

## Exercise, Training and Gut Function – a Physiological Perspective

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**ABSTRACT:**

Gastrointestinal (GI) tract adapts and cope with demands of the physical activity to exert maximum output in an athlete. Various GI functions, e.g., secretion, motility, circulation, emptying pattern, absorption, colonic function, etc., undergoes various alterations not only during the event but also during the warranted demand of the training and recovery phases. It depends on the duration, intensity and frequency of the exercise mode. However, often the GI responses to exercise are individual specific. Hyperthermia induced gastric dysfunction have also been documented. Accordingly the adaptations are also evident in the gut depending on the athletic need. However, the gut barrier can prevent the extreme load on the GI tract imposed during working in the excessive stressed conditions.

**Keywords:**

Gastric emptying, gut barrier, exercise and training, adaptation, circulation.

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## INTRODUCTION :

Sports scientists and athletes think about the lungs, heart, muscles, vascular properties and even psychological aspects while training to withstand the rigors of vigorous exercise. Very few of them take into consideration that the gut, the gastrointestinal tract, also must cope with and adapt to the demands of heavy exercise not only during the performance in their events but also during the unwarranted training schedule. Human gut is well designed to meet demands of even most vigorous exercise and possesses the capacity to adjust to the demands of training that promotes the subject to excel sufficiently in competition (Moses 1990). That is why the gut is now considered as an important athletic organ since it mediates water and nutrient uptake during exercise (Murray 2006). However, the adaptations are mostly transparent because they occur naturally in response to improved fitness and increased daily energy intake provided the athlete drinks sufficiently during training to avoid hypovolemia, hypothermia, hypoglycemia and hyponatremia (Murray 2006). Provisions for fluid, nutrient and electrolyte supplementation during exercise have been recently introduced to ensure the GI-adaptations following training programme (Gisolfi 2000). Several findings may support the common belief that mild physical activity favors the digestive process (Marzio et al 1991).

Murray and Kenny (2008) highlighted in a very explicit manner the key issues involving sodium's role in body fluid balance, cardiovascular function, maintenance of plasma volume, and muscle cramping.

In spite of such enormous capacity of the gut to adjust with the exercise demands, GI complaints among athletes are quite common and often reflect severe and life-threatening disorders. Generally, most of the GI complaints are mild; usually transient, develops little risk to health, yet can be bothersome and have the potential to impair performance (Peters et al. 1999). But sometimes severe problems are encountered as evidenced by athletes who have suffered from ischemic cholangitis, intestinal lesions, increased intestinal permeability, etc., brought on by the combination of physical exhaustion, hyperthermia and hypovolemia (Moses 2005, Eichner 1999).

### GI - Physiology during Exercise

The current scientific consensus is that the majority of GI complaints during exercise occur because of reduced gastric emptying, mal-

absorption of water and nutrients, delayed transit time or a reduction in splanchnic blood flow which can give rise to abdominal cramps, acid reflux, heart burn, bloating, persistent eructation and flatulence, nausea, vomiting, urge to defecate, liquefied and / or bloody stool, diarrhoea, etc., which impair the performance significantly (Murray and Shi 2005, Boley et al. 1981, Schwartz et al. 1990).

### Gastroesophageal function :

Duration, amplitude and frequency of gastroesophageal contractions decrease with increase in exercise intensity. Cyclists did not show any reflux even when exercising at 90% of  $VO_2$ max for 60 min. But runners exhibited gastroesophageal reflux during 45 min of exercise at 75% of  $VO_2$ max. It indicated that the gastroesophageal adaptability of an athlete is attributed to the training status, mode of exercise and the concerned game or sport of the athlete (Soffer et al. 1993). Whereas, van Nieuwenhoven (2000) observed that during dehydration gastric emptying is delayed significantly even among the well trained cyclists.

### Gastric emptying :

To meet the fluid and nutrient demands of the exercise, orally ingested beverages should be emptied from the stomach to the intestine because stomach can't absorb any nutrient or fluid except Vitamin B<sub>12</sub> and some amount of alcohol (ACSM Guideline 2006). Gastric emptying follows an exponential time course that shows marked inter-individual differences (Gisolfi 2000, Lambert et al. 1996). It is primarily dependant on the volume and caloric content of the fluid or beverage or food ingested. The more the volume and caloric content, greater will be the duration for emptying (Bronus 1998, Costill book). It has been observed that when a subject ingests a relatively large volume of fluid (~500 ml), gastric volume declines rapidly. Repeated drinking of ~200 ml of fluid every 10-15 min helps to maintain a high gastric volume and therefore a high gastric emptying rate; but addition of glucose to the solution significantly slows the process. No differences in gastric emptying or secretion were observed between trained and untrained subjects during resting condition (Rehrer et al. 1989).

Exercise intensities up to 70-80% of  $VO_2$ max do not affect the regulation of gastric emptying (Carrio et al. 1989, Brouns 1998). But greater intensities of exercise (> 70%  $VO_2$ max), prolongation of exercise, dehydration and hyperthermia impede gastric emptying which



therefore could be responsible for upper-GI problems, e.g., nausea, eructation, reflux, heart burn and even chest pain (Van Nieuwenhoven 2000). Gastric emptying rate is very sensitive to energy content within the stomach and therefore ingestion of high energy containing hypertonic drinks prior to or during the exercise give rise to greater GI complaints (Shi et al. 2004). "Side-ache" is associated with delayed gastric emptying and appears to be linked to physical tugging on the viscera (Morton and Callister 2000). GI-reflux, another symptom associated with gastric emptying is very common among runners due to increased pre-exercise food intake and may be related to inadequate lower esophageal sphincter closure (Gisolfi 2000).

Reductions in gastric emptying appear to be related to the severity of the thermal strain induced by an exercise or heat stress. The volume of ingested water emptied into the intestine at the completion of each exercise bout is inversely correlated with the rectal temperature. Some observations of Neuffer et al. (1989) concluded that 1) exercise in a hot (49°C) environment impairs gastric emptying rate as compared with a neutral (18°C) environment, 2) exercise in a warm (35°C) environment does not significantly reduce gastric emptying before or after heat acclimation, but 3) exercise in a warm environment (35°C) when hypohydrated reduces gastric emptying rate and stomach secretions.

Researches further deciphered that dehydration of 4% body weight or hypohydration of 5% body weight, when combined with elevated core temperatures (~39°C), impair gastric emptying of ingested fluids during moderate intensity treadmill exercise in a cool environment (18°C) or warm environment (30–35°C) (Neuffer et al. 1989, Rehrer et al. 1990). However, Chang et al. 2006 showed that running-induced hypoglycemia, rather than lactate accumulation, is one of the essential factors leading to enhanced liquid gastric emptying in untrained rats.

#### **GI circulation :**

During severe exercise splanchnic blood flow is markedly reduced and intestinal permeability can increase. Performance of severe exercise in the heat further decreases the splanchnic circulation with reduction in gastric emptying and intestinal absorption rates. When the exercise in the heat is accompanied by dehydration (> 3% of body mass), circulatory and thermal functions are further impaired and gut may be subjected to the combined

effects of ischemia, hypoxia and hyperthermia which may cause necrosis, endotoxemia and circulatory impairment (Gisolfi 2000). Mental stress of competition, vigorous exercise, hyperthermia, hypoglycemia and exhaustion increase sympathetic discharge and can reduce splanchnic blood flow by 50% to 80%. Splanchnic blood flow decreases in proportion to exercise intensity and in dehydration to preserve the central blood volume and pressure (Sawka and Young 2005). Prolonged reduction in GI blood supply during exercise causes ischemia, hypoxia and hyperthermia can cause portions of the intestine to lose functional integrity, tissue lesions, necrosis and increased permeability. Lipopolysaccharides start entering following increased permeability and they initiate inflammatory and immunologic events which further alter gut structure and function and thus finally impair the performance.

#### **GI absorption and colonic function:**

There is little evidence to justify the absorptive capacity of the small intestine during exercise. The intestine is not lengthy enough to compensate the lack of absorption during exercise resulted due to fall in gut blood flow and thus the GI-absorption is impaired during exercise (Murray and Shi 2005). There is evidence that orocecal transit time is either increased or decreased during exercise and the urge to defecate following exercise may be due to a decrease in colonic motility during exercise coupled with an increase in propulsive activity after exercise (Rehrer et al. 1992, Rao et al. 1999). Athletes prone to GI disturbances have longer orocecal transit times and may be more prone to altered intestinal permeability (van Nieuwenhoven et al. 2004). Athletes ingesting of high fibre meal or snack prior to exercise are more prone to GI problems because dietary fibres increase intestinal bulk and water content that in turn hampers the performance (Lambert et al. 1996, Lambert et al. 1997). Sympathetic stimulation during exercise reduces the gut blood flow and this may increase the passage of colonic contents into the rectum which among runners may further be promoted by up and down nature of running (Murray 2006).

In a randomized control study on 102 patients, Johannesson (2011) showed that increased physical activity improved gastrointestinal symptoms in irritative bowel syndrome (IBS). It has been proposed in the study that physically active patients with IBS would face less symptom deterioration compared with physically inactive

patients and accordingly physical activity has been recommended as a primary treatment modality in IBS.

#### **Intestinal Absorption :**

Exercise at 64–78% VO<sub>2</sub>max does not impose consistent effect on glucose, water, electrolyte, or urea transport (Fordtran and Saltin 1967). According to Fordtran and Saltin (1967) exercise had no consistent effect on carrier-mediated or passive absorption, nor was intestinal permeability altered based on the diffusion ratio of passively absorbed solutes. Gisolfi et al. (1991) tested five different solutions but only in one or two subjects; nevertheless, the conclusions regarding intestinal absorption were confirmed. The latter investigators determined the effects of 1-h bouts of cycle ergometer exercise at 30%, 50%, and 70% VO<sub>2</sub>max while perfusing the duodenojejunum of six trained cyclists with water. They found no significant difference in fluid absorption during rest, exercise, or recovery periods, and there were no significant differences among absorption rates for the three exercise intensities.

Gisolfi (2000) reported that addition of carbohydrate to an electrolyte solution increases water absorption to an extent six fold. Perfusion of intestine with water or glucose-electrolyte solution at a rate that exceeds the gastric emptying rate by 70–100%, could not reduce the intestinal absorption. Significant decline in active absorption in the heat (38°C) was attributed to as a combined effect of exercise and heat stress. Therefore, it is justified to state that intestinal absorption does not serve as a limiting factor in fluid or nutrient provision during mild to heavy exercise (30–80% VO<sub>2</sub>max) lasting as long as 60–90 min.

There are numerous evidences suggesting that acute and chronic exercises reduces transit time and increases colonic motility that act as a major cause of runners' diarrhoea which is of colonic origin. However, exercise and physical activity are beneficial to withstand constipation. Recent evidences suggest that colonic motility was significantly reduced by graded exercise but increased in recovery. Such exercise induced increase in colonic motility is thus speculated as one of the rationale towards lower prevalence of colon cancer, diverticular diseases and haemorrhoids among active individuals and sports persons.

Prolonged bouts of hyperthermic and dehydrating exercises impose direct effects on colonic function and are major causes of abdominal

cramps, gas, urge of defecation during or immediately after exercise and diarrhoea among athletes (Moses 1990). Since colon is lacking collateral circulation, it is more sensitive to ischemia due to decreased splanchnic blood flow during exercise. Loose stools frequently observed early in a training program are transformed to normal bowel movements with continued training. This point requires investigation, but indicates together with the other evidence above that the gut may adapt to the effects of chronic exercise.

However, the scientists have agreed that solution osmolality and total solute flux are the major factors governing the water absorption in the small intestine not only during exercise but also at rest. Earlier studies suggested that hyperosmotic solution ingestion causes net water movement into the intestinal lumen (Rehrer et al. 1992). Moreover, ingestion of carbohydrate electrolyte solution of different osmolalities ranging from 186 to 403 mosmol/kgH<sub>2</sub>O also increased net water movement into the distal duodenum and proximal jejunum (50 cm segment) (Shi et al. 1994).

#### **GI adaptations to training :**

##### **Signs of GI Adaptation :**

There are some evidences indicating that GI adaptations take place with training. The volume of empty stomach is about 50-100 ml, but can expand to about 1000ml without any change in intragastric pressure (ACSM 2006). Greater expansion with enhancement of gastric emptying rate and orocecal transit time has been reported in the endurance athletes (Gisolfi 2000, Leiper et al. 2005, Harris et al. 1991). Endurance runners show a significantly accelerated basal gastric emptying rate which was attributed as the “learning effect” that happens out of conditioning process (Carrio et al. 1989).

Increased energy demands causes hyperphagia, small bowel adaptations, quicker food processing and speedy small intestinal transit along with increased absorptive capacity in animal models as well as in highly trained endurance athletes who consume up to four times more calories than their sedentary counterparts. Evidences suggest that endurance training improves gastric emptying and augments GI transit without any decrease in absorptive capacity. Furthermore, endurance-trained animals provide evidence of improved gut-barrier function (Gissolf 2000).

The percentage of amount of test meal retained in the stomach with half time for gastric emptying (*t*<sub>1/2</sub>) was 67.7 ± 5.9 min for endurance runners and 85.3 ± 4.5 min for sedentary controls





( $P < 0.001$ ), respectively (Carrio et al. 1989). It indicated that endurance runners (2–7 yr of training and  $171 \pm 12$  min marathon time) possess a significantly faster rate of basal gastric emptying. It also provides an evidence of adaptation in gastric motility following exercise training.

Harris et al. (1991) demonstrated faster orocecal transit time (OCTT, mouth-to-colon lactulose transit) in athletes that occurred without a decrease in intestinal absorption, indicating that the hyperphagia of chronic exercise was associated with adaptations within the GI system. However, the caloric intake was much higher in endurance athletes than the sedentary subjects.

#### **GUT Barrier Function :**

It is generally well accepted that physically fit individuals have greater heat tolerance than unfit individuals and this might be the reason for improved gut-barrier function in trained persons. Blood flow decreases in the liver and intestines during exercise to compensate the excess demand of cutaneous circulation, especially in the heat. Evidences also suggest that reduced mesenteric blood flow reduces intestinal barrier function and promotes endotoxin leak (Fink et al. 1991) that accumulated endotoxin in the blood which in turn reduces heat tolerance. Animal experiment proved that indomethacin infusion increased the heat tolerance due to block of endotoxin-induced fever (Sakurada and Hales 1998). However, in a group of physically fit animals the similar results were obtained without indomethacin infusion. These results concluded that the difference in heat tolerance between sedentary and physically fit animals is in part attributable to endotoxin leaving the gut. It has been postulated that training improves GI and splanchnic blood supply not only in normal environmental conditions, but also during heat stress and that in turn maintains a better GI barrier to endotoxin translocation from gut lumen to plasma.

Valdés-Ramos et al. (2010) explained the effects of diet, nutrients or exercise on the GI-mucosal immune system. According to the study the gut is not only responsible for the entry of nutrients into the organism, but also for triggering the primary immune response to orally ingested antigens. The lymphoid tissue containing large amount of immune cells, disseminated all along the intestine in Peyer's patches and lamina propria may be influenced by light to moderate exercise to modulate the immune system tissue, whereas exhaustive acute exercise may cause

immunosuppression. However, high-fat diets combined with exercise are able to induce an increase in CD3+ lymphocytes due to increased CD8+ cells and a decrease in B-cells.

#### **GI function under conditions of severe stress :**

Prolonged exercise that promotes ischemia, hypoxia, and hyperthermia combined with dehydration and/or heat develops variety of GI symptoms including bloating, nausea, vomiting, diarrhoea, and intestinal bleeding (Bronus and Beckers 1993). Hypothesis proposes that exercise stress produces biochemical changes that uncouple oxidative phosphorylation, reducing ATP production and increasing  $\text{Ca}^{2+}$  efflux from mitochondria and endoplasmic reticulum. These events lead to increased cytosolic  $\text{Ca}^{2+}$  concentration, the generation of reactive oxygen species, and loss of tight junction control, producing increased intestinal permeability leading to gut-barrier dysfunction (Madara and Stafford 1989). Actually, exercising under thermal stress increases heat storage and core body temperature that in turn causes splanchnic ischemia, cellular hypoxia and nitric oxide (NO) induction (Hall et al. 1994). These physiological effects increase the intestinal permeability that leads to secretion of interferon- $\gamma$ , leakage of endotoxin, production of inflammatory mediators, hypotension, and circulatory shock / heat stroke that may be fatal to the subject (Sakurada and Halles 1998, Madara and Stafford 1989).

#### **CONCLUSION :**

Altered gastric functions during different types of exercise, training and events are influenced by various factors. The gastroesophageal functions vary with the duration and intensity of exercise and splanchnic circulation reduce markedly during severe exercise. Proper amount of fluid intake is an important parameter to withstand the adverse effects of dehydration mediated gastric abnormalities in players. Gastric emptying related upper-GI problems, e.g., nausea, eructation, reflux, heart burn and even chest pain are closely associated with high-moderate to heavy exercise intensities. All these are in turn associated with hyperthermia induced problems in orocecal functions and gastrointestinal motility in athletes. Gut-barrier function and gastrointestinal adaptations following training are also not consistent in all types of athletes and those are very much individual specific. Therefore, it is quite clear that gastrointestinal function of an athlete needs to

be clearly evaluated before selection of an individual for a particular sport and it may also be implemented as a part of not only for general selection of an athlete but also should be precisely appraised for event selection of an athlete.

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