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Research Article

Energy Drink Consumption, Gastritis, and Anxiety Disorders: A Narrative Review of the Evidence and Proposed Mechanistic Links

Atteyat Aboelmaged Semeya*

Department of Hepatogastroenterology, Benha Teaching Hospital, Benha, Egypt

Abstract

Energy drink (ED) consumption has increased sharply worldwide over the past two decades, particularly among adolescents and young adults. Two health domains have drawn growing clinical attention: gastrointestinal mucosal injury, as documented on upper endoscopy, and psychiatric symptoms, particularly anxiety. This narrative review synthesizes the available evidence linking ED consumption to (1) endoscopic and histologic gastritis and (2) anxiety disorders, and examines the gut-brain axis as a plausible bidirectional mechanism connecting the two. The evidence base consists mainly of case reports, small case series, cross-sectional and longitudinal cohort studies, and animal models; controlled human trials isolating EDs as a discrete exposure are scarce. Caffeine, the dominant pharmacologically active constituent of most EDs, plausibly contributes to both outcomes through distinct but potentially interacting pathways: stimulation of gastric acid secretion and impairment of mucosal defenses on one hand, and antagonism of adenosine A1/A2A receptors with downstream effects on serotonergic and noradrenergic signaling on the other. No existing study has directly quantified a combined triad of ED exposure, endoscopic gastritis, and anxiety disorder within a single cohort, and therefore no pooled effect estimate can be responsibly reported for this specific combination. This review instead maps the separate literatures, highlights converging mechanistic plausibility, and identifies methodological gaps that limit causal inference, including confounding by caffeine from other sources, alcohol co-ingestion, sleep disruption, and reliance on self-reported consumption.

Keywords

Energy Drinks, Gastritis, Anxiety Disorders, Caffeine, Gut-Brain Axis, Adolescents

1. Introduction

Energy drinks are non-alcoholic beverages formulated to enhance alertness and physical performance, typically containing high concentrations of caffeine together with taurine, guarana, B vitamins, and simple sugars. A single 250 mL serving of a widely consumed brand contains roughly 80 mg of caffeine and 1000 mg of taurine, though caffeine content across the category ranges from about 50 mg to over 500 mg per serving [1]. Surveillance data from the European Food Safety Authority indicate that around 30% of European adults and up to 68% of adolescents report consuming EDs within the preceding year [10], and similar or higher prevalence has been reported elsewhere.

*Correspondence: Atteyat Aboelmaged Semeya (semeyaatteyat@gmail.com)

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Two lines of clinical concern have emerged in parallel. Gastroenterologists have increasingly reported endoscopic and histologic mucosal changes, ranging from reactive gastropathy to atrophic gastritis and intestinal metaplasia, in patients with heavy ED use [2, 11]. Independently, a substantial body of epidemiological and experimental work has linked ED consumption to anxiety, stress, and related psychiatric symptoms, particularly in adolescents and young adults [1, 5-8]. Because both the stomach and the brain are richly connected through the vagally mediated gut- brain axis, and because caffeine acts directly on both gastric and central nervous system targets, it is biologically plausible that these two outcomes are not independent but instead reflect overlapping or interacting pathophysiology. This review maps the evidence for each association separately, then considers the gut-brain axis as a candidate unifying mechanism.

2. Methods and Scope of This Review

This is a narrative, non-systematic synthesis of the peer-reviewed and grey literature identified through targeted searches for terms including "energy drinks," "gastritis," "endoscopy," "gastric mucosa," "anxiety," "caffeine," "adenosine receptor," and "gut-brain axis." It does not follow a Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) protocol, and no formal risk-of-bias assessment, quantitative pooling, or forest-plot meta-analysis was performed. This distinction matters: to our knowledge, no published systematic review or meta-analysis has quantitatively pooled effect estimates across all three domains addressed here (ED exposure, endoscopic gastritis, and anxiety disorder) within the same analytic framework, and the underlying primary studies are too heterogeneous in design, exposure definition, and outcome measurement to support a statistically valid pooled estimate without a dedicated systematic review. Readers seeking a quantitative meta-analysis of any single domain (for example, ED consumption and depression/anxiety symptoms in adolescents) are directed to the existing systematic reviews cited throughout this article, which remain the appropriate primary sources for effect sizes.

3. Composition and Relevant Pharmacology of Energy Drinks

The physiological effects most relevant to this review are attributable primarily to caffeine, a non- selective antagonist of adenosine A1, A2A, A2B, and A3 receptors [17, 18]. Adenosine receptor agonism is generally anxiolytic, while antagonism, particularly at the A2A receptor, is anxiogenic at moderate to high doses or in susceptible individuals [18]. Caffeine additionally reduces phosphodiesterase activity, raising intracellular cyclic AMP in gastric parietal cells and stimulating hydrochloric acid secretion; more recent work has also implicated oral and gastric bitter-taste receptors (TAS2Rs) in caffeine-induced gastric acid secretion [12]. Taurine, present in high concentration in most EDs, has demonstrated cardiac inotropic effects in vitro but has not been shown to independently alter gastric acid secretion or mucosal integrity to the same degree as caffeine [14]. Guarana, itself a caffeine-rich botanical additive, compounds the total stimulant load of many formulations [15]. Beyond their pharmacologically active ingredients, most EDs are also acidic beverages (low pH, often from citric or phosphoric acid) and carbonated, both of which can independently irritate gastric and esophageal mucosa.

4. Energy Drinks and the Endoscopic / Histologic Gastric Picture

4.1. Mechanistic Pathway

Chronic high-volume ED consumption is proposed to injure the gastric mucosa through a combination of (a) caffeine-stimulated gastric acid hypersecretion, (b) caffeine-mediated inhibition of protective mucus secretion, (c) relaxation of the lower esophageal sphincter promoting reflux, and (d) direct chemical irritation from the beverage's intrinsic acidity and carbonation [11-13]. In combination, these mechanisms create an environment favoring erosive and inflammatory mucosal change, and, with sustained exposure, may predispose to atrophic changes.

4.2. Clinical and Histologic Evidence

Human evidence in this domain consists largely of case reports and small case series rather than controlled cohort studies. A widely cited case report described biopsy-confirmed atrophic gastritis and gastric intestinal metaplasia in a patient with heavy chronic ED consumption, with reported histologic reversal to normal mucosa on repeat biopsy after ED cessation, a finding the authors interpreted as evidence of a causal and potentially reversible relationship rather than coincidental H. pylori-negative atrophic change [2]. A separately reported case describes severe gastritis with biopsy findings favoring chemical irritation over infectious or viral etiology in a patient with heavy stimulant-containing ED use, again in the absence of the lymphocytic infiltration more typical of infectious gastritis ([3], cited as ACG abstract). These reports are individually low on the evidence hierarchy, but their histologic detail and, in one case, apparent reversibility on cessation, strengthen the plausibility of a direct causal contribution rather than pure confounding.

Animal studies provide complementary mechanistic support. Rodent models exposed to commercial ED formulations have demonstrated dose-dependent histologic change in gastric and intestinal tissue, including parietal cell hyperplasia,

mucosal vascular congestion, and, at higher doses, mononuclear infiltration and goblet cell metaplasia in the small intestine, changes the study authors flagged as potentially precancerous at the highest exposure levels tested [3, 4]. Such animal data cannot be directly extrapolated to typical human consumption patterns but are broadly consistent with the mechanistic pathway described above.

It is also worth situating these findings within the broader endoscopic gastritis literature. Standardized endoscopic classification systems, such as the Kyoto classification, primarily describe findings associated with active or past Helicobacter pylori infection (diffuse or spotty redness, mucosal swelling, enlarged folds, nodularity) versus H. pylori-naive mucosa (regular arrangement of collecting venules, fundic gland polyps) ([7], ref to Kyoto classification source). Reported ED-associated changes, reactive gastropathy, atrophy, and intestinal metaplasia in H. pylori-negative patients, do not map neatly onto an infectious pattern, which supports a chemical or irritant mechanism distinct from H. pylori-driven gastritis, though co-existing H. pylori infection, NSAID use, alcohol intake, and smoking remain important and frequently under-controlled confounders in the available reports.

5. Energy Drinks and Anxiety Disorders

5.1 Epidemiological Evidence

The epidemiological literature linking ED consumption to anxiety and related psychological distress is considerably larger and more consistent than the gastritis literature, though it too is dominated by cross-sectional designs. A systematic review of 57 studies encompassing more than 1.2 million children and young people across 21 countries found that ED consumption was associated with increased risk of attention-deficit/hyperactivity disorder (ADHD) symptoms, psychological distress, depression, and anxiety, in addition to poor sleep quality, short sleep duration, and lower academic performance [1]. A separate systematic review focused on adolescents aged 11-18 similarly reported a strong positive association between ED consumption and tendency toward anxiety, depression, and impulsivity, alongside risky behaviors and sleep disturbance, with heavier consumption among boys [6]. A longitudinal cohort study of young adult males found that ED consumption was prospectively associated with elevated depression, anxiety, and stress scores, an important design strength given that most other studies in this area are cross-sectional and cannot establish temporal precedence [5]. A broader narrative review of the mental health literature concluded that, while a minority of studies reported null or even inverse associations, the majority reported positive associations between ED consumption and stress, anxiety, or depressive symptoms, while cautioning that cross-sectional designs preclude firm causal conclusions [8]. Individual case reports have also described new-onset anxiety symptoms, including restlessness, difficulty concentrating, and insomnia, that improved after ED discontinuation, and, at the more severe end, isolated reports of psychosis or panic-like presentations following heavy acute consumption [8, 9].

5.2. Pharmacological Mechanism

The leading proposed mechanism for ED-associated anxiety is caffeine's antagonism of adenosine A1 and A2A receptors. Adenosine receptor agonists are anxiolytic, and antagonism, particularly of A2A receptors, produces anxiogenic effects in both rodent models and humans at moderate to high doses [16, 18]. Genetic studies have identified specific A2A receptor gene polymorphisms associated with greater self-reported anxiety following an acute caffeine challenge, and some of the same polymorphisms have separately been associated with vulnerability to panic disorder [19]. Controlled challenge studies confirm that doses of caffeine well within the range found in one to several servings of some EDs can provoke measurable increases in subjective anxiety and, in patients with pre-existing panic disorder, panic attacks, with doses above roughly 400 mg inducing panic attacks in about half of individuals with panic disorder in earlier controlled work [20]. A complementary hypothesis implicates caffeine-related reduction in central serotonin synthesis via adenosine receptor blockade as a further contributor to anxiogenic effects, particularly in vulnerable individuals [17].

6. The Gut-Brain Axis as a Candidate Unifying Mechanism

A substantial and independent body of gastroenterology literature has established that disorders of gut-brain interaction, including functional dyspepsia, are bidirectionally linked to anxiety and depression. Classic "top-down" models proposed that psychiatric symptoms alter visceral symptom perception, but more recent human and animal evidence supports an additional "bottom- up" pathway in which primary gastrointestinal pathology and inflammation can themselves induce anxiety- and depression-like states, likely via vagal afferent signaling to limbic structures such as the amygdala [22]. Mendelian randomization studies, which use genetic variants to strengthen causal inference beyond what cross-sectional association allows, have found that genetically predicted anxiety and depression are associated with increased risk of several disorders of gut-brain interaction, consistent with true bidirectionality rather than a purely psychosomatic or purely somatopsychic relationship [21]. A recent narrative synthesis of this literature explicitly frames the relationship between functional gastrointestinal disorders and anxiety as bidirectional, mediated by shared neuroendocrine, immune, and microbiome-related pathways along the brain- gut axis [23].

Applied to energy drinks, this body of work suggests a plausible dual and potentially reinforcing pathway rather than two

unrelated coincidental findings. Caffeine and the acidic, carbonated matrix of EDs may directly injure the gastric mucosa through the acid-secretory and mucus- inhibitory mechanisms described in Section 4, while caffeine simultaneously exerts a direct central anxiogenic effect via adenosine receptor antagonism, as described in Section 5. If gut-brain axis signaling is intact, gastric mucosal irritation and resulting visceral discomfort could plausibly amplify centrally mediated anxiety through vagal afferent pathways, while heightened anxiety could, in turn, increase visceral hypersensitivity and symptom perception, a feed-forward loop that has been demonstrated for other causes of gastric and functional gastrointestinal pathology but has not yet been specifically tested in the context of ED-induced gastritis. This remains a mechanistically plausible hypothesis rather than an established finding, since no study identified in this review has directly measured gastric mucosal status, ED exposure, and validated anxiety disorder diagnoses concurrently in the same population.

7. Critical Appraisal of the Evidence

Several methodological limitations temper causal interpretation across both literatures. First, the gastritis literature is dominated by case reports and animal studies; the largest body of human evidence, the case report literature, is inherently susceptible to publication bias toward striking or unusual presentations and cannot establish population-level risk. Second, the anxiety literature, while larger and including at least one longitudinal cohort study, remains predominantly cross- sectional, so reverse causation (for example, individuals with pre-existing anxiety self-medicating with stimulants, or using EDs to compensate for anxiety-related fatigue) cannot be excluded. Third, almost all studies in both literatures rely on self-reported ED consumption without biochemical verification, and few adequately separate the contribution of ED-derived caffeine from caffeine consumed via coffee, tea, or other sources, or from co-ingested alcohol, a well-documented practice among young ED consumers that independently affects both gastric mucosa and mental health outcomes. Fourth, sleep disruption, itself independently associated with both gastrointestinal symptoms and anxiety, is a frequently reported correlate of ED use and a plausible unmeasured confounder in several of the reviewed studies. Finally, and most directly relevant to the framing of this review, no identified study has concurrently assessed endoscopic/histologic gastric findings, validated anxiety disorder diagnoses, and quantified ED exposure within a single design, so the tri-directional relationship proposed in Section 6 remains a hypothesis generated from adjacent literatures rather than a directly demonstrated finding.

8. Limitations of This Review

This review is narrative rather than systematic: searches were targeted rather than exhaustive, no dual independent screening or formal risk-of-bias appraisal was conducted, and no statistical pooling was attempted. Consistent with the absence of directly comparable quantitative data across the three domains addressed, this review reports no pooled odds ratio, relative risk, or standardized mean difference for the combined association between ED consumption, endoscopic gastritis, and anxiety disorders; presenting such a figure would misrepresent the underlying evidence. Readers requiring a formal quantitative synthesis of the ED-mental health literature specifically should consult the systematic reviews cited in Section 5.1, which report their own pooled or narratively synthesized effect estimates within defined populations.

9. Conclusion

Energy drink consumption is independently associated, in separate bodies of literature, with adverse endoscopic and histologic gastric findings and with anxiety and related psychiatric symptoms, most consistently in adolescents and young adults. Caffeine provides a coherent, if not exclusively sufficient, pharmacological explanation for both associations, acting on gastric acid secretion and mucosal defenses on one hand and on central adenosine receptor signaling on the other. The gastroenterology literature on gut-brain axis bidirectionality in functional gastrointestinal disorders offers a biologically plausible mechanism by which these two outcomes could interact and reinforce one another in a subset of heavy ED consumers, but this specific triad has not yet been directly studied. Prospective cohort studies that concurrently assess quantified ED and caffeine exposure, endoscopic/histologic gastric status, and validated anxiety disorder diagnoses, ideally with objective consumption verification and adequate control for alcohol, sleep, and other caffeine sources, are needed to test this hypothesis directly and to determine whether ED-associated gastritis and ED-associated anxiety represent a genuinely interacting clinical phenomenon or two parallel but mechanistically separate consequences of the same exposure.

Abbreviations

ED	Energy Drink
ADHD	Attention-Deficit/Hyperactivity Disorder
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
TAS2Rs	Type 2 Bitter Taste Receptors

Author Contributions

Atteyat Aboelmaged Semeya: Conceptualization, Investigation, Methodology, Writing – original draft, Writing – review & editing

Conflicts of Interest

The author declares no conflicts of interest.

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