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EXERCISE-INDUCED GASTROINTESTINAL SYMPTOMS IN ENDURANCE ATHLETES: MECHANISMS, PREVENTION, AND MANAGEMENT – A NARRATIVE REVIEW

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ABSTRACT

Background: Gastrointestinal (GI) symptoms are common among endurance athletes and may impair performance, particularly during prolonged, high-intensity exercise in hot environments or under conditions of dehydration. These disturbances are collectively referred to as Exercise-Induced Gastrointestinal Syndrome (EIGS).
Objective: This review summarizes current evidence on the epidemiology, pathophysiological mechanisms, consequences, and prevention strategies for EIGS in endurance athletes.
Methods: A narrative review of the literature was conducted, focusing on the prevalence of GI symptoms during endurance exercise, the underlying mechanisms of EIGS—including splanchnic hypoperfusion, epithelial barrier disruption, ischemia-reperfusion injury, mechanical stress, nutritional factors, and neuroendocrine responses—and evidence-based prevention and management strategies.
Results: GI symptoms affect approximately 30–50% of endurance athletes, with the highest prevalence reported in marathon, ultra-endurance, and triathlon events. EIGS results from the interaction of reduced splanchnic blood flow, hyperthermia, epithelial injury, and nutritional and mechanical stress, leading to increased intestinal permeability, inflammation, impaired fluid and nutrient absorption, dehydration, and reduced athletic performance. Hypertonic carbohydrate intake, high-FODMAP foods, and nonsteroidal anti-inflammatory drug use may increase symptom risk. Individualized nutrition and hydration strategies, gut training, and low-FODMAP dietary approaches in susceptible athletes may reduce symptom severity.
Conclusions: EIGS is a multifactorial condition driven by interacting circulatory, thermal, mechanical, nutritional, and neuroendocrine stressors. Although no single intervention completely prevents GI symptoms, individualized fueling strategies and gut training appear to be the most effective current approaches. Further research is needed to optimize prevention and identify factors underlying individual susceptibility.

KEYWORDS

Gastrointestinal Symptoms; Endurance Sports; Exercise-Induced Gastrointestinal Syndrome; Intestinal Permeability; Splanchnic Blood Flow; Gut Training

CITATION

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1. Introduction

The popularity of endurance and ultra-endurance sports has increased substantially over recent decades, attracting both elite and recreational athletes worldwide (1, 2). Participation in activities such as marathon running, long-distance triathlon, and ultra-trail racing imposes considerable physiological demands that require coordinated adaptations across multiple organ systems. While cardiovascular, respiratory, and musculoskeletal responses to exercise have been extensively investigated, the gastrointestinal (GI) tract has emerged as an important determinant of exercise tolerance, nutritional intake, and athletic performance during prolonged endurance exercise (3, 4).

Gastrointestinal symptoms are frequently reported by endurance athletes and represent a common challenge during both training and competition. Epidemiological studies indicate that approximately 30–50% of endurance athletes experience at least one GI symptom during exercise, with prevalence increasing substantially under conditions of prolonged exercise duration, dehydration, and environmental heat stress (3, 4, 5). Symptoms may involve either the upper or lower gastrointestinal tract and range from mild discomfort to severe manifestations that impair performance or necessitate withdrawal from competition.

The concept of Exercise-Induced Gastrointestinal Syndrome (EIGS) has been introduced to describe the complex physiological disturbances associated with strenuous exercise. This framework encompasses a spectrum of exercise-related gastrointestinal perturbations, including reductions in splanchnic blood flow, disruption of epithelial barrier function, altered gastrointestinal motility, and immune activation (6). These

responses are influenced by multiple interacting factors, including exercise intensity and duration, environmental conditions, nutritional practices, and individual susceptibility.

The pathophysiology of exercise-induced GI symptoms is multifactorial. Current evidence suggests that gastrointestinal disturbances arise through the interaction of ischemic, mechanical, nutritional, and neuroendocrine mechanisms, which collectively contribute to impaired gastrointestinal function during exercise (4, 6). Beyond their impact on athlete comfort, GI symptoms have been associated with reduced exercise performance, compromised nutritional intake, and an increased likelihood of race withdrawal in susceptible individuals (7).

Therefore, the aim of this narrative review is to summarize current evidence regarding the epidemiology, pathophysiological mechanisms, and clinical consequences of exercise-induced gastrointestinal symptoms in endurance athletes. Furthermore, this review discusses evidence-based nutritional and practical strategies that may help reduce symptom burden and optimize gastrointestinal function during endurance competition.

2. Research materials and methods

This work was carried out as a narrative literature review aimed at synthesizing the current scientific evidence oregarding the Gastrointestinal Symptoms in Endurance Athletes. Publications indexed in PubMed, Scopus, Google Scholar, and MEDLINE were reviewed. The search strategy employed combinations of the following keywords: “gastrointestinal symptoms,” “endurance activity,” “endurance sport,” “gut permeability,” “splanchnic blood flow ” and “sports nutrition” In total, 43 articles were included in the analysis. Only English-language studies relevant to the topic were considered. Preference was given to recent review papers, clinical research studies, and international guidelines to ensure the inclusion of up-to-date and evidence-based information.

3. Research results

3.1. Epidemiology and clinical manifestations

3.1.1. Prevalence Rates Across Endurance Disciplines

The prevalence of exercise-induced gastrointestinal symptoms varies considerably according to sport discipline, exercise intensity, event duration, environmental conditions, and methods of symptom assessment. Across endurance sports, approximately 30–50% of athletes report one or more GI symptoms during exercise, although substantially higher rates have been observed during competitive events and under conditions of heat stress (3, 4, 5).

The occurrence and severity of GI symptoms appear to increase with exercise duration. Higher rates of moderate-to-severe symptoms have been reported during marathon and ultra-marathon events, suggesting a cumulative effect of prolonged physiological stress on gastrointestinal function (2). Furthermore, field observations indicate that severe GI symptoms may contribute to impaired performance and, in some cases, withdrawal from competition (7).

Differences in symptom prevalence have also been reported between endurance disciplines. Long-distance runners consistently demonstrate the highest rates of lower gastrointestinal complaints, including abdominal cramping, urgency to defecate, and diarrhea (5, 8). In contrast, cyclists generally report lower frequencies of lower GI symptoms but may experience a greater prevalence of upper gastrointestinal complaints, particularly reflux-related symptoms (5, 8). Triathletes are exposed to multiple physiological and mechanical stressors across sequential disciplines and frequently report symptoms involving both the upper and lower gastrointestinal tract (5).

Several individual factors may further influence susceptibility to GI distress. Previous studies have suggested higher symptom prevalence among less experienced athletes and have reported a greater burden of GI symptoms in female competitors, although findings remain inconsistent across populations (5).

3.1.2. Clinical Presentation

Exercise-induced gastrointestinal symptoms are commonly classified according to their anatomical origin as either upper or lower gastrointestinal manifestations (9).

3.1.2.1. Upper Gastrointestinal Symptoms

Include gastroesophageal reflux, heartburn, nausea, vomiting, epigastric pain, early satiety, and upper abdominal bloating (9). These symptoms are frequently reported during prolonged endurance exercise and may impair the athlete’s ability to maintain adequate fluid and carbohydrate intake. Among endurance disciplines, upper GI complaints appear particularly common in cycling and triathlon events, where athletes often report nausea, gastric discomfort, and reflux-related symptoms (5).

3.1.2.2. Lower Gastrointestinal Symptoms

Typically include abdominal cramping, flatulence, urgency to defecate, loose stools, diarrhea, and, in rare cases, gastrointestinal bleeding associated with ischemic colitis (9). Such symptoms are especially prevalent among runners and ultra-endurance athletes, with diarrhea and abdominal pain representing some of the most frequently reported complaints during competition (2, 5). Although many episodes are transient and self-limiting, severe lower GI symptoms may significantly impair performance and contribute to premature race termination.

Table 1. Characteristics of gastrointestinal symptoms across endurance sport disciplines.

Discipline	Predominant gastrointestinal symptoms	Relative symptom burden
Marathon running	Abdominal cramping, urgency to defecate, diarrhea, occasional gastrointestinal bleeding	High
Ultra-marathon running	Severe lower GI symptoms, nausea, abdominal pain, diarrhea	Very high
Cycling	Gastroesophageal reflux, nausea, bloating, epigastric discomfort	Moderate
Triathlon	Mixed upper and lower GI symptoms, including nausea, reflux, abdominal cramping, and diarrhea	High

Classification is based on findings reported in endurance athlete cohorts (5,8, 9).

As summarized in Table 1, both the prevalence and clinical presentation of GI symptoms differ across endurance disciplines, reflecting the distinct physiological demands associated with each sport.

3.2. Pathophysiological Mechanisms

Exercise-induced gastrointestinal symptoms are increasingly recognized as a consequence of disturbances in splanchnic circulation and intestinal barrier function. Within the Exercise-Induced Gastrointestinal Syndrome (EIGS) framework, prolonged endurance exercise initiates a series of circulatory and cellular responses that may ultimately impair gastrointestinal integrity and function (6).

3.2.1. Physiological and Ischemic Mechanisms

3.2.1.1. Splanchnic Hypoperfusion

During prolonged exercise, blood flow is redistributed away from the gastrointestinal tract toward exercising skeletal muscles and the skin to support metabolic demands and thermoregulation. This process is mediated by sympathetic nervous system activation and results in marked vasoconstriction within the splanchnic circulation (6; 10).

The magnitude of splanchnic hypoperfusion is related to exercise intensity and duration. Previous studies have demonstrated substantial reductions in gastrointestinal blood flow during prolonged exercise, particularly when exercise is performed at moderate-to-high intensities (10). During strenuous exercise, splanchnic blood flow may decrease by up to 60-80%, depending on exercise intensity and environmental conditions (10). Reduced perfusion limits oxygen and nutrient delivery to the intestinal mucosa, rendering enterocytes vulnerable to ischemic injury (6, 11).

Evidence from experimental studies indicates that exercise-induced reductions in splanchnic perfusion are associated with increased circulating concentrations of intestinal fatty acid-binding protein (I-FABP), a sensitive marker of enterocyte damage (12). The observed increase in I-FABP occurred during exercise and was accompanied by evidence of impaired intestinal function, suggesting that even transient reductions in intestinal blood flow may induce measurable mucosal injury (12).

Collectively, current evidence supports splanchnic hypoperfusion as a primary initiating event in the development of exercise-associated gastrointestinal disturbances (6, 12).

3.2.1.2. Epithelial Barrier Dysfunction

The intestinal epithelium forms a critical barrier that regulates the passage of nutrients, water, and electrolytes while limiting the translocation of luminal microorganisms and their products into the systemic circulation. Maintenance of this barrier depends on adequate mucosal perfusion and the integrity of intercellular tight junctions (6).

Exercise-induced ischemia can disrupt epithelial homeostasis and compromise barrier integrity. Within the EIGS framework, reductions in splanchnic blood flow promote enterocyte injury, which may subsequently increase intestinal permeability (6). Evidence suggests that intestinal permeability increases following strenuous running exercise, with greater permeability disturbances observed at higher exercise intensities (13).

In addition to ischemia, exercise-associated hyperthermia may further compromise epithelial barrier function. The combined effects of reduced splanchnic perfusion and elevated core temperature are considered key contributors to exercise-induced increases in intestinal permeability (6). Interestingly, nutritional interventions may partially attenuate these effects. Carbohydrate and protein ingestion during exercise performed under heat stress conditions has been associated with reduced intestinal epithelial injury and lower increases in small intestinal permeability, suggesting that nutritional strategies may help preserve barrier integrity despite ongoing physiological stress (25).

Pharmacological factors may further exacerbate exercise-induced gastrointestinal injury. In particular, NSAID use has been associated with greater epithelial damage and increased intestinal permeability during exercise, suggesting that commonly used medications may further compromise intestinal barrier integrity in endurance athletes (11).

Taken together, available evidence indicates that ischemia and hyperthermia contribute to epithelial barrier dysfunction, resulting in a transient increase in intestinal permeability, often referred to as 'leaky gut' (6).

3.2.1.3. Ischemia–Reperfusion Injury

Although reduced splanchnic blood flow is considered the primary trigger of exercise-induced intestinal injury, restoration of perfusion following exercise may also contribute to mucosal damage through ischemia-reperfusion mechanisms (10, 14).

Ischemia-reperfusion injury occurs when previously hypoxic tissues are re-exposed to oxygenated blood, leading to the generation of reactive oxygen species and activation of inflammatory pathways. These processes may exacerbate cellular injury beyond that caused by ischemia alone and have been implicated in the pathogenesis of gastrointestinal symptoms during strenuous exercise (10).

Reperfusion-associated oxidative stress may further disrupt epithelial barrier function and contribute to increased intestinal permeability. Consequently, intestinal injury during endurance exercise should be viewed as a dynamic process involving both the ischemic phase and subsequent reperfusion-related inflammatory responses (6, 14).

Within the EIGS model, ischemia-reperfusion injury represents an important link between exercise-induced hypoperfusion and downstream consequences, including impaired barrier function, translocation of luminal bacterial products, and activation of systemic inflammatory pathways (6).

Overall, available evidence supports the EIGS framework, in which exercise-induced splanchnic hypoperfusion serves as the primary initiating factor, while hyperthermia and ischemia-reperfusion responses act synergistically to promote epithelial barrier dysfunction and gastrointestinal dysfunction.

3.2.2. Mechanical Mechanisms

Mechanical stress has also been proposed as a contributing factor to exercise-induced gastrointestinal symptoms, particularly in endurance sports involving repetitive impact or prolonged postural constraints. While mechanical factors are likely to contribute to symptom development in some athletes, the supporting evidence remains less robust than that for ischemic mechanisms, and their relative importance may vary across endurance disciplines (5,9).

3.2.2.1. Repetitive Impact and Visceral Jostling

Long-distance running is associated with repetitive ground reaction forces that generate continuous movement of abdominal organs. This phenomenon, commonly described as "visceral jostling", has been suggested to contribute to the high prevalence of lower gastrointestinal symptoms observed in runners, including abdominal cramping, urgency to defecate, diarrhea, and, in rare cases, gastrointestinal bleeding (5, 9). Mechanical stress has been proposed as one of several factors contributing to gastrointestinal disturbances during endurance exercise, alongside exercise-induced hypoperfusion and thermal stress (8).

3.2.2.2. Posture and Position-Related Effects

In contrast to running, cycling is characterized by prolonged maintenance of a flexed aerodynamic posture. This position may increase intra-abdominal pressure and increase the likelihood of upper gastrointestinal symptoms such as gastroesophageal reflux, nausea, and epigastric discomfort (5, 9). Similar mechanisms may contribute to symptom development during triathlon events, particularly during the cycling segment. Furthermore, the ingestion of fluids and carbohydrates while maintaining a prolonged seated position may exacerbate sensations of gastric fullness and upper abdominal discomfort in susceptible athletes (9).

In summary, mechanical factors are likely to interact with circulatory, thermal, and nutritional stressors rather than act as independent causes of gastrointestinal dysfunction. Their contribution may therefore partly explain the sport-specific differences in gastrointestinal symptom patterns observed across endurance disciplines. (8, 9).

3.2.3. Nutritional and Pharmacological Triggers

Nutritional practices adopted before and during endurance exercise play a crucial role in the development and severity of gastrointestinal symptoms. While appropriate carbohydrate and fluid intake are essential for sustaining performance, certain dietary strategies may inadvertently exacerbate gastrointestinal distress. In addition, the use of commonly available pharmacological agents, particularly non-steroidal anti-inflammatory drugs (NSAIDs), may further compromise gastrointestinal integrity. Current evidence suggests that nutritional and pharmacological factors interact with exercise-induced circulatory and thermal stress, thereby amplifying the pathophysiological processes underlying EIGS (4, 6).

3.2.3.1. Hypertonic Carbohydrate Solutions

Carbohydrate supplementation during endurance exercise is widely recommended to maintain energy availability and delay fatigue. However, the composition and concentration of ingested carbohydrate products may substantially influence gastrointestinal tolerance (4, 15).

Highly concentrated carbohydrate beverages and gels can increase the osmotic load within the gastrointestinal lumen, potentially delaying gastric emptying and promoting fluid shifts from the circulation into the intestinal lumen (4). These mechanisms may contribute to symptoms such as abdominal bloating, nausea, abdominal discomfort, and diarrhea, particularly when large quantities of carbohydrates are consumed without adequate fluid intake (4).

Higher carbohydrate intakes during competition have been associated with an increased prevalence of gastrointestinal symptoms, suggesting that excessive carbohydrate consumption may contribute to symptom development in susceptible individuals (7). Nevertheless, gastrointestinal tolerance appears to be highly individualized and may be improved through repeated exposure and gastrointestinal adaptation (15).

3.2.3.2. FODMAPs

Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) represent a group of short-chain carbohydrates that are poorly absorbed in the small intestine and readily fermented by colonic bacteria. Their osmotic activity increases luminal water content, while bacterial fermentation promotes gas production and intestinal distension (16, 17).

These physiological effects may be particularly problematic during endurance exercise, when gastrointestinal function is already compromised by exercise-induced hypoperfusion and altered motility. Consequently, high dietary FODMAP intake may exacerbate symptoms such as bloating, abdominal pain, flatulence, and diarrhea in susceptible athletes (16,17).

Preliminary evidence suggests that short-term low-FODMAP dietary interventions may reduce exercise-associated gastrointestinal symptoms in susceptible athletes. Reductions in gastrointestinal symptom burden during training and competition have been reported following implementation of a low-FODMAP dietary approach, although the overall evidence base remains limited and further research is required (16, 17,18).

Consistent with these findings, surveys of endurance athletes indicate that many individuals voluntarily restrict foods perceived to trigger gastrointestinal symptoms, including several foods rich in FODMAPs, particularly in the days preceding competition (19).

3.2.3.3. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

The use of NSAIDs is common among endurance athletes seeking to manage pain and exercise-related musculoskeletal discomfort. However, accumulating evidence suggests that these medications may adversely affect gastrointestinal function and increase susceptibility to exercise-induced intestinal injury (6, 14).

NSAIDs impair mucosal defense mechanisms through inhibition of cyclooxygenase enzymes and reduced prostaglandin synthesis, thereby compromising intestinal blood flow and epithelial protection (14).

When combined with exercise-induced splanchnic hypoperfusion, these effects may further disrupt epithelial barrier integrity and increase intestinal permeability (6).

Ibuprofen administration before exercise has been associated with greater exercise-induced intestinal injury and permeability disturbances compared with exercise alone (14). Athletes using NSAIDs have been shown to exhibit greater epithelial damage and permeability disturbances, supporting the hypothesis that pharmacological and exercise-related stressors act synergistically at the level of the intestinal mucosa (14).

Accordingly, current evidence identifies NSAID use as a modifiable risk factor for exercise-induced gastrointestinal dysfunction. Consequently, avoidance of unnecessary NSAID use before prolonged endurance events is increasingly recommended as part of gastrointestinal symptom prevention strategies (6).

Overall, nutritional and pharmacological factors should be viewed as important modifiers of gastrointestinal function during endurance exercise. By interacting with exercise-induced ischemic and thermal stress, hypertonic carbohydrate solutions, high-FODMAP foods, and NSAID use may substantially increase the likelihood and severity of gastrointestinal symptoms in endurance athletes.

3.2.4. The Brain–Gut–Exercise Axis and Psychophysiological Stress

Exercise-induced gastrointestinal symptoms cannot be fully explained by circulatory, mechanical, and nutritional factors alone. Increasing evidence suggests that bidirectional communication between the central nervous system and the gastrointestinal tract, commonly referred to as the gut-brain axis, contributes to the development and modulation of gastrointestinal symptoms during endurance exercise (20, 21, 22). Within the context of endurance sports, this interaction may be viewed as part of a broader brain-gut-exercise axis.

The gut-brain axis comprises a complex network involving the central and autonomic nervous systems, the hypothalamic-pituitary-adrenal (HPA) axis, the enteric nervous system, immune mediators, and the intestinal microbiota. Through these interconnected pathways, psychological and physiological stressors can directly influence gastrointestinal motility, secretion, visceral sensitivity, and epithelial barrier function (20, 21, 22).

In endurance athletes, psychophysiological stress may develop both before and during competition. Pre-race anxiety, competitive pressure, environmental stressors, and prolonged physical exertion may activate the sympathetic nervous system (SNS) and the HPA axis, leading to increased secretion of catecholamines and cortisol (9, 23). These neuroendocrine responses may alter gastrointestinal function by delaying gastric emptying, modifying intestinal motility, and lowering the threshold for visceral hypersensitivity. Consequently, athletes may experience symptoms such as nausea, abdominal discomfort, bloating, urgency to defecate, or diarrhea even in the absence of severe structural gastrointestinal injury (23).

Psychological factors may also contribute to the considerable inter-individual variability observed in exercise-induced gastrointestinal symptoms. Athletes exposed to similar physiological workloads often report markedly different symptom severity, suggesting that cognitive and emotional factors influence both symptom perception and overall symptom burden. Anxiety, attentional focus, previous negative gastrointestinal experiences, and symptom expectation may amplify gastrointestinal symptom perception during exercise (23). However, the precise contribution of psychophysiological stress remains difficult to quantify, as much of the available evidence is derived from observational studies and conceptual models rather than controlled intervention trials (23).

Current conceptual models suggest that neuroendocrine pathways may interact with exercise-induced circulatory disturbances rather than acting independently. Psychological stress may exacerbate the physiological consequences of exercise-induced hypoperfusion and hyperthermia, thereby contributing to gastrointestinal dysfunction and symptom development (24). Furthermore, growing evidence suggests that gastrointestinal barrier function and gut-brain signaling are closely interconnected, emphasizing the complex interaction between physiological and psychological factors involved in the development of exercise-induced gastrointestinal symptoms (20, 21, 22, 23).

Collectively, exercise-induced gastrointestinal symptoms appear to result from the complex interplay of ischemic, mechanical, nutritional, and neuroendocrine mechanisms. The relative contribution of each pathway varies according to individual and environmental factors, contributing to the heterogeneous symptom profile observed among endurance athletes (6, 22, 23).

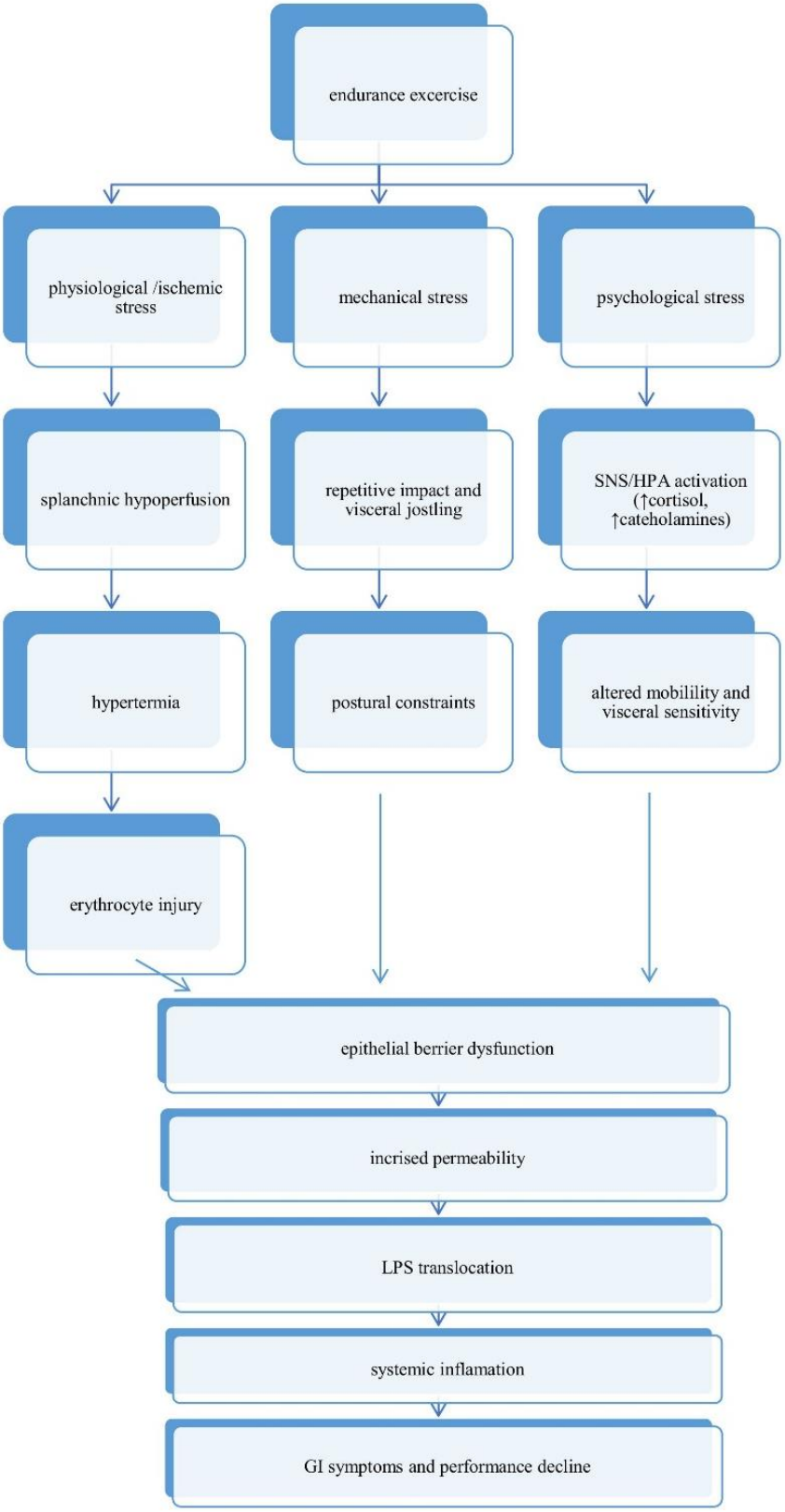

Fig. 1. *Integrated Pathophysiological Mechanisms of Exercise-Induced Gastrointestinal Syndrome (EIGS)*

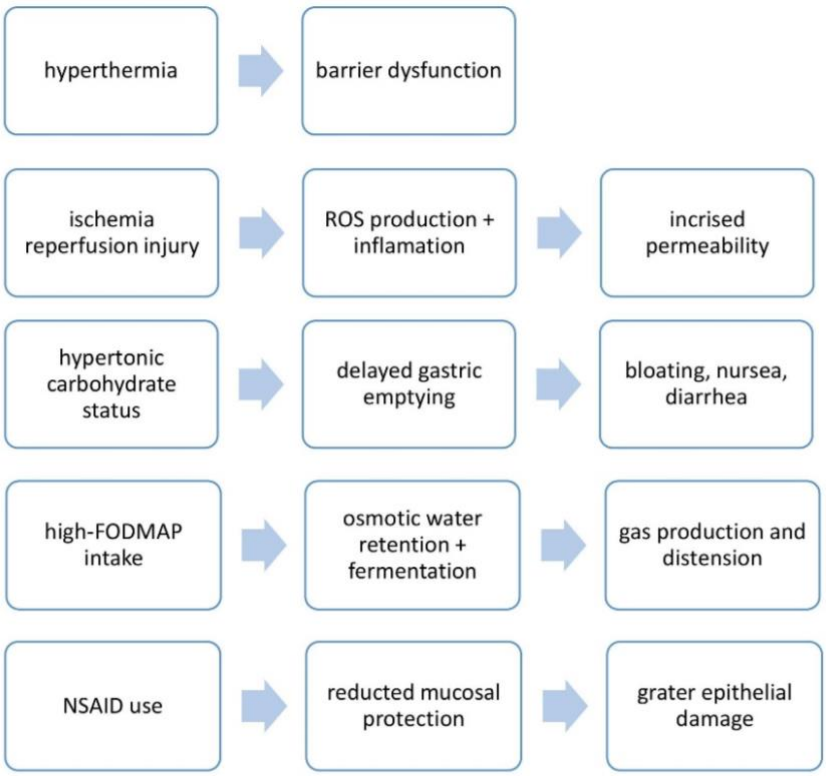

Fig. 2. *Factors Modulating the Development and Severity of Exercise-Induced Gastrointestinal Symptoms in Endurance Athletes.*

3.3. Systemic Consequences of GI Distress

Exercise-induced gastrointestinal distress can have consequences that extend beyond local digestive symptoms, contributing to systemic physiological disturbances that may impair performance and recovery. One important mechanism is increased intestinal permeability ("leaky gut"), which develops secondary to splanchnic hypoperfusion, hyperthermia, and mechanical stress during prolonged endurance exercise (26). Disruption of the intestinal epithelial barrier facilitates the translocation of luminal bacterial components, particularly lipopolysaccharide (LPS), into the circulation, resulting in exercise-associated endotoxemia. This process activates innate immune pathways and stimulates a systemic inflammatory cascade, marked by elevated circulating pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) (27). Although transient increases in circulating endotoxin may be clinically insignificant in many athletes (28), severe or repeated episodes have been associated with exaggerated inflammatory responses and severe fatigue (6,29), as well as an increased risk of exertional heat illness and multi-organ strain via systemic inflammatory cascades (30,31,32).

In addition to promoting systemic inflammation, gastrointestinal dysfunction compromises nutrient and fluid absorption at a time when substrate delivery is critical. Damage to the intestinal mucosa and accelerated transit time reduce the absorption of carbohydrates, electrolytes, and water. Malabsorption of ingested carbohydrates increases intraluminal osmotic pressure and enhances colonic fermentation. Together, these mechanisms accelerate gas production, abdominal cramping, and osmotic diarrhea, further exacerbating gastrointestinal distress and rendering standard fueling strategies (e.g., 60-90 g/h of carbohydrates) impossible to maintain (14,15). Concurrent impairment of fluid and electrolyte uptake contributes to dehydration and disturbances in electrolyte balance, which negatively affect cardiovascular function, thermoregulation, and neuromuscular performance. Consequently, the combination of endotoxemia, systemic inflammation, malabsorption, and dehydration creates a self-perpetuating cycle that amplifies physiological strain, reduces endurance performance, and increases susceptibility to exertional heat-related illness (33).

3.4. Management and Mitigation Strategies

3.4.1. Nutritional Conditioning ("Gut Training")

The concept of "gut training" has emerged as a promising strategy to reduce exercise-induced gastrointestinal symptoms while enhancing carbohydrate delivery during prolonged endurance exercise (34). Repeated exposure to carbohydrate ingestion during training sessions is thought to induce physiological adaptations, including accelerated gastric emptying, increased intestinal carbohydrate transporter expression, improved absorptive capacity, and enhanced gastrointestinal tolerance (34,37). Regular practice of race-day fueling protocols may also reduce subjective symptoms such as nausea, bloating, and abdominal discomfort by promoting habituation to fluid and carbohydrate intake under exercise conditions. Although current evidence suggests that gut training can improve both gastrointestinal comfort and exogenous carbohydrate oxidation, optimal training duration, carbohydrate doses, and individual responsiveness remain areas of ongoing investigation (38,39). Upregulation of intestinal carbohydrate transporters such as SGLT1 and GLUT5 has been proposed as one of the mechanisms underlying these adaptations.

3.4.2. In-Competition Nutritional Strategies

Acute nutritional strategies during competition should aim to maximize energy availability while minimizing gastrointestinal stress. Individualized carbohydrate intake, selection of well-tolerated carbohydrate sources, and gradual progression toward higher carbohydrate intakes may reduce the risk of gastrointestinal symptoms (29,34). The use of multiple transportable carbohydrates (e.g., glucose-fructose mixtures) can enhance carbohydrate absorption and reduce intestinal carbohydrate accumulation compared with single-source carbohydrates when consumed in appropriate quantities (37). Adequate hydration and electrolyte replacement are equally important, particularly in hot environments where dehydration may exacerbate splanchnic hypoperfusion and intestinal permeability (12,41). Conversely, excessive fluid intake should be avoided to reduce the risk of gastrointestinal discomfort and exercise-associated hyponatremia (42,43). Athletes with recurrent symptoms may also benefit from avoiding high-fiber, high-fat, or highly concentrated hyperosmolar foods in the hours preceding competition and from trialing all nutritional strategies during training rather than implementing them for the first time on race day (4).

3.4.3. Pharmacological and Supplemental Interventions

Several pharmacological and nutritional supplements have been investigated for the prevention or treatment of exercise-induced gastrointestinal symptoms, although evidence supporting their routine use remains limited. Antidiarrheal agents such as loperamide may reduce diarrhea in selected circumstances but are not generally recommended for routine prophylactic use because of potential adverse effects and concerns regarding altered thermoregulation (29,35). While acid-suppressive therapies, including proton pump inhibitors and antacids, are indicated for managing clinically confirmed gastroesophageal reflux disease (GERD) in athletes, their empirical or non-targeted administration should be avoided due to potential secondary impacts on nutrient absorption and gastrointestinal kinetics (40, 41). Among nutritional supplements, probiotics have received considerable attention for their potential to improve intestinal barrier integrity and reduce gastrointestinal symptoms, although findings remain inconsistent across strains and study populations (42). Similarly, glutamine, bovine colostrum, nitrate supplementation, and antioxidant compounds have been explored for their potential to attenuate exercise-induced increases in intestinal permeability and systemic inflammation, but current evidence is insufficient to support widespread recommendations (43). Overall, pharmacological and supplemental interventions should be considered adjunctive to, rather than substitutes for, individualized nutritional strategies and appropriate training adaptations. In addition pre-race nutritional and behavioral protocols are commonly implemented to minimize the risk of gastrointestinal disturbances and optimize athletic performance. These protocols typically include the avoidance of nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol consumption, and high-fiber foods during the acute pre-competition period (4). A summary of the recommended timing, objectives, and strategies for gastrointestinal preparation and management before, during, and after competition is presented in Table 2.

Table 2. Recommended timing, objectives, and strategies for gastrointestinal preparation and management.

Time period	Objective	Recommended strategy
2-4 weeks before competition	Gut adaptation	Gut training with progressively increasing carbohydrate intake during exercise (>60-90 g/h)
24-72 h before competition	Reduce fermentable substrate load	Low-FODMAP diet, reduction of excess dietary fiber, avoidance of NSAIDs
2-4 h before start	Optimize gastric emptying	Low-fat, low-fiber pre-race meal; individualized carbohydrate intake
During competition	Maximize nutrient absorption	Multiple transportable carbohydrates (glucose-fructose blends), individualized fluid replacement
During competition (if symptoms occur)	Symptom management	Reduce exercise intensity, adjust fluid/carbohydrate intake, cooling strategies when appropriate
Recovery period	Restore GI integrity	Rehydration, carbohydrate replenishment, avoidance of unnecessary NSAID use

4. Future Directions

Despite substantial advances in the understanding of exercise-induced gastrointestinal syndrome (EIGS), several important knowledge gaps remain. Much of the current evidence is derived from controlled laboratory-based studies, whereas gastrointestinal symptoms most commonly occur under real-world competition conditions characterized by prolonged exercise duration, environmental stress, and complex nutritional practices. Consequently, there is a need for more field-based investigations that better reflect the physiological demands encountered during endurance events.

Considerable inter-individual variability in symptom susceptibility and severity also remains poorly understood. Emerging evidence suggests that differences in gut microbiota composition, intestinal barrier function, nutritional tolerance, and psychological responses to exercise may influence the development of gastrointestinal symptoms. Future research should therefore focus on identifying factors that contribute to individual susceptibility and on developing personalized prevention and management strategies.

In addition, female athletes remain underrepresented in the current literature. Given the potential influence of sex-specific physiological factors, including hormonal fluctuations, gastrointestinal transit characteristics, and differences in substrate metabolism, further studies are required to clarify whether the prevalence, mechanisms, and optimal management of exercise-induced gastrointestinal symptoms differ between male and female endurance athletes.

5. Conclusions

Exercise-induced gastrointestinal symptoms are highly prevalent among endurance athletes and represent an important challenge for both health and performance. Current evidence indicates that gastrointestinal disturbances arise through the interaction of multiple mechanisms, including exercise-induced splanchnic hypoperfusion, epithelial barrier dysfunction, ischemia-reperfusion injury, mechanical stress, nutritional factors, and neuroendocrine responses. Together, these pathways contribute to gastrointestinal dysfunction, increased intestinal permeability, systemic inflammatory responses, and impaired exercise performance.

Although no single intervention can completely eliminate gastrointestinal symptoms, several evidence-based strategies may reduce symptom burden and improve gastrointestinal tolerance during exercise. Among the most promising approaches are gut training, individualized carbohydrate and fluid intake strategies, short-term low-FODMAP dietary interventions in susceptible athletes, and avoidance of unnecessary NSAID use before competition. Continued advances in the understanding of EIGS pathophysiology will facilitate the development of more targeted and individualized prevention strategies, ultimately supporting both athlete health and endurance performance. Future studies should also investigate the role of gut microbiota composition and microbiota-targeted interventions in modulating susceptibility to EIGS.

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Conceptualization: KP, AK and LS
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Software: AK;
Check: NS, AD and; DT
formal analysis: NS;
investigation: NW;
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writing - rough preparation: NZ;
writing - review and editing: AK;
visualization: NZ and LS;
supervision: JR;
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