects or increasing cardiovascular risk through pharmacodynamic interactions [**27**].

From a public health perspective, the growing popularity of energy drinks raises concerns regarding their potential cardiovascular effects. Although moderate caffeine consumption is generally considered safe for healthy adults, excessive intake may lead to undesirable cardiovascular effects in sensitive individuals [**28**]. Adolescents and young adults represent a major consumer group, and longitudinal studies have suggested associations between frequent energy drink consumption and adverse health behaviors or increased cardiovascular risk factors in younger populations [**22**].

Several limitations should be considered when interpreting the available evidence. Many studies included relatively small sample sizes and were conducted in controlled experimental settings involving healthy individuals, which may limit the generalizability of the findings. Additionally, considerable heterogeneity exists in energy drink formulations, caffeine concentrations, and consumption volumes across

studies. Most research has also focused primarily on acute physiological effects, while long-term cardiovascular consequences of chronic energy drink consumption remain insufficiently investigated.

In conclusion, current evidence suggests that energy drink consumption may induce acute hemodynamic, autonomic, and electrophysiological changes that could increase the risk of cardiac arrhythmias. While occasional consumption in healthy individuals may not necessarily lead to clinically significant arrhythmias, excessive intake or consumption in individuals with underlying cardiovascular conditions may pose a greater risk. Further large-scale prospective studies are needed to better understand the long-term cardiovascular effects of energy drink consumption and to identify populations at increased risk.

5. Conclusions

Energy drinks should not be regarded as harmless beverages in the context of cardiac rhythm disturbances. Their complex composition, combining caffeine, sugars, and other bioactive compounds, can acutely influence cardiac electrophysiology and autonomic regulation, thereby creating conditions that may facilitate the initiation of arrhythmias. The arrhythmogenic risk associated with energy drink consumption is not uniform and appears to be dose-dependent, modulated by individual susceptibility, and amplified by coexisting factors such as underlying cardiac disease, autonomic imbalance, physical exertion, or concurrent use of alcohol and other stimulants. While many healthy individuals may tolerate occasional intake without clinically apparent consequences, susceptible populations may experience subclinical or overt rhythm disturbances even after acute exposure. Current evidence underscores significant gaps in knowledge, particularly regarding long-term effects, cumulative exposure, and interactions between individual ingredients. Consequently, large-scale randomized clinical trials and well-designed prospective cohort studies are needed to better define risk thresholds, identify vulnerable subgroups, and inform evidence-based recommendations for safe consumption. These findings also highlight important public health implications, emphasizing the need for increased awareness, targeted education, and regulatory considerations regarding energy drink consumption.

Author Contributions: Conceptualization, P.M., W.R. P.M. and Z.P.; Methodology, A.P., S.P. and B.P.; Software, P.M., P.M.; Validation, All authors; Formal Analysis, P.M., S.P., M.G. and M.H.; Investigation, All authors; Resources, All authors; Data Curation, A.P., P.M. and M.H.; Writing – Original Draft Preparation, P.M., A.P. and G.D.; Writing – Review & Editing, P.M., W.R., P.M. and G.D.; Visualization, P.M. and B.P.; Supervision, P.M.; Project Administration, P.M.

All authors have read and agreed with the published version of the manuscript.

Funding: This research was conducted without any specific funding from public, commercial, or nonprofit organizations.

Data Availability Statement: All data analyzed during this study are included in this published article and are available from the cited literature.

Conflict of interest: The authors declare that Paulina Michalska and Paweł Michalski are related (daughter and father). This relationship did not influence the study design, data analysis, or interpretation of results.

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

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

In preparing this work, the authorsused ChatGPT (OpenAI) for the purpose of improving language and readability as well as translating specificterms. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES

1. Hajsadeghi, S., Mohammadpour, F., Manteghi, M. J., Kordshakeri, K., Tokazebani, M., Rahmani, E., & Hassanzadeh, M. (2016). Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: An experimental study on healthy young adults. *Anatolian Journal of Cardiology, 16*(2), 94–99. https://doi.org/10.5152/akd.2015.5930
2. Goldfarb, M., Tellier, C., & Thanassoulis, G. (2014). Review of published cases of adverse cardiovascular events after ingestion of energy drinks. *American Journal of Cardiology, 113*(1), 168–172. https://doi.org/10.1016/j.amjcard.2013.08.058
3. De Giorgi, A., Valeriani, F., Gallè, F., Ubaldi, F., Bargellini, A., Napoli, C., Liguori, G., Romano Spica, V., Vitali, M., & Protano, C. (2022). Alcohol mixed with energy drinks (AmED) use among university students: A systematic review and meta-analysis. *Nutrients, 14*(23), Article 4985. https://doi.org/10.3390/nu14234985
4. Kozik, T. M., Shah, S., Bhattacharyya, M., Franklin, T. T., Connolly, T. F., Chien, W., Charos, G. S., & Pelter, M. M. (2016). Cardiovascular responses to energy drinks in a healthy population: The C-energy study. *American Journal of Emergency Medicine, 34*(7), 1205–1209. https://doi.org/10.1016/j.ajem.2016.02.068
5. Brothers, R. M., Christmas, K. M., Patik, J. C., & Bhella, P. S. (2017). Heart rate, blood pressure and repolarization effects of an energy drink as compared to coffee. *Clinical Physiology and Functional Imaging, 37*(6), 675–681. https://doi.org/10.1111/cpf.12357
6. Phan, J. K., & Shah, S. A. (2014). Effect of caffeinated versus noncaffeinated energy drinks on central blood pressures. *Pharmacotherapy, 34*(6), 555–560. https://doi.org/10.1002/phar.1419
7. Lorenzo Calvo, J., Fei, X., Domínguez, R., & Pareja-Galeano, H. (2021). Caffeine and cognitive functions in sports: A systematic review and meta-analysis. *Nutrients, 13*(3), Article 868. https://doi.org/10.3390/nu13030868
8. Cappelletti, S., Piacentino, D., Sani, G., & Aromatario, M. (2015). Caffeine: Cognitive and physical performance enhancer or psychoactive drug? *Current Neuropharmacology, 13*(1), 71–88. https://doi.org/10.2174/1570159X13666141210215655
9. Çalışkan, S. G., & Bilgin, M. D. (2021). Acute effects of energy drink on hemodynamic and electrophysiologic parameters in habitual and non-habitual caffeine consumers. *Clinical Nutrition ESPEN, 42*, 333–338. https://doi.org/10.1016/j.clnesp.2021.01.011
10. Rubio, C., Cámara, M., Giner, R. M., González-Muñoz, M. J., López-García, E., Morales, F. J., Moreno-Arribas, M. V., Portillo, M. P., & Bethencourt, E. (2022). Caffeine, D-glucuronolactone and taurine content in energy drinks: Exposure and risk assessment. *Nutrients, 14*(23), Article 5103. https://doi.org/10.3390/nu14235103
11. Çalışkan, Ş. G. (2024). Acute effects of caffeine and taurine on linear and nonlinear measures of the cardiovascular system in young adults. *Applied Sciences, 14*(11), Article 4912. https://doi.org/10.3390/app14114912
12. Eckstein, M. L., Brockfeld, A., Haupt, S., et al. (2022). Acute changes in heart rate variability to glucose and fructose supplementation in healthy individuals: A double-blind randomized crossover placebo-controlled trial. *Biology, 11*(2), Article 338. https://doi.org/10.3390/biology11020338
13. Chapman, C. L., Reed, E. L., Worley, M. L., et al. (2021). Sugar-sweetened soft drink consumption acutely decreases spontaneous baroreflex sensitivity and heart rate variability. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 320*(5), R641–R652. https://doi.org/10.1152/ajpregu.00310.2020
14. Christiani, M., Grosicki, G. J., & Flatt, A. A. (2021). Cardiac-autonomic and hemodynamic responses to a hypertonic, sugar-sweetened sports beverage in physically active men. *Applied Physiology, Nutrition, and Metabolism, 46*(10), 1189–1195. https://doi.org/10.1139/apnm-2021-0138
15. Porto, A. A., Gonzaga, L. A., Benjamim, C. J. R., et al. (2022). Acute effects of energy drink on autonomic and cardiovascular parameters recovery in individuals with different cardiorespiratory fitness: A randomized, crossover, double-blind and placebo-controlled trial. *Arquivos Brasileiros de Cardiologia, 119*(4), 553–561. https://doi.org/10.36660/abc.20210625
16. Clark, N. W., Herring, C. H., Goldstein, E. R., et al. (2020). Heart rate variability behavior during exercise and short-term recovery following energy drink consumption in men and women. *Nutrients, 12*(8), Article 2372. https://doi.org/10.3390/nu12082372
17. Pérez-Mañá, C., Mateus, J. A., Díaz-Pellicer, P., et al. (2022). Effects of mixing energy drinks with alcohol on driving-related skills. *International Journal of Neuropsychopharmacology, 25*(1), 13–25. https://doi.org/10.1093/ijnp/pyab051
18. Shah, S. A., Szeto, A. H., Farewell, R., et al. (2019). Impact of high volume energy drink consumption on electrocardiographic and blood pressure parameters: A randomized trial. *Journal of the American Heart Association, 8*(11), Article e011318. https://doi.org/10.1161/JAHA.118.011318
19. Fletcher, E. A., Lacey, C. S., Aaron, M., et al. (2017). Randomized controlled trial of high-volume energy drink versus caffeine consumption on ECG and hemodynamic parameters. *Journal of the American Heart Association, 6*(5), Article e004448. https://doi.org/10.1161/JAHA.116.004448

20. Mandilaras, G., Li, P., Dalla-Pozza, R., et al. (2022). Energy drinks and their acute effects on heart rhythm and electrocardiographic time intervals in healthy children and teenagers: A randomized trial. *Cells, 11*(3), Article 498. https://doi.org/10.3390/cells11030498
21. Gray, B., Ingles, J., Medi, C., et al. (2017). Cardiovascular effects of energy drinks in familial long QT syndrome: A randomized cross-over study. *International Journal of Cardiology, 231*, 150–154. https://doi.org/10.1016/j.ijcard.2016.12.019
22. Svensson, Å., Warne, M., & Gillander Gådin, K. (2021). Longitudinal associations between energy drink consumption, health, and norm-breaking behavior among Swedish adolescents. *Frontiers in Public Health, 9*, Article 597613. https://doi.org/10.3389/fpubh.2021.597613
23. Menzel, J., Spinka, F., Pie, M. J., et al. (2025). Chronic high consumption of energy drinks and cardiovascular risk in adolescents—results of the EDKAR-study. *European Journal of Epidemiology, 40*, 1355–1368. https://doi.org/10.1007/s10654-025-01292-z
24. García, A., Romero, C., Arroyave, C., et al. (2017). Acute effects of energy drinks in medical students. *European Journal of Nutrition, 56*(6), 2081–2091. https://doi.org/10.1007/s00394-016-1246-5
25. Oberhoffer, F. S., Li, P., Jakob, A., et al. (2022). Energy drinks decrease left ventricular efficiency in healthy children and teenagers: A randomized trial. *Sensors, 22*(19), Article 7209. https://doi.org/10.3390/s22197209
26. Martinez, K. A., Bains, S., Neves, R., et al. (2024). Sudden cardiac arrest occurring in temporal proximity to consumption of energy drinks. *Heart Rhythm, 21*(7), 1083–1088. https://doi.org/10.1016/j.hrthm.2024.02.018
27. Hladun, O., Papaseit, E., Martín, S., et al. (2021). Interaction of energy drinks with prescription medication and drugs of abuse. *Pharmaceutics, 13*(10), Article 1532. https://doi.org/10.3390/pharmaceutics13101532
28. EFSA Panel on Dietetic Products and Nutrition and Allergies. (2015). Scientific opinion on the safety of caffeine. *EFSA Journal*. https://doi.org/10.2903/j.efsa.2015.4102
29. Verster, J. C., Benson, S., Johnson, S. J., et al. (2018). Alcohol mixed with energy drink (AMED): A critical review and meta-analysis. *Human Psychopharmacology, 33*(2), Article e2650. https://doi.org/10.1002/hup.2650