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Exercise-induced Gastrointestinal Syndrome in Triathletes – a Systematic Review

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Abstract**Introduction and Purpose**

In recent decades, there has been a significant increase in interest in triathlon among amateur athletes. Despite the undeniable health benefits, participating in such a demanding discipline also carries numerous issues, such as injuries and gastrointestinal disorders. According to some studies, as many as 93% of individuals competing in triathlon events report symptoms such as nausea, abdominal pain, or diarrhea during exertion. This paper is devoted to examining these issues.

Aim of the study

We have discussed the most important pathophysiological mechanisms that lead to them, such as splanchnic ischemia, damage to the intestinal epithelium, and increased permeability of intercellular junctions in the intestines.

Methods

The following paper analyzed studies found on PubMed using keywords such as “exercise-induced gastrointestinal syndrome”, “gastrointestinal disorder in triathlon”, “EIGS management”, and “triathlon”. Articles include randomized controlled trials, meta-analyses,

and systematic reviews published from 1990 to the present day, prioritizing resources published after 2020.

exercise-induced gastrointestinal syndrome, gastrointestinal disorder in triathlon, EIGS management, triathlon

Conclusions

Unfortunately, despite numerous studies being conducted, there are still no sufficiently effective methods to prevent these ailments. According to current data, the most effective measures include reduced fiber intake in the pre-competition period and gut training to prepare them for such exertion.

Keywords

exercise-induced gastrointestinal syndrome, gastrointestinal disorder in triathlon, EIGS management, triathlon

Introduction

Triathlon is a multidisciplinary sport consisting of three consecutive segments: swimming, cycling, and running. The most prestigious format is the Ironman distance, comprising 3.8 km of swimming, 180 km of cycling, and a 42.195 km marathon run. In addition to the full-distance Ironman, numerous events are held over shorter distances such as the half (1/2), quarter (1/4), and eighth (1/8) Ironman, as well as longer ultradistance formats, including triple and quintuple Ironman races (1). The broad range of race distances and the frequent organization of events have contributed in recent years to a significant increase in the sport's popularity, particularly among recreational and amateur athletes (2). However, due to commonly observed training errors and the lack of regular supervision by physiotherapists and dietitians, this population is particularly vulnerable to various health issues associated with the high physical demands of the sport (3).

Exercise-induced Gastrointestinal Syndrome (EIGS) is a prevalent condition encountered during competitions and training among triathletes and other endurance athletes. The symptoms associated with this syndrome arise from both the upper and lower gastrointestinal tracts and may include abdominal pain, vomiting, diarrhea, and, in some cases, bleeding (4). Current estimates suggest that such gastrointestinal complaints may affect as many as 93% of triathletes and long-distance runners (5). The frequency of these symptoms is significantly influenced by

the nature of the exertion undertaken, with heightened prevalence reported during running compared to cycling. Additionally, the duration of the activity plays a crucial role; as the effort prolongs, the likelihood of symptom occurrence increases. For this reason, during races at distances such as Ironman 140.6 and marathons, the reported disorders frequently occur. According to some data, they may account for approximately 35% of unfinished races (6). Currently, due to the growing popularity of endurance sports and competitions, numerous studies have been conducted regarding the etiology, pathophysiology, and management of these disorders. Given the extensive range of diverse symptoms, it is suspected that several mechanisms may be responsible for the reported ailments. The most frequently described and best-documented mechanisms are splanchnic hypoperfusion leading to ischemia, damage to the epithelial barrier associated with the gut microbiome, and mechanical injuries to the epithelial lining of the gastrointestinal tract related to motility. These mechanisms typically coexist, and the activation of the sympathetic nervous system, as well as the hypothalamic-pituitary-adrenal (HPA) axis, also plays a crucial role. Additionally, the nutritional strategy implemented during exercise significantly impacts these mechanisms (7). Consequently, in recent years, efforts have been made to establish the most rational dietary practices and to develop strategies aimed at preventing the described disorders. This review aims to explain the potential pathomechanisms underlying exercise-induced gastrointestinal syndrome (EIGS) and, based on the latest literature, to describe how to counteract them.

Symptoms

Gastrointestinal symptoms are commonly categorized into those affecting the upper and lower segments of the gastrointestinal tract. Upper gastrointestinal symptoms typically include reflux, belching, regurgitation, vomiting, and retrosternal burning sensations. In contrast, lower gastrointestinal symptoms encompass abdominal pain, bloating, urgency to defecate, diarrhea, flatulence, and gastrointestinal bleeding (8). In a survey-based study by Peters et al., triathletes reported the occurrence of gastrointestinal symptoms slightly more frequently during training sessions than during competition. Among the three triathlon segments, symptoms were least commonly reported during the swim. During the cycling phase, upper gastrointestinal symptoms—such as nausea, belching, or reflux—were observed more frequently than lower gastrointestinal complaints (52% vs. 47%) (9). This may be attributed to the aerodynamic position assumed on a triathlon bicycle, which increases intra-abdominal pressure and may predispose athletes to upper GI discomfort (8). In contrast, the running segment was associated with a markedly higher prevalence of lower gastrointestinal symptoms, including cramping, urgency, and diarrhea (49% vs. 95%). This phenomenon may result from repetitive mechanical

forces acting on the gastrointestinal tract during running, as well as the cumulative physiological strain experienced by the athlete toward the end of prolonged exertion (9).

Mechanisms

Splanchnic hypoperfusion and ischemia

The arterial supply to the organs of the gastrointestinal system is primarily provided by three major branches of the abdominal aorta: the celiac trunk and the superior and inferior mesenteric arteries. The venous circulation is facilitated by the portal system. The splanchnic blood flow typically amounts to 30 ml/min per 100 grams of tissue, which constitutes approximately 25% of the cardiac output (10). This flow undergoes significant variations under the influence of the endocrine system and humoral factors. During exercise, the adrenal glands release a substantial amount of norepinephrine, which, by acting on alpha-adrenergic receptors, leads to the constriction of splanchnic vessels, resulting in an increase in vascular resistance (11,12). Moreover, during intense physical exercise, hyperventilation occurs, leading to a decrease in partial pressure of CO₂, which in turn contributes to vasoconstriction (13). This results in a significant reduction in splanchnic blood flow and an increased volume of blood delivered to the most active organs, such as the lungs, heart, muscles, and skin (14). Research conducted by Ter Steege demonstrated that the decrease in blood flow was proportional to the intensity of exercise, measured as lactate concentration. In athletes presenting with gastrointestinal symptoms, a significant reduction in splanchnic blood flow was observed even during submaximal exercise, a phenomenon not evident in asymptomatic individuals (14,15). A reduction in blood flow results in hypoxia of gastrointestinal cells, which promotes the development of localized inflammation and the release of reactive oxygen species (ROS), further contributing to cellular injury (16).

Damage of epithelial barrier

The intestinal epithelium is composed of a single layer of specialized epithelial cells that include enterocytes, goblet cells, Paneth cells, and enteroendocrine cells. This cellular layer forms a physical and biochemical barrier between the host and the intestinal lumen. It plays a crucial role in protecting the host from microbial invasion by regulating the passage of antigens and microorganisms, producing antimicrobial peptides, and maintaining tight junction integrity. Additionally, it interacts dynamically with the immune system to modulate responses to commensal and pathogenic bacteria (17).

The aforementioned reduction in splanchnic blood flow during exercise leads to intestinal hypoxia, which contributes directly to the disruption of this epithelial barrier. Ischemia-induced injury of the intestinal lining can be monitored by assessing the serum concentration of

intestinal fatty acid-binding protein (I-FABP), a low-molecular-weight protein predominantly localized in the upper half of the intestinal villi. I-FABP levels rise rapidly in response to an ischemic insult to the villous epithelium (18). In a study involving healthy male participants, I-FABP concentrations were measured at rest, during, and after a 60-minute cycling exercise performed below the lactate threshold. A significant increase in serum IFABP concentration was observed, rising from 309 ± 46 pg/ml to 615 ± 118 pg/ml. The subjects did not develop any gastrointestinal symptoms, and the concentration returned to baseline approximately one hour after the exercise concluded. However, this indicates that even moderate exercise can lead to damage of the intestinal epithelial cells (16). If exercise is performed frequently, lasts longer, and is of greater intensity, as occurs with individuals training for triathlons, damage to the intercellular junctions in the intestinal epithelium may occur (19). Moreover, during high-intensity running, repetitive mechanical forces and vibrational stress applied to the abdominal cavity contribute to further epithelial disruption, which explains why, during the running stage of a triathlon, there are significantly more reported gastrointestinal complaints compared to the swimming and cycling stages (20). This phenomenon can lead to the development of endotoxemia due to the translocation of gut microbiota into the bloodstream. This occurrence has been well documented, showing high concentrations of LPS in the bodies of athletes, as well as a significant increase in pro-inflammatory cytokine levels (21,22). This leads to the development of a local inflammatory state, resulting in increased vascular permeability and the translocation of fluids into the intestinal lumen (23). These findings underscore the vulnerability of the intestinal epithelial barrier to ischemia induced by physical exertion, particularly in endurance athletes. The resulting epithelial damage and increased permeability not only compromise nutrient absorption but also promote systemic inflammatory responses due to the translocation of microbial products into the bloodstream. While ischemic injury plays a central role in the development of gastrointestinal symptoms during exercise, it often occurs in conjunction with other contributing mechanisms.

Other physiological mechanisms

The function of the gastrointestinal system is regulated by numerous physiological mechanisms, including hormonal control (24). One of the roles of the gastrointestinal tract is the secretion of gut-derived peptides, such as glucagon-like peptide-1 (GLP-1), cholecystokinin (CCK), and peptide YY (PYY), which act in a paracrine manner on nearby cells (25). These peptides primarily regulate gastrointestinal motility and, through their effects on the satiety center, help prevent excessive food intake. Under physiological conditions, their secretion increases during food consumption; however, numerous studies have demonstrated that their concentrations also

rise during intense physical exercise (26). Although a direct causal relationship between these substances and exercise-induced gastrointestinal symptoms has not yet been conclusively established, it is assumed that they play a significant role by contributing to the slowing of gastrointestinal motility (27).

In a cohort of triathletes participating in an Ironman 70.3 event, the relationship between gastrointestinal (GI) symptom frequency and dietary intake on the day of the race as well as the preceding day was examined. No associations were found between nutritional intake on the day prior to the race and the occurrence of GI distress during competition. However, a positive correlation was identified between the size of the pre-race meal consumed on the morning of the event and upper gastrointestinal (UGI) symptoms experienced during the cycling segment. Additionally, a higher frequency of lower gastrointestinal (LGI) symptoms during the running segment was observed among athletes who ingested caffeine in doses ranging from 75 to 300 mg prior to the race (28). There is substantial evidence that caffeine increases fluid secretion in the jejunum and ileum approximately one-hour post-ingestion, which may account for the delayed onset of symptoms, particularly during the running leg of the triathlon (29). In another study, it was demonstrated that the presence of gastrointestinal symptoms is significantly influenced by the consumption of foods that slow gastrointestinal motility or promote fluid shifts into the intestinal lumen. Such foods include those high in protein, fat, or fiber. Hyperosmolar beverages have also been shown to exert a negative effect, as they facilitate the movement of water from hypotonic body fluids into the intestinal lumen (8). Some studies have further reported a positive correlation between the severity of symptoms and the amount of carbohydrates ingested during a race. Among female athletes, those who consumed large quantities of carbohydrates during running (1–1.5 g/min) experienced more severe symptoms compared to those who ingested smaller amounts (0–0.5 g/min) (30). Subsequent studies conducted on a larger cohort of full-distance triathletes confirmed a correlation between high carbohydrate intake and the occurrence of nausea and belching. However, it was also demonstrated that athletes who consumed greater amounts of carbohydrates achieved significantly better performance outcomes (6).

Methods of preventing EIGS

Dietary Strategies

Due to the established relationship between the consumption of certain foods and the occurrence of gastrointestinal symptoms, many athletes choose to eliminate specific food items during the peri-competition period. A survey study conducted by Parnell revealed that among a

group of 388 runners, 32% excluded meat, 31% dairy products, 28% fish and seafood, 24% poultry, and 23% high-fiber foods. It was also observed that such dietary restrictions were more frequently adopted by professional athletes compared to amateurs, and more often among younger athletes than older ones (31). Unfortunately, there is currently a lack of well-controlled studies directly linking the elimination of specific dietary components with a measurable reduction in gastrointestinal symptoms (7). Existing evidence primarily supports a notable decrease in symptom prevalence when fiber intake is reduced for at least three days prior to competition (32).

Another strategy increasingly adopted by endurance athletes is the low-FODMAP diet (33). FODMAPs, an acronym for fermentable oligosaccharides, disaccharides, monosaccharides, and polyols, are short-chain carbohydrates. It is known to increase the amount of gas, water, and metabolic by-products in the intestinal lumen. These effects can lead to intestinal distension and are often associated with the onset of lower gastrointestinal symptoms (34). Among professional athletes, daily FODMAP intake has been reported to reach as high as 81 grams (35), significantly exceeding the estimated average intake of 24 grams per day in the general population (36). Several studies have indicated that short-term restriction of FODMAPs—typically for 24 to 48 hours prior to exercise—may be effective in reducing the severity of gastrointestinal symptoms related to endurance activity, particularly when total intake is kept below 5 grams per day (37). Nevertheless, despite promising results, the evidence remains somewhat inconsistent. This is largely due to the fact that FODMAP-rich foods also contribute positively to gut microbiota diversity and short-chain fatty acid production, both of which play important roles in gut health. Interestingly, in a study, simulating prolonged exercise under heat stress conditions (a 2-hour run at 60% $\text{VO}_{2\text{max}}$ at approximately 35 °C), athletes following a low-FODMAP diet exhibited more pronounced intestinal injury and a greater degree of bacterial translocation compared to those consuming a high-FODMAP diet. These findings suggest that while a low-FODMAP approach may offer symptomatic relief in the short term, it could potentially impair certain physiological adaptations or protective mechanisms in the gut (37).

The impact of a gluten-free diet was also investigated in individuals without celiac disease and without non-celiac gluten intolerance. It has been shown that eliminating gluten from the diet did not bring any benefits regarding the intensity of symptoms of EIGS (38). The studies also showed no improvement in individuals following a ketogenic diet (35,38).

Due to the significant role that oxidative stress and reactive oxygen species (ROS) play in the development of EIGS, numerous studies have been conducted analyzing the impact of

antioxidant supplementation, such as L-ascorbic acid, tocopherol, and capsaicin. However, despite a decrease in the concentration of some markers of the inflammatory process, no significant improvement in symptoms was observed during their use (39–42). Additionally, numerous studies have shown that non-steroidal anti-inflammatory drugs, especially ibuprofen, significantly increase the risk of developing EIGS (16,43). As previously mentioned, another factor playing a role in EIGS is the gut microbiota. For this reason, the impact of prebiotics, probiotics, and synbiotics on the severity and frequency of symptoms has been studied. Unfortunately, studies have also failed to provide conclusive evidence of a reduction in symptoms as a result of their supplementation (44,45).

One of the best-documented and most effective nutritional interventions for reducing the severity of EIGS is known as gut training. This approach involves consuming large amounts of carbohydrates during training sessions. It leads to an increased tolerance of the digestive system to food during exercise and enhances carbohydrate absorption, which plays a significant role in long-duration triathlons (46). A study conducted by Costa showed that in a research group that consumed 90g of carbohydrates during one-hour runs for two weeks, the severity of EIGS symptoms decreased by 61%. In the group receiving a placebo, no statistically significant change in the severity of symptoms was observed (47). Besides the benefits of reducing gastrointestinal discomfort, individuals undergoing gut training also showed significant improvements in sports performance. This can be attributed to the ability to deliver a greater amount of carbohydrates during exercise, which are the primary fuel for muscles (33).

Non-dietary strategies

Wilson conducted an observational study in which he sought to find a relationship between sleep problems and EIGS. The initial results of this study indicated that such a correlation exists. However, after considering the anxiety reported by the participants regarding competitions, it turned out that anxiety was responsible for the increased level of discomfort. In individuals reporting sleep problems but denying the presence of anxiety, the severity of EIGS symptoms was similar to that of the group without sleep deficiency (48). Efforts were made to examine the impact of interventions aimed at reducing perceived anxiety, such as incorporating meditation into the training period and support from a sports psychologist, on the symptoms. These studies showed only a slight improvement in 4.7% and approximately 2% of the athletes surveyed, respectively (7,49). Unfortunately, there is currently a lack of research on other non-nutritional strategies that provide significant improvement for individuals with EIGS.

Summary

In recent years, triathlon has ceased to be a sport practiced exclusively by professionals and is gaining an increasing audience among amateurs (50). Like any sport, in addition to its undeniable health benefits, it also carries risks for those who participate. Among the most common risks are injuries (51) and gastrointestinal disorders (52) which we examined in the above work. Based on current knowledge, EIGS may be associated with processes such as splanchnic ischemia, damage to the intestinal villi epithelium, or increased permeability of intercellular junctions in the intestines leading to endotoxemia. Furthermore, these disorders can be exacerbated by nutritional mistakes, excessive fiber intake, or a large meal consumed before the race (20). Various methods for preventing these issues have been analyzed. Currently, the most effective strategies include gut training, reducing fiber intake in the diet, and proper hydration during competitions (44). Unfortunately, their effectiveness is less than one hundred percent, and there is still a lack of more effective methods. It seems that EIGS often partially correlates with anxiety before the competition. However, there is a lack of studies involving large groups of athletes that would assess the impact of prolonged psychological care or short-term use of anxiolytic medications on these disorders (49). There is also a lack of clear evidence-based recommendations regarding nutrition during competitions, which would help reduce the severity of EIGS. Due to the complexity of the issue and its prevalence among both amateurs and professional athletes, this opens substantial opportunities for future research.

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