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Energy Drinks as a Risk Factor for Systemic Disorders: The Role of Caffeine and Taurine - A Literature Review

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Abstract

Background. Energy drinks are widely consumed products containing caffeine, taurine, sugars, and other biologically active compounds. Their increasing popularity, particularly among adolescents and young adults, has raised concerns regarding potential adverse systemic health effects.

Aim. The aim of this literature review was to summarize current evidence regarding the potential effects of energy drink consumption, with particular emphasis on the role of caffeine and taurine in the cardiovascular and central nervous systems.

Material and methods. This narrative literature review was based on publications retrieved from scientific databases and current review literature concerning the systemic effects of energy drinks and their major biologically active constituents, particularly caffeine and taurine. The available evidence was analyzed thematically with emphasis on cardiovascular and neuropsychiatric outcomes.

Results. Available evidence indicates that energy drink consumption may be associated with transient increases in blood pressure and heart rate, electrocardiographic alterations including QTc prolongation, and case reports of tachyarrhythmias and acute cardiovascular events. In the central nervous system, consumption has been associated with sleep disturbances, anxiety symptoms, psychomotor agitation, and-particularly in susceptible individuals-potential triggering of seizures and exacerbation of psychiatric symptoms. Current data also suggest associations between frequent consumption and problematic behavioral patterns resembling substance use disorders, although much of the available evidence remains observational.

Conclusions. The current body of evidence suggests that energy drinks possess clinically relevant systemic effects involving both the cardiovascular and central nervous systems. Although causal relationships remain difficult to establish in many cases, the available data support cautious

consumption, particularly among children, adolescents, and individuals with pre-existing medical or psychiatric vulnerabilities.

Key words: energy drinks, caffeine, taurine, cardiovascular system, central nervous system, adverse effects

1. Introduction

Energy drinks (EDs) are widely consumed beverages containing caffeine, taurine, sugars, and other biologically active substances, including guarana, vitamins, and herbal additives. Originally marketed as products intended to enhance alertness, physical performance, and concentration, they have become increasingly popular worldwide, particularly among adolescents and young adults [3, 20]. Their widespread availability and frequent consumption in educational, recreational, and sports settings have raised growing concerns regarding potential adverse health effects associated with acute and chronic exposure to high doses of stimulatory compounds.

Among the ingredients present in energy drinks, caffeine remains the principal psychoactive component responsible for many of their physiological effects. Taurine, another commonly added constituent, possesses neuromodulatory and metabolic properties and may interact with caffeine and other ingredients through complex biological pathways [3, 20, 21]. Although moderate caffeine intake is generally considered safe in healthy adults, the combination of caffeine with taurine and other biologically active compounds contained in energy drinks has become an area of increasing scientific and clinical interest.

Over the last decade, a growing body of experimental, epidemiological, and clinical evidence has suggested potential associations between energy drink consumption and adverse effects involving multiple organ systems. Particular attention has focused on the cardiovascular and central nervous systems, where reported effects include hemodynamic alterations, electrocardiographic changes, arrhythmias, sleep disturbances, anxiety symptoms, and other neuropsychiatric manifestations. However, the available evidence remains heterogeneous, and causal relationships are frequently difficult to establish due to the predominance of observational studies and case-based reports.

The aim of this narrative literature review was to summarize and critically discuss current evidence regarding the systemic effects of energy drink consumption, with particular emphasis on the cardiovascular and central nervous systems and the potential roles of caffeine and taurine.

2. Research Materials and Methods

2.1. Literature Sources and Search Strategy

This study was conducted as a narrative literature review. The literature search was performed using PubMed/MEDLINE, Scopus, and Google Scholar databases to identify English-language scientific publications concerning the systemic effects of energy drink consumption and the biological activity of their major constituents, particularly caffeine and taurine.

The search primarily covered literature published between January 2015 and January 2026. Earlier seminal publications were additionally included when considered essential for mechanistic, physiological, or neurobiological background.

The search strategy included combinations of the following keywords: “energy drinks”, “caffeine”, “taurine”, “cardiovascular system”, “blood pressure”, “heart rate”, “QTc”, “arrhythmia”, “central nervous system”, “sleep”, “anxiety”, “seizures”, “psychiatric symptoms”, and “energy drink use disorder”.

2.2. Study Selection and Evidence Synthesis

The review included English-language publications of clinical, observational, experimental, and review character, as well as selected case reports and case series relevant to the investigated topic. Priority was given to studies demonstrating direct clinical or mechanistic relevance to the cardiovascular and central nervous systems.

Duplicate publications, non-English studies, reports considered unreliable, and papers demonstrating poor thematic relevance were excluded. The collected evidence was analyzed narratively and synthesized thematically according to major organ-system effects and biological mechanisms discussed in the literature.

2.3. AI-Assisted Language and Editorial Support

Artificial intelligence tools were used exclusively for linguistic and editorial assistance during manuscript preparation. Their use included academic English refinement, grammatical and syntactic verification, stylistic editing, and consistency assessment of translated scientific text.

No artificial intelligence tools were used for literature screening, study selection, data extraction, evidence synthesis, interpretation of findings, or formulation of scientific conclusions. All final interpretations, assessments of scientific evidence, and conclusions were performed exclusively by the authors under human supervision.

3. Research results

3.1. Cardiovascular System

3.1.1. Hemodynamic Effects: Blood Pressure and Heart Rate

Numerous clinical studies have demonstrated that acute energy drink consumption may cause a transient increase in blood pressure in healthy young individuals, whereas its effect on heart rate is less consistent and appears to depend on the studied population, dose, and study design [1, 2, 3]. A systematic review including participants aged ≥13 years (37 studies, 1,597 individuals) demonstrated an increase in heart rate in approximately 61% of participants, while elevations in systolic and diastolic blood pressure (SBP and DBP) were observed in more than 50% of the analyzed studies following the consumption of standard servings of energy drinks [4].

Dobrek (2025) indicated that, in healthy adults, acute energy drink consumption is associated with a transient increase in systolic and diastolic blood pressure (SBP and DBP) as well as heart rate, typically observed within 1-2 hours after consumption [3]. Similar observations regarding increases in blood pressure have also been reported in studies investigating the acute effects of energy drinks during physical exercise. A meta-analysis by Grinberg et al. (21 randomized controlled trials, 296 participants) demonstrated a significant increase in systolic blood pressure response to exercise following energy drink consumption compared with placebo [5].

A study by Basrai et al., using an energy drink containing caffeine, taurine, sugars, and other ingredients, demonstrated increases in blood pressure and alterations in HRV parameters following energy drink consumption [6]. A similar pattern of changes was described in a 2024 study by Çalışkan evaluating the effects of caffeine and taurine on linear and nonlinear cardiovascular function indices in young adults, in which caffeine significantly increased heart rate and systolic blood pressure, whereas taurine appeared to partially attenuate these effects [7].

Pediatric data suggest that energy drink consumption may also elicit significant hemodynamic and electrophysiological responses in children and adolescents. Mandilaras et al. described changes in heart rhythm and electrocardiographic parameters following energy drink consumption in a pediatric population, whereas a randomized study by Oberhoffer et al. confirmed significant increases in both systolic and diastolic blood pressure following energy drink consumption in children and adolescents [2, 8].

In summary, the short-term hemodynamic effect of energy drinks consists of moderate but reproducible increases in blood pressure and heart rate, which may be clinically relevant in individuals with hypertension, heart disease, or in children.

3.1.2. Electrocardiographic Changes – QTc and Other Parameters

Systematic reviews indicate that energy drink consumption may be associated with prolongation of the corrected QT interval (QTc) and other electrocardiographic changes [4]. In the review by Mandato et al. (2025), among studies evaluating electrocardiographic parameters, QTc prolongation was among the most frequently reported findings, while other ECG abnormalities-including alterations in the PR interval, QRS complex, and T-wave morphology-were observed in 57.9% of the analyzed studies [4].

Lee (2019) demonstrated that energy drink consumption was associated with significant QTc prolongation compared with placebo, accompanied by an increase in peripheral blood pressure, whereas the PR interval and QRS duration remained unchanged [9]. The effects of taurine on cardiac repolarization and electrophysiology appear complex and dependent on the biological context as well as the coexistence of other energy drink constituents. At the same time, available evidence suggests that under real-world conditions of energy drink consumption-characterized by high caffeine doses and multicomponent stimulant mixtures-the combined hemodynamic and electrophysiological effects may promote destabilization of cardiac rhythm, particularly in susceptible individuals [1, 3, 9].

Shah et al. (J Am Heart Assoc, 2019) demonstrated that consumption of a large-volume energy drink produced significant prolongation of the corrected QT interval (QTc) and increases in blood pressure compared with a control beverage containing an equivalent dose of caffeine, suggesting the involvement of constituents other than caffeine in the observed effects [11].

3.1.3. Ventricular and Supraventricular Arrhythmias and Sudden Cardiac Arrest

Reviews of clinical case reports indicate that energy drink consumption has been temporally associated with the occurrence of tachyarrhythmias, both supraventricular and ventricular, including isolated episodes of severe and potentially life-threatening presentation [1,10,12]. In the classic study by Goldfarb et al. (2014), the authors presented a review of case reports in which energy drink consumption was temporally associated with cardiovascular disturbances, particularly tachycardia, supraventricular and ventricular arrhythmias, QT interval prolongation, and rare episodes of cardiac arrest [13].

In the review by Higgins et al. (2015), the authors discussed reports suggesting a potential proarrhythmic effect of energy drinks, including QTc prolongation, the occurrence of supraventricular and ventricular tachyarrhythmias, and case reports of arrhythmias observed following energy drink consumption in predisposed individuals, including those with concealed channelopathies such as long QT syndrome and Brugada syndrome [10].

In the review article by Mangi et al. (2017), specific clinical reports were cited indicating a potential association between energy drink consumption and the triggering of severe arrhythmias in patients with concealed arrhythmogenic predispositions. These included the case of an adolescent patient with congenital long QT syndrome type 1 (Dufendach et al.), in whom consumption of a high-caffeine energy drink led to marked QT prolongation and the occurrence of serious arrhythmias. The same review also cited the case of a young man (Rutledge et al.) in whom energy drink consumption combined with alcohol preceded the manifestation of electrocardiographic features of Brugada syndrome and the occurrence of ventricular fibrillation [12].

Kaszuba et al. (2024) described the case of a 26-year-old man who experienced sudden cardiac arrest in a shockable rhythm at his workplace following chronic consumption of very large amounts of energy drinks; the authors suggested that long-term exposure to high doses of caffeine and other stimulants may have constituted a potential trigger for a serious ventricular arrhythmia, in the absence of significant coronary artery abnormalities and without detectable structural heart disease [14].

Ramcharan et al. (2025) described the case of a 24-year-old man without comorbidities who developed ventricular fibrillation and sudden cardiac arrest in a shockable rhythm following an episode of intensive energy drink consumption, with successful resuscitation and interventional treatment; in their

discussion, the authors highlighted the possible role of high doses of caffeine and other energy drink constituents, as well as underlying predispositions, in the manifestation of this severe event [15].

The collected clinical case reports indicate that energy drink consumption may be temporally associated with severe cardiac arrhythmias, including ventricular fibrillation and sudden cardiac arrest, particularly in young individuals, often without previously diagnosed structural heart disease. The reported events include both patients with concealed arrhythmogenic predispositions, such as channelopathies (long QT syndrome and Brugada syndrome), and individuals without detectable abnormalities on standard cardiologic evaluation. In most cases, a temporal association with high or sudden consumption of large quantities of energy drinks is emphasized, suggesting a possible role of the stimulants contained in these beverages-primarily caffeine, often in combination with taurine-as factors triggering the manifestation of life-threatening arrhythmias, without allowing definitive causal inference.

3.1.4. Acute Coronary Syndromes and Other Ischemic Events

Cases of acute myocardial infarction and other acute coronary syndromes have been described in the literature among young, apparently healthy individuals in whom symptom onset was temporally associated with energy drink consumption; however, these data are based predominantly on case reports and do not allow definitive causal conclusions, although they suggest a possible temporal relationship [12,16].

Pallangyo et al. described the case of a 28-year-old man in whom chest pain and ischemic ECG changes developed several hours after consumption of five bottles of an energy drink; coronary angiography demonstrated complete thrombotic occlusion of the proximal left anterior descending (LAD) coronary artery responsible for STEMI, and in their discussion the authors considered potential prothrombotic and endothelial mechanisms related to energy drink consumption [16]. Levent et al. reported a case of NSTEMI following consumption of two cans of an energy drink mixed with alcohol, in which coronary angiography demonstrated LAD occlusion; the authors discussed the possible contribution of the combination of caffeine, taurine, and ethanol to the pathophysiology of acute coronary syndrome, without establishing a definitive causal mechanism [17].

In the review by Azarma et al. (2024), clinical reports involving young adults were analyzed in whom concomitant consumption of energy drinks and alcohol was temporally associated with acute cardiovascular events, including acute coronary syndromes and severe cardiac arrhythmias. The authors emphasized the potential synergistic effects of caffeine and alcohol, which may increase cardiovascular burden and mask symptoms of alcohol intoxication, thereby promoting excessive consumption of both substances [18].

Reviews (Mangi et al., 2017; Mandato et al., 2025) indicate that energy drink consumption is associated with increases in blood pressure and heart rate, accompanied by mechanisms related to enhanced catecholaminergic activity and electrophysiological alterations. Clinical reports have also described episodes of acute ischemia and acute coronary syndromes potentially associated with coronary vasospasm and thrombotic events within the coronary arteries [4, 12].

3.1.5. High-Risk Populations

Higgins et al. and other reviews indicate that the adverse effects of energy drinks may be particularly relevant in children, adolescents, and young adults, as well as in individuals with pre-existing arrhythmias or predisposition to QT prolongation (e.g., long QT syndrome [LQTS]), especially following the consumption of large quantities of energy drinks over a short period of time [10].

In the EDKAR study (Menzel et al.), involving adolescents with chronically high energy drink consumption, no significant or clinically relevant differences were observed in basic cardiologic parameters, including blood pressure, heart rate, and electrocardiographic findings, compared with the control group; however, individuals with high energy drink consumption more frequently exhibited coexisting lifestyle factors with potentially adverse cardiovascular effects, such as tobacco smoking, alcohol consumption, and shorter sleep duration. Due to the cross-sectional design of the study, the authors emphasized that causal inference was not possible [19].

Review authors indicate that energy drink consumption has been associated with potential proarrhythmic effects, including QT interval prolongation, and that susceptibility to such events may be greater among individuals with pre-existing cardiovascular disease and predispositions-including potentially genetic predispositions-particularly in the presence of additional coexisting risk factors [10, 12].

3.1.6. Summary for the Cardiovascular System

The collected evidence indicates that energy drink consumption is associated with:

- reproducible acute increases in blood pressure and alterations in heart rate,
- electrocardiographic changes, including QTc prolongation,
- reports of tachyarrhythmias and sudden cardiac arrest, often in young individuals,
- cases of acute coronary syndromes, sometimes occurring in the absence of classical risk factors.

Although a substantial proportion of the available evidence derives from short-term studies, observational research, and case reports, the collective body of evidence indicates a biologically and clinically relevant potential for adverse effects of energy drinks on the cardiovascular system, particularly among susceptible individuals and those belonging to high-risk groups [10, 12, 13, 14, 15, 16].

3.2. Central Nervous System

3.2.1. Neurobiological Mechanisms: Caffeine + Taurine

Caffeine acts primarily through antagonism of adenosine A_1 and A_2A receptors, leading to increased release of dopamine, noradrenaline, and glutamate and, clinically, to enhanced alertness, shortened reaction time, but also anxiety and insomnia at higher doses [20].

Taurine is an endogenous aminosulfonic acid with pronounced neuromodulatory properties, capable of activating GABA-A and glycine receptors; its effects on extrasynaptic GABA-A receptors and its involvement in tonic neuronal inhibition are particularly well documented, which may reduce neuronal excitability and modulate the balance between inhibitory and excitatory transmission [21, 22]. Numerous experimental studies have demonstrated that taurine increases chloride conductance, eliciting GABAergic and glycinergic currents through activation of GABA-A and glycine receptors; its affinity depends on receptor subtype and localization-while taurine exhibits relatively low affinity toward many glycine receptors and classical GABA-A receptors, selected extrasynaptic GABA-A populations may be activated by taurine already within the micromolar range, conferring physiological relevance to this effect [23, 24, 25].

Although in the mature nervous system taurine predominantly exhibits neuromodulatory potential of an inhibitory nature through interactions with GABA-A and glycine receptors, its effects on neurons remain dependent on the developmental stage of the central nervous system as well as local receptor and network properties. Experimental data further indicate that during early stages of brain development taurine modulates key processes of neurogenesis, cellular migration, and synaptogenesis, participating in the maturation and organization of neuronal networks [3, 23, 24]. From a neurobiological perspective, caffeine and taurine exhibit distinct mechanisms of action-caffeine promotes excitation through blockade of adenosine receptors, whereas taurine possesses inhibitory neuromodulatory properties related to activation of GABA-A and glycine receptors. In clinical practice, energy drink consumption may be accompanied by subjective symptoms of stimulation and alterations in sleep and anxiety regulation [20, 21].

3.2.2. Sleep Disturbances and Excessive Daytime Sleepiness

Large population-based studies involving children, adolescents, and young adults consistently associate energy drink consumption with poorer subjective sleep quality, shorter total sleep duration, and delayed sleep timing. Epidemiological data further indicate an association between regular consumption of these products and prolonged sleep latency, as well as more frequent failure to meet recommendations regarding adequate nocturnal sleep duration. In adult populations, this habit tends to

co-occur within clusters of risk behaviors, being associated with tobacco smoking, unhealthy dietary patterns, and symptoms of psychophysical overload and burnout [26, 27, 28, 29, 30].

A population-based study conducted among Korean adolescents demonstrated a significant association between sleep disturbances and energy drink consumption-short sleep duration (less than 5 hours) and low satisfaction with nocturnal sleep were associated with an increased likelihood of consuming these products, even after adjustment for potential confounding factors [26]. In turn, a Norwegian cross-sectional study published in BMC Public Health demonstrated a dose–response relationship between the frequency of energy drink consumption and sleep parameters; among adolescents regularly consuming these products, shorter total sleep duration and prolonged sleep latency were observed [27].

Literature reviews concerning children and adolescents indicate that the high caffeine content of energy drinks is associated with sleep disturbances, including difficulty initiating sleep, more frequent nocturnal awakenings, and reduced sleep duration. These disturbances are, in turn, associated with poorer concentration, impaired school functioning, and more frequent behavioral and emotional problems among young consumers [3, 31, 32].

More recent analyses indicate that sleep-related problems observed among energy drink consumers frequently co-occur with psychosocial and emotional difficulties. The review by Ajibo et al. focusing on children and adolescents found that energy drink consumption was associated with a higher prevalence of emotional problems, poorer well-being, and unfavorable aspects of psychosocial functioning [28, 29, 30]. Dobrek (2025) further highlighted that sleep disturbances associated with energy drink consumption may contribute to the development of a self-reinforcing behavioral pattern in which fatigue increases the tendency to consume energy drinks, while their consumption subsequently co-occurs with further deterioration in sleep quality and worsening fatigue [3, 29].

3.2.3. Anxiety, Agitation, and Psychiatric Symptoms

High caffeine intake has been associated with increased anxiety symptoms, agitation, and irritability and, in predisposed individuals, may provoke panic symptoms; however, this response exhibits considerable interindividual variability [20, 32]. The review by Ishak et al. further indicates that high caffeine consumption is associated with worsening anxiety and sleep disturbances and may co-occur with certain psychotic symptoms, particularly in susceptible individuals [20].

Kożuchowska et al. (2025) summarized that in children and adolescents, caffeine derived from energy drinks may be associated with excessive psychomotor agitation, anxiety, hyperactivity, and difficulties with concentration and cognitive functioning, particularly in the presence of coexisting sleep disturbances. Moreover, high doses of this substance may lead to symptoms of acute caffeine intoxication, manifested, among others, by muscle tremors, cardiac rhythm disturbances, and increased nervousness and irritability [3, 32].

The literature also describes cases of severe psychiatric symptoms temporally associated with excessive energy drink consumption. Silva-Maldonado et al. presented the case of a patient without previous psychiatric history who, following a period of intensive energy drink consumption, developed severe anxiety-depressive symptoms, distress, and psychomotor agitation. Subsequent psychological assessment led to diagnoses including panic disorder and generalized anxiety disorder; however, the authors emphasized the observational nature of this relationship and the need for further specialist psychiatric evaluation [33].

Reviews concerning mental health emphasize that caffeine may exacerbate pre-existing psychiatric disorders, whereas energy drinks-as concentrated sources of caffeine and other biologically active substances, such as guarana and taurine-may act as triggers or aggravating factors for symptoms in individuals with psychiatric predispositions, although the contribution of individual constituents remains incompletely understood. Cases of severe psychiatric symptoms and decompensation of mental status temporally associated with high consumption of caffeine and energy drinks have also been reported; however, the available data are predominantly observational and do not allow definitive causal conclusions [3, 30, 34].

3.2.4. Seizures and Seizure Threshold

Seizures constitute severe neurological complications described in association with energy drink consumption in both adults and children. Among pediatric patients, a case of seizure temporally associated with energy drink consumption has been described; however, the authors emphasized that although a causal relationship could not be definitively established, the energy drink may have acted as a triggering factor for the episode [35].

The review by Kycler et al. (2024) and the publication by Dobrek (2025) indicate that severe acute medical complications reported following energy drink consumption include neurological disturbances, among them seizures and disturbances of consciousness. Severe cardiovascular complications, such as tachycardia and marked elevations in blood pressure, have also been described in the literature and may constitute direct health threats, particularly in individuals with pre-existing cardiovascular disease [3, 36].

Although the direct involvement of taurine in seizure induction remains unclear, this substance-through modulation of GABA-A and glycine receptors-affects the balance between inhibitory and excitatory neurotransmission within the central nervous system. The significance of these effects may depend on local receptor properties and the developmental stage of the brain, which appears particularly relevant in the developing nervous system [21, 23, 24].

3.2.5. Mood Disturbances, Cognitive Function, and "Energy Drink Use Disorder"

Observational studies and reviews involving adolescent populations indicate that frequent energy drink consumption is associated not only with sleep disturbances but also with a higher prevalence of emotional problems, poorer psychosocial functioning, and-in some analyses-greater severity of depressive symptoms and psychological burden [26, 27, 28, 29, 30].

The literature concerning mental health and risk-taking behaviors emphasizes an association between regular energy drink consumption and elevated anxiety levels, impulsivity, poorer emotional regulation, and a greater propensity toward aggressive behavior and other behavioral problems, although determining the direction of this relationship remains difficult due to the observational nature of most studies [3, 20, 29, 34].

An increasingly discussed phenomenon is "energy drink use disorder"-a proposed behavioral construct referring to problematic energy drink use that exhibits features resembling substance use disorders, such as loss of control over consumption, risky use, and symptoms of tolerance and withdrawal [3, 34].

Chronic high consumption of energy drinks may be associated with the development of caffeine dependence-like features and withdrawal symptoms, including headaches, fatigue, dysphoria, and irritability following reduction or cessation of consumption. These phenomena are often accompanied by coexisting sleep disturbances and psychological burden [3, 20].

3.2.6. Summary – Central Nervous System

From the perspective of the central nervous system, energy drinks containing caffeine and taurine:

- are associated with sleep disturbances, shortened sleep duration, and impaired sleep quality,
- contribute to the occurrence of anxiety symptoms, agitation, and exacerbation of pre-existing psychiatric disorders,
- may act as triggering factors for seizures in predisposed individuals,
- are associated with greater severity of emotional problems and-in some studies-depressive symptoms among adolescents,
- may lead to a problematic pattern of use resembling substance use disorders ("energy drink use disorder") [3, 26, 29, 32, 34].

Although mechanistically taurine predominantly exhibits inhibitory neuromodulatory effects through interactions with GABA-A and glycine receptors, in energy drinks it coexists with high doses of caffeine and other biologically active substances. Consequently, the overall impact of these products on the central nervous system remains complex and may influence the balance between excitatory and

inhibitory transmission, particularly in children, adolescents, and individuals with pre-existing psychiatric disorders or epilepsy [3, 21, 23, 24].

4. Discussion

The present narrative review summarizes current evidence concerning the cardiovascular and central nervous system effects associated with energy drink consumption, with particular attention to the biological roles of caffeine and taurine. The collected literature suggests that energy drinks may exert clinically relevant physiological and pathological effects involving multiple organ systems, although the strength of available evidence varies substantially depending on the analyzed outcome and study design [1, 3, 4].

Among cardiovascular outcomes, the most consistently reported findings concern acute hemodynamic and electrophysiological alterations. Numerous clinical and experimental studies indicate that energy drink consumption is associated with transient increases in blood pressure and, less consistently, heart rate [1, 3, 4, 5, 6, 7, 8]. Electrocardiographic changes-particularly QTc prolongation-have also been repeatedly reported in both experimental and clinical settings [4, 9, 11]. Although these changes are generally moderate and often reversible in healthy individuals, their potential clinical relevance should not be underestimated, particularly in susceptible populations. The reproducibility of these findings across independent studies supports the biological plausibility of cardiovascular effects associated with energy drink consumption [1, 4, 10].

At the same time, interpretation of severe cardiovascular events requires caution. Reports of ventricular arrhythmias, sudden cardiac arrest, and acute coronary syndromes derive predominantly from case reports and case series, which inherently limit causal inference [12, 13]. Nevertheless, the recurring temporal association between excessive or rapid energy drink consumption and severe cardiovascular events-particularly among young individuals and those with concealed arrhythmogenic predispositions-raises clinically important concerns [10, 12, 13, 14, 15, 16, 17, 18]. The available evidence suggests that energy drinks may function not necessarily as independent causal agents but rather as potential triggering or precipitating factors in biologically susceptible individuals.

The central nervous system findings demonstrate a similarly heterogeneous but clinically relevant pattern. Among neurological and psychiatric outcomes, disturbances of sleep and circadian regulation appear to represent the most consistently documented effects [26, 27, 28, 29,30, 31]. Population-based studies involving children, adolescents, and young adults repeatedly associate energy drink consumption with reduced sleep duration, delayed sleep onset, prolonged sleep latency, and impaired subjective sleep quality [26, 27, 30]. Importantly, these disturbances frequently coexist with emotional and psychosocial difficulties, suggesting that the effects of energy drinks may extend beyond transient stimulation and involve broader domains of mental well-being and daily functioning [28, 29, 30].

Psychiatric and behavioral outcomes require more cautious interpretation. Although observational studies and reviews consistently report associations between energy drink consumption and anxiety symptoms, agitation, emotional dysregulation, and behavioral difficulties, the directionality of these relationships remains uncertain [3, 20, 32, 33, 34]. It is plausible that psychological distress, sleep disturbances, and stimulant consumption interact bidirectionally, forming self-reinforcing behavioral patterns rather than simple linear cause–effect relationships. Similarly, reported cases of psychiatric decompensation and severe psychological symptoms temporally associated with excessive energy drink intake warrant clinical awareness but cannot be interpreted as definitive evidence of causation [20, 33, 34].

The phenomenon described as “energy drink use disorder” further expands discussion beyond classical toxicological models. Emerging evidence suggests that, in some individuals, chronic and excessive energy drink consumption may exhibit behavioral characteristics resembling substance use disorders, including impaired control, withdrawal symptoms, and continued use despite adverse consequences [3, 34]. Although this construct remains preliminary and lacks formal diagnostic consensus, it highlights the need to consider problematic energy drink use not solely as a nutritional or lifestyle issue but potentially also as a behavioral health concern.

From a mechanistic perspective, the interaction between caffeine and taurine remains complex and incompletely understood. Caffeine exerts predominantly stimulatory effects through antagonism of adenosine receptors and enhancement of catecholaminergic and glutamatergic signaling, whereas taurine demonstrates neuromodulatory properties associated primarily with GABA-A and glycine receptor activation [20, 21, 22, 23, 24, 25]. Experimental evidence suggests that taurine may exhibit inhibitory and potentially protective effects under certain physiological conditions [21, 22, 23, 24, 25]. However, real-world energy drink consumption involves simultaneous exposure to high caffeine doses and multicomponent stimulant mixtures, making extrapolation from isolated ingredient studies difficult. Consequently, the biological effects of commercially available energy drinks cannot be reduced to the action of caffeine or taurine alone [1, 3, 20].

The present review should also be interpreted in light of several limitations inherent to the available literature. Much of the evidence derives from short-term studies, observational investigations, and clinical case reports [3, 4, 12]. Considerable heterogeneity exists regarding energy drink composition, serving volume, caffeine content, patterns of consumption, and the coexistence of alcohol or other stimulants [1, 4, 18]. These methodological differences complicate direct comparisons across studies and limit the ability to establish dose–response relationships or definitive causal mechanisms. Furthermore, vulnerable populations-including adolescents and individuals with pre-existing cardiovascular or psychiatric conditions-remain relatively understudied in prospective research [10, 19, 32].

Future investigations should therefore prioritize prospective and mechanistically oriented studies using standardized exposure assessment and clearer characterization of energy drink formulations. Particular attention should be directed toward pediatric and adolescent populations, high-risk cardiovascular groups, and the long-term neurobehavioral consequences of habitual energy drink consumption [2, 19, 26, 27, 28, 29, 30, 31, 32]. Improved understanding of interactions between caffeine, taurine, and other biologically active ingredients may also help clarify whether adverse outcomes result primarily from cumulative stimulant burden or from specific multicomponent interactions unique to energy drinks.

Overall, the available evidence indicates that energy drinks should not be regarded merely as conventional caffeinated beverages. While many observed physiological effects are transient and moderate, the accumulated literature suggests biologically and clinically relevant potential for adverse cardiovascular and neuropsychiatric consequences, particularly in predisposed individuals and under conditions of excessive consumption [1, 3, 4, 10, 12].

5. Conclusions

Energy drink consumption is associated with measurable cardiovascular and central nervous system effects, most consistently involving transient hemodynamic alterations, electrocardiographic changes, and disturbances of sleep and emotional functioning. While many observed effects are moderate and reversible in healthy individuals, the available evidence indicates clinically relevant potential for adverse outcomes under conditions of excessive intake or biological susceptibility.

Current evidence remains limited by the predominance of observational studies, heterogeneous exposure patterns, and case-based reports, restricting definitive causal interpretation. Nevertheless, the accumulated literature supports a cautious approach to energy drink consumption, particularly among children, adolescents, and individuals with pre-existing cardiovascular or psychiatric vulnerabilities.

Further prospective and mechanistic research is required to clarify long-term consequences, dose–response relationships, and the contribution of individual energy drink components to observed adverse effects.

Disclosure

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Abbreviations

The following abbreviations are used in this manuscript:

A_1 – Adenosine A_1 receptor

A_2A – Adenosine A_2A receptor

ACS – Acute coronary syndrome

CNS – Central nervous system

DBP – Diastolic blood pressure

ECG – Electrocardiogram

ED(s) – Energy drink(s)

GABA-A – Gamma-aminobutyric acid type A receptor

HRV – Heart rate variability

LAD – Left anterior descending coronary artery

LQTS – Long QT syndrome

NSTEMI – Non-ST-elevation myocardial infarction

PR – PR interval

QRS – QRS complex

QTc – Corrected QT interval

SBP – Systolic blood pressure

STEMI – ST-elevation myocardial infarction

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