The impact of physical exercise on the gastrointestinal tract

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Purpose of review

Physical exercise can be both beneficial and harmful for the gastrointestinal tract in a dose–effect relationship between its intensity and health. Mild-to-moderate intensity exercises play a protective role against colon cancer, diverticular disease, cholelithiasis and constipation, whereas acute strenuous exercise may provoke heartburn, nausea, vomiting, abdominal pain, diarrhea and even gastrointestinal bleeding. This review focuses on mechanisms involved in those symptoms and their associations with type of exercises in humans.

Recent findings

One quarter to one half of elite athletes are hampered by the gastrointestinal symptoms that may deter them from participation in training and competitive events. Vigorous exercise-induced gastrointestinal symptoms are often attributed to altered motility, mechanical factor or altered neuroimmunoendocrine secretions. Training, lifestyle modifications, meal composition, adequate hydration and avoidance of excessive use of some medications are the recommendations.

Summary

Strenuous exercise and dehydrated states would be the causes of gastrointestinal symptoms referred by 70% of the athletes. Gut ischemia would be the main cause of nausea, vomiting, abdominal pain and (bloody) diarrhea. The frequency is almost twice as high during running than during other endurance sports as cycling or swimming and 1.5–3.0 times higher in the elite athletes than the recreational exercisers.

Keywords

gastrointestinal benefits, gastrointestinal distress, physical exercise

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Introduction

At the onset of exercise, impulses from motor centers in the brain as well as from working muscles elicit a work-load-dependent increase in sympathoadrenal activity and in release of pituitary hormones. These changes then control the secretion of subordinate endocrine cells resulting in depression of insulin secretion, stimulation of renin–angiotensin–aldosterone system and increased secretion of some peptides related to digestory tract homeostasis [1].

The increase in sympathoadrenal activity is of major importance for the cardiovascular adaptation, thermoregulation and energy-yielding substrate in exercise. Cardiac output is enhanced and blood volume and flow redistributed in favor of working muscle and skin [2].

Beneficial effects of exercise

Both aerobic (walking, cycling) and resistance (strength) exercises enhance muscle protein synthesis and mitochondrial biogenesis [3] improving functional capacity associated with increased muscle mass and reduced fat mass [4]. Repeated bouts of aerobic exercise decrease blood pressure, fasting glucose, insulin and atherogenic lipids [5]. It is well known that regular exercise, independently of body mass index, offers protection against all-cause mortality, primarily by protecting against atherosclerosis, hypertension, type 2 diabetes and colon cancers [6,7].

Observational studies indicate that exercise after onset of colorectal cancer could reduce the risk of death from the disease [8] and provide better quality of life [9,10•].

Several studies indicate an inverse relationship between physical activity and risk of other gastrointestinal-related disease such as diverticular diseases, constipation and cholelithiasis [11–13]. That is probably because regular, moderate exercise can accelerate gut transit of chime and fecal residues [11] and intraluminal gas [14]. In these senses light and moderate exercise are well tolerated and can benefit patients with inflammatory bowel disease [15,16•].

Exercise distress for gastrointestinal tract

Physical exercise leads to gastrointestinal distress mainly when it is vigorous and practiced in a hot environment without adequate training and/or proper hydration. The major pathophysiological mechanisms are related to ischemic, mechanic or neuroendocrine factors.

Ischemic factors

The sympathetic nerve activity in static and dynamic exercises allows the increased cardiac output to meet the body metabolic demand [17]. The resetting of arterial baroreflexes in direct relation to the work intensity enables increased perfusion of the active tissues of skeletal muscle, heart, lung and brain [18]. As blood is shunted from the viscera to these organs during maximum exercise the splanchnic blood flow is decreased by as much as 80%, at 70% of VO_{2max} [19]. So, strenuous exercises reduce gastrointestinal blood flow, what makes the gut mucosa susceptible to ischemic injury, increases mucosa permeability and enhances occult blood loss as well as translocation of protective microbiota and generation of endotoxins that can induce diarrhea [13]. It is known that mucosal ischemia depletes cellular ATP, leading to cell death and mucosal inflammation [20]. Ischemic colitis has been postulated to be a major cause of gastrointestinal bleeding during and after intense exercise [21].

Apart from gut mucosal ischemia, endurance exercises when conducted in hot environments induce higher skin loss of water by sweating. This dehydrated state can be worsened by the fact that, by the excitement of competition, elevated catecholamines suppress thirst [22]. Actually, a slower gastric emptying already associated with high-intensity exercise (above 70% VO_{2max}) can be further slowed by the consumption of hypertonic carbohydrate beverages, usually given after running [13].

The exercise-related dehydrated state can be also associated with the athlete 'dry mouth' (xerostomia) and incidence of upper respiratory tract infection (URTI) [23].

Mechanic factors and motility

The mechanic causes of gastrointestinal distress include enhanced intra-abdominal pressure and organs bouncing. The increased vibration of the abdominal wall and bouncing of the organs are common causes of gastrointestinal distress in runners [13].

The enhanced intra-abdominal pressure as seen in sports such as American football, weightlifting and cycling increases the pressure gradient between the stomach and esophagus which in association with the relaxation of the lower esophageal sphincter, found in high intensity exercises, result in gastroesophageal reflux disease (GERD) [13].

The effects of exercise on gut motility are controversial. Running has been shown to delay small bowel transit time [24,25] and accelerate colonic transit time [25]. Others have found that exercise does not alter small bowel or colonic motility [26] and transit time does not appear to be influenced by short bouts of high-intensity exercise and is independent of the mechanical bouncing of abdominal contents [27].

Distance runners, however, are most susceptible to lower intestinal motility disorders that include diarrhea and irritable bowel syndrome [25,28]. Although the precise pathophysiology remains largely undefined [13], the possible causative factors include enteric fluid and electrolyte balance, mesenteric ischemia, increased mucosal permeability, mechanical trauma and altered colonic motility [13].

Neuroimmune endocrine factors

The less precise mechanism of gastrointestinal distress includes various neuroendocrine and gastrointestinal peptides related to gastrointestinal homeostasis, athletes immunity and feeding behavior [25].

Exercise stress stimulates hypothalamus–pituitary–adrenal axis leading to an increased secretion of immunosuppressive hormones. One of these hormones, cortisol, is consistent with the increased susceptibility to URTI by athletes [29]. Cortisol has been shown to inhibit transepithelial transport of salivary immunoglobulin A (sIgA) [30] and to inhibit *in vivo* B-lymphocyte antibody synthesis after exercise [31].

Endurance athletes are at risk of contracting opportunist infections and studies have shown that suppression of sIgA, commonly found in exercises, is associated with increased incidence of URTI in elite athletes [32,33].

The stress hormones secretion by vigorous exercise stimulus leads to negative energy balance. The major behavior mechanisms to compensate are reduction of nonexercise energy expenditure and/or increasing energy intake [34].

Vigorous exercise has been found to suppress hunger acutely, a phenomenon described as 'exercise-induced anorexia' [35].

Short (1–2 days) to medium-term (7–16 days) exercise can produce a negative balance with no substantial compensatory responses in energy intake being observed. In the long term, (>16 days) energy intake starts to increase,

although the observed compensation is usually partial and incomplete [36].

The mechanisms involved in exercise-related appetite control are likely to fall into three categories: long term, including leptin and insulin; intermediate, including a postabsorptive signal associated with macronutrient oxidation [e.g. glucose and nonesterified fatty acids (NEFA)] and short-term satiety signals arising from gastrointestinal tract in response to food intake [37]. However, no significant changes were found in fasting insulin, glucose, triacylglycerol or NEFA levels with exercise intervention. Changes in the release of satiety hormones by the gastrointestinal tract are therefore likely to be involved in the improvement in short-term appetite control [38,39].

Symptoms of gastrointestinal tract and related disease caused by exercise

The most frequent gastrointestinal symptoms caused by high-intensity exercise are heartburn, chest pain, bloating, belching, nausea, vomiting, abdominal cramps, frequent urge to defecate, diarrhea and constipation [13]. They can be divided into either upper or lower gastrointestinal symptoms [12,40]. The most common related diseases are GERD, diarrhea, gastrointestinal bleeding, immune deficiency and reduced appetite.

The gastrointestinal symptoms occur in 20–50% of high-performance athletes, more often in females than in males and youngsters. They are more frequent in runners and less in sports with gliding movements such as cycling [11,13,15,25]. The running-induced gastrointestinal symptoms and complaints seem related to gastrointestinal ischemia [41••].

Gastroesophageal reflux disease

Symptomatic reflux of gastric contents into the esophagus is encountered in approximately 60% of athletes and occurs more frequently during exercise than at rest [13].

The suggested mechanisms to acid reflux during exercise include gastric dysmotility, relaxation of the lower esophageal sphincter, enhanced pressure gradient between the stomach and esophagus, gastric distention, delayed gastric emptying (specially in the dehydrated state), enhanced intra-abdominal pressure in sports such as football, weightlifting and cycling and increased mechanical stress by bouncing organs [13].

The number of gastroesophageal reflux episodes and duration of esophageal acid exposure are correspondingly increased during the exercise at 90% VO_{2max}. These matches with the duration, amplitude and frequency of esophageal contractions decline with exercise intensity at or above 90% of VO_{2max}. Additionally, increase in reflux episodes has been observed in symptomatic and asymptomatic cyclists at 70% of VO_{2max} despite a lack of differences in esophageal peristaltic velocity and pressure. These data taken together suggest that an increased number of lower esophageal sphincter relaxations may contribute to increased reflux during exercise and be intensity-dependent [42].

The degree of acid reflux is sports-specific. Most heartburn and reflux are demonstrated in athletes involved in predominantly anaerobic sports such as weightlifters, wild symptoms and moderate reflux in runners. Cyclists present wild symptoms and reflux when compared with weightlifters [42]. Athletes with symptoms of GERD tolerate better exercises with less body movements such as cycling [15].

Athletes with exercise-related GERD may present with several symptoms that include substernal chest pressure, pain or burning that mimic angina [43] and some may present with atypical cough, hoarseness and wheezing symptoms that mimic either exercise-induced bronchospasm or vocal cord dysfunction [13].

Exercise not only exacerbates GERD but is also a contributing factor to reflux in health volunteers. Symptoms of heartburn, chest pain and regurgitation were omnipresent even in reduced acid reflux by proton pump inhibition therapy in runners, suggesting a multifactorial cause of GERD during exercise.

The good success in treating GERD in athletes with lifestyle modification includes changing postmeal behavior. Athletes frequently eat shortly before bedtime, a deleterious habit that can enhance postprandial reflux. So by avoiding laying down to sleep within 4 h of the evening meal, avoiding postprandial exercise and avoiding excessive consumption of foods that relax the lower esophageal sphincter, including chocolate, peppermint, onions, high-fat foods, alcohol, tobacco, coffee and citrus products, sleeping on two pillows to enhance gravity-associated esophageal clearance and reduce the symptoms of GERD [13].

Diarrhea

In health athletes, acute diarrhea is either induced by running, food poisoning, travelers or viral gastroenteritis [44]. Typically the diarrhea is self-limited. The acute exercise-induced diarrhea, also know as 'runner's trots', is considered as physiologic and does not result in dehydration or electrolyte imbalances and tends to improve with fitness levels [24].

The relationship of irritable bowel syndrome (IBS) with physical exercise has a two-way version. The role of regular exercise in treating IBS has been studied in sedentary individuals who have IBS and has been shown to be a fairly effective treatment modality [45]. On the contrary, athletes who have IBS rarely have nocturnal symptoms and do not manifest systemic signs of illness or hematochezia. The proposed mechanism for athletes' IBS include increased motor reactivity to various stimuli including stress, impaired transit of bowel gas [46,47], impaired reflex control that delays gas transit [48], autonomic dysfunction, visceral hypersensivity, cholecystocinin and altered immune activation [49].

Various lifestyle and dietary modifications are effective in treating IBS: reassurance, stress reduction, consumption of small meals during the day, high-fiber diet and avoidance of foods such as those containing lactose and candy that contains sorbitol are helpful first-line measures [13].

Gastrointestinal bleeding

The occurrence of exercise-induced gastrointestinal bleeding has been well documented, particularly in distance runners [13]. Eighty-five percent of ultra-marathoners (100 miles) and triathletes [50] have been found to have occult blood positivity. It is more prevalent in younger athletes to have quicker times, and is more common during competition [13].

Postexercise endoscopic colonic mucosal biopsy samples have revealed congestive and hemorrhagic vascular lesions [51,52]. Other proposed causes of exercise-induced gastrointestinal bleeding include hemorrhagic colitis [13] or gastritis secondary to the frequent use of NSAIDS [53]. Fortunately, gastrointestinal bleeding following exercise is usually transient and occult; however, there are case reports documenting substantial upper and lower tract bleeds [13].

The lower incidence of occult bleeding in cyclists and the absence of occult blood in walkers support the theory that mechanical trauma to the gut in runners contributes to occult blood loss. As group runners have been shown to have lower ferritin, serum iron and haptoglobulin [13]. The frequency of iron deficiency is related to the distance and the intensity of running [50].

Gastrointestinal bleeding is frequently occult and athletes typically present with dyspnea on exertion, fatigue, and decreased endurance and strength–anemic symptoms that may be compounded in female athletes who have heavier-than-usual menstrual cycles. Early recognition and diagnosis of occult bleeding is critical to prevention of poor athletic performance. Recommendations have been made for endoscopy prior to instituting therapy for gastrointestinal symptoms and anemia in runners [51].

Immune function

Exercise is thought to modulate immune function [54]. Twelve months of mixed exercises (resistance and endurance) increased both the concentration and secretion of sIgA in elderly runners [55]. The relation between exercise (intensity and duration combined) and URTI has been modeled as a 'J'-form curve [54]. The results show a high incidence of URTI after competition in distance runners compared with similarly trained but noncompeting runners [56] and age-matched nonrunners [57].

Studies using combined exercise and fluid restriction in thermoneutral and hot environments show large reductions in saliva flow rate. However, this could be prevented when individuals receive sufficient water to offset fluid loss during exercise. These studies [58,59] suggest a more likely role for dehydration *per se*, than a neuroendocrine role, in the decrease in saliva flow rate with prolonged exercise.

When comparing intermittent exercise (soccer) with a continuous exercise at the same overall work rate, the former neither suppressed sIgA nor altered the cortisol secretion [60].

The soccer-specific intermittent (moderate intense) exercise was shown neither to suppress sIgA secretion nor to increase salivary cortisol when played 48 and 24 h apart [61], or even the same day with 2.25 h of rest [62].

Appetite

Acute exercise was found to increase postprandial levels of satiety hormones (polypeptide YY, GLP-1 and PP), but to have no impact on ghrelin [37]. In moderate-intensity cycling the modulation effect of exercise on plasma ghrelin was related to the reduction in the postprandial rather than preprandial concentration [63]. The acute exercise-induced increase of plasma PP levels was found not only on fasting [64] but also postprandially [65]. The increase is dependent on the intensity of exercise [66] and decrease with training [67].

Timing of exercise to meal consumption may influence appetite and its hormonal regulators. Postmeal exercise may extend the suppressive effects of meal consumption on appetite [68••].

Thus, exercise has been shown to have beneficial effects on short-term appetite control by enabling a more 'sensitive' eating behavior in response to previous energy

intake. Moreover, it does not appear to prompt any acute physiologic adaptations that would lead to an increase in hunger and/or energy intake in response to increased energy expenditure. The beneficial impact of exercise on the energy balance equation is therefore two-fold, not only increasing energy expenditure but also modulating energy intake.

Conclusion

(1) Physical exercise while in mild-to-moderate intensity has a protective role against gastrointestinal transit-related disorders.
(2) High-intensity exercises often lead to gastrointestinal distress when associated with dehydration states and/or intra-abdominal pressure.
(3) Training and proper hydration would give gastrointestinal protection against high-intensity exercises.

References and recommended reading

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