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

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IMPACT OF LONG DURATION HIGH-INTENSITY PHYSICAL EXERCISE ON SMALL AND LARGE INTESTINAL FUNCTION IN COMPETITIVE ATHLETES – A LITERATURE REVIEW

Michalina Pastuszka (Corresponding Author, Email: michalina.pastuszka10@wp.pl)

Medical University of Lublin, Lublin, Poland

ORCID ID: 0009-0006-6531-5021

Kamil Nieroda

Medical University of Lublin, Lublin, Poland

ORCID ID: 0009-0004-0923-2630

Bartłomiej Andrzej Sałapski

Jan Kochanowski University, Kielce, Świętokrzyskie, Poland

ORCID ID: 0009-0008-8366-1272

Aleksandra Małgorzata Obarzanek

Jan Kochanowski University, Kielce, Świętokrzyskie, Poland

ORCID ID: 0009-0004-6635-9849

Michał Nowak

Medical University of Łódź, Łódź, Poland

ORCID ID: 0009-0006-2995-3210

Natalia Julia Szafranec

Szpital Kielecki Św. Aleksandra, Kielce, Poland

ORCID ID: 0009-0001-7544-4007

Dominika Brożyna

Jan Kochanowski University, Kielce, Świętokrzyskie, Poland

ORCID ID: 0009-0002-1542-6904

Katarzyna Anna Sobczyk

Szpital Kielecki Św. Aleksandra, Kielce, Poland

ORCID ID: 0009-0000-8740-5016

ABSTRACT

Long-duration, high-intensity endurance exercise places unique stress on the gastrointestinal tract. In competitive endurance disciplines such as triathlon, repeated exposure to prolonged exertion, heat strain, and dehydration can disturb normal gastrointestinal physiology. A key response to intense exercise is redistribution of blood flow away from the splanchnic circulation toward working muscles and thermoregulatory tissues, which may reduce intestinal perfusion. When this reduction is marked or sustained, it can contribute to mucosal injury, and subsequent restoration of blood flow may further intensify damage through inflammatory and oxidative processes. These mechanisms can weaken epithelial tight junctions, increase intestinal permeability, and promote translocation of luminal microbial products, potentially amplifying systemic inflammatory responses. Clinically, such changes are reflected by the high prevalence of gastrointestinal symptoms during training and competition, which may limit fueling tolerance and impair performance. In rare cases, more severe outcomes such as ischemic colitis or gastrointestinal bleeding may occur.

The aim of this narrative review is to summarize current evidence on how long-duration, high-intensity endurance exercise affects small and large intestinal perfusion, epithelial barrier integrity, and gut microbial ecology in competitive athletes, and to discuss the practical significance of these alterations for symptoms, performance, and adverse gastrointestinal events.

Results: Prolonged high-intensity endurance exercise markedly reduces splanchnic perfusion, predisposing the intestine to hypoperfusion and ischemia–reperfusion–related oxidative and inflammatory injury. Therefore, epithelial integrity may be compromised through tight junction disruption and enterocyte damage, resulting in increased intestinal permeability (“leaky gut”) and facilitating translocation of luminal bacterial components with subsequent immune activation. In addition, repeated endurance training and sport-specific dietary patterns appear to modulate gut microbiota composition and function, and some exercise-associated microbial shifts may correlate with metabolic adaptation and endurance performance.

Materials and methods: reviewing recent literature research up to 15 years ago based PubMed, Google scholar reaserche based on following key words: leaky gut syndrome in athletes, gut microbiota in athletes, ischemic colitis in athletes.

KEYWORDS

Gut Microbiota In Athletes, Splanchnic Hypoperfusion, Intestinal Permeability, Endurance Athletes

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1. Introduction

The lower gastrointestinal (GI) tract plays a central role in digestion and absorption of macro- and micronutrients, processes that are necessary to maintain health and support athletic performance. Beyond nutrient handling, the GI tract contributes to systemic regulation through immune signaling, inflammatory balance, and neuroendocrine communication within the gut-brain axis, which may indirectly affect recovery, illness susceptibility, and performance [1]. The gut microbiome is a major component of this system and can be influenced by everyday factors including diet, physical activity, and antibiotic exposure [5][6].

Despite its relevance, the GI tract has historically received less research attention in sports science than other physiological systems. This gap is notable because endurance sports such as triathlon combine prolonged exertion, heat stress, dehydration risk, and repeated nutritional challenges—conditions that can acutely disrupt intestinal physiology. Recent work suggests that athletes may exhibit distinct microbiome signatures compared with non-athletes, reflecting sport-specific metabolic and environmental pressures [5][6]. This narrative review focuses on the impact of long-duration high-intensity exercise on intestinal perfusion, epithelial integrity (“leaky gut”), and gut microbiota in competitive endurance athletes.

2. Intestinal perfusion and ischemia-reperfusion injury

2.1 Exercise-induced redistribution of blood flow

At rest, a considerable portion of cardiac output is delivered to the splanchnic organs via the celiac, superior mesenteric, and inferior mesenteric arteries [4]. During exercise, sympathetic activation and reduced vagal tone redirect blood flow to working muscle and skin (thermoregulation), reducing splanchnic perfusion. In a classic human study, superior mesenteric artery blood flow decreased by approximately 43% immediately after treadmill exercise (5 km/h, 20% gradient, 15 minutes) [3]. These findings support the concept that intestinal hypoperfusion is a common physiological response to exertion and may occur even in healthy individuals.

2.2 Progression to injury: ischemia and reperfusion

When exercise is intense and prolonged—especially when compounded by dehydration, hyperthermia, or increased blood viscosity—hypoperfusion may become severe enough to impair mucosal function. Importantly, restoration of splanchnic blood flow after exercise can trigger ischemia-reperfusion injury (IRI), characterized by oxidative stress and inflammatory activation [4]. IRI may contribute to epithelial damage, loss of barrier integrity, and local inflammatory changes, setting the stage for increased permeability and GI symptoms.

2.3 Colon vulnerability and clinically significant events

The colon may be particularly susceptible to hypoperfusion due to relatively lower blood supply per gram of tissue and vulnerable “watershed” areas. Severe outcomes, while uncommon, are reported in athletic settings. Case literature describes ischemic colitis associated with intense exertion, including presentations linked with heat stroke and rhabdomyolysis [2]. Reviews focusing on athletes also document gastrointestinal bleeding events attributed to exertional ischemia, with rare cases requiring surgical intervention [4]. Although these events are not typical for most athletes, they illustrate that exercise-induced perfusion changes can cross into clinically significant pathology in susceptible contexts.

3. Exercise-associated intestinal permeability (“leaky gut”)

3.1 Symptom prevalence and performance relevance

Gastrointestinal complaints are frequent in endurance sports. Observational research in long-distance runners, cyclists, and triathletes reports a high prevalence of symptoms during or shortly after events, which can compromise fueling strategies and performance [18]. While symptoms are often transient, they reflect underlying physiological stress affecting the intestinal barrier.

3.2 Tight junction disruption and epithelial injury

A central mechanism of “leaky gut” is impairment of epithelial tight junctions (TJs), which regulate paracellular permeability between enterocytes. Heat and exercise stress can alter TJ protein expression and localization, including proteins such as occludin, claudins, and ZO-1, weakening the barrier and increasing permeability [11][15]. Mechanistic reviews describe how strenuous exercise can disrupt intestinal integrity through combined effects of hypoperfusion, thermal load, metabolic strain, and inflammatory signaling [13][17].

Controlled studies also support a direct link between reduced splanchnic perfusion and measurable gut dysfunction. Exercise-induced splanchnic hypoperfusion has been shown to induce functional markers of gut injury in healthy participants, consistent with perfusion-driven epithelial stress [16].

3.3 Inflammatory activation and luminal translocation

When permeability increases, luminal components—including bacterial products—may cross the epithelial barrier and activate innate immune pathways. This can amplify inflammatory signaling and potentially worsen epithelial disruption, creating a self-reinforcing cycle during or after prolonged exertion [14][17]. The clinical expression of this process varies and is strongly influenced by environmental heat, hydration status, exercise intensity, and individual tolerance to nutrition and fluids during activity [17].

3.4 Nutritional strategies and emerging interventions

Given the performance impact of GI symptoms, nutritional interventions are of high interest. Acute oral glutamine supplementation has been studied as a potential strategy to attenuate exercise-induced increases in permeability, though translation to standardized athlete guidance requires careful, individualized interpretation [12]. In practice, any GI-focused strategy should be tested during training and adapted to sport modality and conditions rather than introduced for the first time during competition.

4. Gut microbiota in endurance athletes

4.1 Functional relevance of the microbiome

The gut microbiota contributes to host metabolism by fermenting nondigestible substrates into short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate, which can influence energy balance and immunometabolic regulation [5]. Current reviews suggest that “health-associated” microbiota is a more appropriate concept than a single “healthy” composition, as multiple taxonomic profiles may support similar functional outputs due to metabolic redundancy [6]. Moreover, the gut microbiome shows partial long-term stability in adults, although inter-individual variation remains substantial [7].

4.2 Diet-training interactions in endurance sport

Endurance athletes commonly consume high amounts of carbohydrates to support training and competition demands [8]. Alongside high weekly training volume, this dietary pattern may shape microbial ecology and metabolic capacity [5][6]. Evidence from elite athletes indicates that microbiome composition and metabolomic profiles can differ across athlete subgroups, likely reflecting combined influences of training load, diet, environment, and recovery practices [9].

4.3 A mechanistic example: Veillonella and lactate metabolism

A notable line of evidence links endurance exercise with enrichment of *Veillonella* species. A metabolomics study reported increased abundance of *Veillonella* after marathon running and demonstrated that inoculating mice with *Veillonella atypica* isolated from post-race samples improved treadmill performance. The proposed mechanism involves utilization of exercise-generated lactate and production of propionate, a metabolite with potential relevance to endurance physiology [10]. While these findings do not yet support routine clinical “microbiome optimization” for performance, they demonstrate that microbial pathways may plausibly interact with host metabolism in an exercise-dependent manner.

5. Practical implications for athletes and support teams

Intestinal perfusion matters: high intensity and long duration, especially with heat or dehydration, can meaningfully reduce splanchnic blood flow and contribute to epithelial injury [3][4][16].

Barrier disruption is a key pathway: heat stress and exercise strain can weaken tight junctions and increase permeability, which is associated with symptoms and inflammatory activation [11][13][15][17]. Serious complications exist but are uncommon: ischemic colitis and GI bleeding have been described in athletes, highlighting the need for awareness in high-risk contexts [2][4].

Microbiome is relevant but not yet “prescribable”: training and diet shape microbiota; emerging work suggests performance-linked microbial mechanisms, but clinical translation remains preliminary [5][6][10].

Individualization is essential: symptom patterns and triggers differ across athletes and sports; nutrition and supplementation strategies should be tested in training and adjusted to environmental conditions [12][17][18].

6. Limitations of current evidence

Available literature includes heterogeneous study designs (laboratory physiology, observational cohorts, case reports, and reviews), with variability in exercise protocols, environmental conditions, nutrition exposure, and symptom definitions. Microbiome studies are also limited by inter-individual variability, sampling timing, and the challenge of separating training effects from dietary and lifestyle confounders [6][7]. Therefore, evidence supports plausible mechanistic frameworks, but individualized responses remain difficult to predict.

7. Discussion

This narrative review suggests that the gastrointestinal tract should be viewed as a performance-relevant organ in endurance sport. The central mechanism linking long-duration high-intensity exercise with intestinal dysfunction is reduced splanchnic perfusion, which may progress from transient hypoperfusion to mucosal injury. When blood flow is restored, reperfusion-related oxidative and inflammatory responses can further compromise epithelial stability. These effects are commonly amplified by dehydration and heat stress, which are frequent in long events and can increase physiological strain on the intestinal wall.

A key implication is that exercise-associated GI symptoms arise from multiple interacting factors. Hemodynamic stress initiates vulnerability, while thermal and metabolic strain contribute to tight junction disruption and enterocyte damage, increasing intestinal permeability (“leaky gut”). This may worsen symptoms and facilitate translocation of luminal bacterial components, promoting immune activation. Although most athletes experience only short-lived discomfort, the same pathway provides a plausible

continuum toward more severe outcomes in susceptible individuals or extreme conditions. Therefore, distinguishing typical self-limited symptoms from atypical presentations (e.g., severe pain, persistent symptoms, or visible blood) is clinically important. The review also highlights that endurance training and sport-specific nutrition can influence gut microbiota composition and function. However, microbiome findings should currently be interpreted cautiously, as inter-individual variability and dietary confounding limit causal conclusions. At present, microbiota research is best viewed as mechanistic support rather than a basis for standardized performance interventions. Practical prevention strategies align with these mechanisms and include individualized hydration and heat mitigation, as well as systematic “gut training” to improve tolerance of race nutrition. Future studies should use more ecologically valid designs that integrate heat, dehydration, and real-world fueling, and should link physiological markers to meaningful outcomes such as symptom burden, recovery, and performance.

8. Conclusions

Long-duration, high-intensity endurance exercise can transiently impair small and large intestinal function through splanchnic hypoperfusion, heat stress, oxidative and inflammatory injury, and disruption of epithelial tight junctions. These mechanisms contribute to the high prevalence of GI symptoms in endurance of athletes and help explain rare but important complications such as ischemic colitis and gastrointestinal bleeding. In parallel, endurance training and sport nutrition interact with the gut microbiota, which may influence metabolic pathways relevant to endurance performance. Future athlete-focused studies should refine risk stratification and evaluate interventions that protect gut integrity without compromising fueling or training adaptation.

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