SYSTEMATIC REVIEW
Vigorous exercise and diarrhoea.
Roy J. Shephard

Abstract
Objective: To define the prevalence of exercise-induced diarrhoea, to consider its possible causes, and to suggest appropriate management by the coach and the sports physician. Methods: Information on exercise-induced diarrhoea in the endurance athlete has been garnered by a detailed search of Ovid/Medline, PubMed and Google Scholar databases. Results: The prevalence of exercise-induced diarrhoea is greater in the young than the elderly, and in women rather than men; the risk increases with the intensity of effort, but is diminished by training. Endurance running is the activity most commonly implicated, with as many as a half of participants in running events passing a stool during competition at least once during their career. The problem can also occur, although less frequently, during or following bouts of long-distance cycling and skiing. Exercise-related diarrhoea can also occur in patients with inflammatory bowel disease, irritable bowel disorder, and following abdominal irradiation for prostate cancer, although a much smaller volume of physical activity usually precipitates the problems in such individuals. Factors contributing to exercise-induced diarrhoea may include an inappropriate diet, mechanical stimulation of the colon, visceral ischaemia, exercise-induced hormonal changes, and pre-race stress and anxiety. After excluding more serious underlying pathologies, the management of exercise-induced diarrhoea may stress an optimization of diet, enhancement of physical condition by cross training, emptying of the colon before competition, and provision of adequate toilet facilities along the race route. Moderate doses of loperamide may be considered for individuals with persistent problems. Conclusions: Exercise-induced diarrhoea is a potential problem for many active individuals. The resulting social embarrassment can have a negative impact upon motivation to engage in regular endurance activity. However, with appropriate management, difficulties can be minimized and most athletes can reach desired levels of physical activity.

Introduction
In Runner's diarrhoea, or the "athlete's trots," was first described by Scobie (1970) and Fogoros (1980). It is a well-recognized problem among endurance runners. Perhaps the best-known example is Paula Radcliffe, the current female record holder for the marathon. When leading the women's pack at the 22nd mile of the 2005 London marathon, she was forced to stop by the roadside and pass diarrhoea in full view of the television cameras. Her symptoms had begun at mile 15, and she blamed the incident on eating too much, including a dinner of grilled salmon she had taken the previous evening (The Scotsman, 18th April, 2005). A second "poster child" for the condition is the Grand Rapids-based writer, editor and journalist Lindsay Patton-Carson. She knew that she often had to defaecate after completing a training run, but when participating in the local marathon, all went well until she reached the 19th mile. There, she drank some pickle juice, expecting it to ease a cramp. Five minutes later, she had an urgent need to defaecate, and she failed to reach the next track-side "port-a-potty" before an uncontrollable bowel movement occurred (Patton-Carson, 2014). Many competitors have at least occasionally faced a similar irresistible urge to defaecate in the latter part of a long distance run, Sometimes, the outcome has been a frank diarrhoea, with
How fast must you trot? Vigorous exercise and diarrhoea.

or without rectal bleeding, and in other instances the runner has passed very soft faeces, at the upper end of the Bristol scale of stool consistency (Lewis and Heaton, 1997).

The problem of exercise-induced diarrhoea is not confined exclusively to endurance athletes. Many men who have undergone extensive abdominal irradiation for the treatment of prostatic cancer also suffer from persistent diarrhoea, particularly if the radiotherapy has been supplemented by androgen suppressant drugs (it is less widely recognized that this problem is exacerbated by quite modest bouts of moderate endurance exercise). Individuals with inflammatory bowel disease and irritable bowel syndrome may also be affected. Apart from the immediate social embarrassment of public defaecation, the condition has the unfortunate potential of discouraging the affected individuals from taking the amount of exercise that they wish, and even the minimum currently recommended for the maintenance of good health.

Despite the prevalence of exercise-induced diarrhoea, the condition has received relatively little scientific attention. Thus, the topic receives quite brief mention in a recent major text on diarrhoea (Triezenberg and Simons, 2010). The objectives of the present review are to explore what is currently known about the condition, and how it may be alleviated. The information has importance not only for those who are dealing with elite long-distance runners, but also for those who offer advice to more modest exercisers who suffer from exercise-related diarrhoea associated with inflammatory bowel disease, irritable bowel syndrome or the late complications of prostatic cancer treatment.

Methods

The database of Ovid/Emeline was searched systematically without restriction of language from January 1996 to June 2015. The terms "exercise" (152,864 hits), "athletes" (4,242 hits), "endurance exercise" (2,104 hits), "race" (512,550 hits), and "running" (9,554 hits) were combined, using the "OR" function, to yield 209,225 unique citations. The terms "diarrhoea" (19,984 hits), "trots" (70 hits), and "Intestinal colic" (23 hits) were likewise combined to yield a total of 20,075 references. A combination of these 2 searches, using the "AND" function, identified 96 abstracts of interest. Study of these abstracts showed only 24 papers relevant to the present enquiry. Twelve of these articles dealt with infections incurred by exercisers, either through contact with infected water or mud, or through travel to distant competitive sites, and the remaining 12 papers covered non-infectious causes of diarrhoea in exercisers. This information was supplemented substantially by parallel searches of Pub-Med, Google Scholar, reference lists and personal files.

Prevalence of exercise-induced diarrhea

Runners who are affected by exercise-induced diarrhoea usually report a general alteration of bowel habits, particularly looser stools and more frequent defaecation than seen in the general population (Sullivan et al., 1994). More specific problems may arise just prior to, during and immediately following an event, with both an urge to defaecate and diarrhoea (Keeffe et al. 1984). Abdominal distress, cramps, and the involuntary passage of stools may
How fast must you trot? Vigorous exercise and diarrhoea.

occur if the runner does not stop to defaecate (Brouns 1991). Indeed, for some competitors, the onset of urgency is a predictable event; they recognize that problems usually develop after covering a specific distance, such as 2, 4, or 10 miles (Keeffe et al. 1984). Peters et al. (1993) found that the runner’s urge to defaecate was correlated with nausea, cramps, and flatulence.

Published reports on the prevalence of exercise-induced diarrhoea vary quite widely (Table 1). Two immediate issues are a low response rate to questionnaires (with the possibility of a selective response from those vulnerable to the condition), and differences in methods of reporting, ranging from diarrhoea during a recently completed event to diarrhoea once during a running career. Samples have also varied in age, sex, level of training and the range and intensity of the sports performed. The problem is more common in young than in older competitors (Keeffe et al., 1984), and in women than in men (Halvorsen et al., 1990; Keeffe et al., 1984; Riddoch and Trinick, 1988). Often, the condition seems to have been precipitated by a recent increase in training mileage or a particularly strenuous work-out (Fogoros 1980), and problems are 3 times more common in elite athletes than in recreational exercisers (de Oliveira and Burini 2009). Some observers have found a relationship to the intensity of effort (Keeffe et al. 1984; Sullivan and Wong 1992) or its duration (Peters et al. 1999), but others have not (Priebe and Priebe 1984; Worobetz and Gerrard 1985). Complaints are most prevalent among distance runners; although cases have also been described in swimmers (Strauss et al., 1988), cyclists (Wilhite and Mellion 1990) and skiers (Kehl et al. 1986); the prevalence is roughly half as great in cycling and swimming as in running (de Oliveira and Burini, 2009; Peters et al., 1999). Finally, the condition seems to be more common in those with irritable bowel syndrome or lactose intolerance (Priebe and Priebe, 1984).

There have been 17 surveys of the prevalence of exercise-induced diarrhoea, conducted by 12 laboratories (Table 1). Most investigators have questioned long-distance runners, but there have also been studies of skiers (Kehl et al., 1986), cyclists (Peters et al., 1999), triathletes (Peters et al., 1999; Rehrer et al., 1990; Worme et al., 1990) and multi-sport competitors (Worobetz and Gerrard, 1985). Among those reporting a high incidence of defaecation during competition (Halvorsen et al., 1990; Keeffe et al., 1984; Riddoch and Trinick, 1998; Worobetz and Gerrard, 1985), the question posed has usually been "have you encountered such a problem occasionally or frequently." Sullivan and Wong (1992) obtained more precise information for a group of recreational runners; 3 of 107 had to stop their running to defaecate on almost every day, and in 17 of the 107 this occurred once a month. However, some surveys found quite low rates during a specific event (Rehrer et al., 1989 and Ter Steege et al., 2008).

A number of reports have also commented on the presence of blood in the stools, a factor important in determining the aetiology of bowel disorders. Again, the reported prevalence is quite varied (Table 1), depending not only upon the intensity and duration of the activity performed, but also upon the criterion of blood loss, which has ranged from the detection of occult blood to the passage of overtly bloody faeces, either occasionally or frequently (Halvorsen et al., 1900; Keefe et al., 1984; McCabe et al., 1980; Peters et al., 1984).
## Table 1: Reported prevalence of diarrhoea and defaecation during and immediately following vigorous exercise.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample</th>
<th>Exercise</th>
<th>Findings</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halvorsen et al. 1990</td>
<td>279 leisure-time marathon runners</td>
<td>Training and marathon running, particularly if fast-paced</td>
<td>GI disturbances in 34%. 20% sufficient to affect performance</td>
<td>Long-lasting GI problems in 25%, improved in 41% with regular training; diarrhoea in 15% during run, 6% post-event; blood in stools in 3/279</td>
</tr>
<tr>
<td>Keeffe et al. 1984</td>
<td>707 marathon runners responding to questionnaire (41.6% response rate)</td>
<td>Hard runs and races</td>
<td>Bowel movements in 35%, diarrhoea in 19% after running &quot;occasionally or frequently&quot;</td>
<td>Races occasionally interrupted by bowel movement (18%) or diarrhoea (9%). Bloody diarrhoea in 1.2-1.8%</td>
</tr>
<tr>
<td>Kehl et al. 1986</td>
<td>41 cross-country skiers</td>
<td>Engadin ski marathon (42 km, with substantial changes of altitude)</td>
<td>8/41 had abdominal pains and/or diarrhoea during or after skiing</td>
<td>Faecal blood loss in 3/41</td>
</tr>
<tr>
<td>McCabe et al. 1986</td>
<td>125 runners</td>
<td>Marathon run</td>
<td>Abdominal cramps in 22/125, diarrhoea in 8/125 (6.4%) during or following event</td>
<td>Frank haematochezia in 6%. Questionnaire response rate 25%</td>
</tr>
<tr>
<td>Peters et al. 1999</td>
<td>199 runners, 197 cyclists, 210 triathletes</td>
<td>Running &amp; cycling events</td>
<td>Lower abdominal symptoms in 69% M, 72% F when running, 60% M, 69% F during cycling; actual defaecation on only 4 occasions</td>
<td>Symptoms included cramps, bloating, urge to defaecate, defaecation, diarrhoea, flatulence &amp; side-ache</td>
</tr>
<tr>
<td>Priebe and Priebe, 1984</td>
<td>425 runners</td>
<td>Distance run</td>
<td>63% had experienced urge to defaecate, 51% an actual bowel movement with exercise, diarrhoea in 30%</td>
<td>Rectal bleeding in 12%. Some runners could control symptoms with prophylactic medication</td>
</tr>
<tr>
<td>Rehrer et al. 1989</td>
<td>114 previously untrained subjects</td>
<td>Marathon preparation</td>
<td>No cases of diarrhoea in a 25 km or a 42 km run</td>
<td></td>
</tr>
<tr>
<td>Rehrer, 1990; Rehrer et al. 1992</td>
<td>70 runners</td>
<td>67 km alpine marathon</td>
<td>Intestinal cramps in 7.6% of men, 25% of women; Diarrhoea in 4% of men, 25% of women</td>
<td>70 of 170 runners responded to questionnaire</td>
</tr>
<tr>
<td>Rehrer et al. 1990</td>
<td>55 male triathletes</td>
<td>Half-triathlon</td>
<td>20% had severe GI complaints when running, 9% when cycling, but no data on defaecation</td>
<td>Symptoms may be related to fermentation of fibre in lower GI tract</td>
</tr>
<tr>
<td>Sullivan, 1981</td>
<td>57 Canadian recreational &amp; competitive running club</td>
<td></td>
<td>30% had urge for bowel movement, diarrhoea in 25%</td>
<td></td>
</tr>
</tbody>
</table>
How fast must you trot? Vigorous exercise and diarrhoea.

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Sample</th>
<th>Exercise</th>
<th>Findings</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sullivan and Wong, 1992</td>
<td>109 distance running club members</td>
<td>Training and running events</td>
<td>On occasion, 12% incontinent while running, 62% had stopped to defaecate while training; in 3 of 109, happened on every run, for 17 of 109 at least once/month</td>
<td>43% had nervous diarrhoea before a competition</td>
</tr>
<tr>
<td>Sullivan et al. 1994</td>
<td>93 runners, 95 controls</td>
<td>Need for bowel movement was a common reason for runner to interrupt an event</td>
<td>Runners had more frequent bowel movements, more often loose and urgent</td>
<td>Runners ate more fibre than controls</td>
</tr>
<tr>
<td>Ter Steege et al. 2008</td>
<td>1281 long-distance runners, Enschede marathon</td>
<td>Need for bowel movement was a common reason for runner to interrupt an event</td>
<td>Urge to defaecate &amp; diarrhoea in less than 1% of runners</td>
<td>62% response rate to 2076 questionnaires. Bloody stools in &lt;0.5%. Complaints more frequent in females &amp; younger runners</td>
</tr>
<tr>
<td>Worme et al. 1990</td>
<td>67 recreational triathletes</td>
<td>Triathlon</td>
<td>Incontinence 2/67, diarrhoea 12/67</td>
<td></td>
</tr>
<tr>
<td>Worobetz and Gerrard, 1985</td>
<td>70 multisport athletes</td>
<td>Triathlon</td>
<td>Incontinence 2/67, diarrhoea 12/67</td>
<td></td>
</tr>
<tr>
<td>Worobetz and Gerrard, 1985</td>
<td>119 marathon runners</td>
<td>Marathon runs</td>
<td>43/70 (61%) had disturbing lower GI symptoms</td>
<td></td>
</tr>
</tbody>
</table>

1986; Ter Steege et al., 1984). Overt blood loss has ranged from <0.5% of runners (Ter Steege et al., 2008) to 6% (McCabe et al., 1986) or even 12% (Priebe and Priebe, 1984).

**Aetiology**

A variety of possible causes of exercise-induced diarrhoea have been suggested, including dietary issues, an acceleration of colonic transit, the development of visceral ischaemia, the mechanical effects of exercise upon the gut contents, exercise-induced changes in hormone concentrations, development of an electrolyte imbalance, and autonomic and hormonal reactions to the stress of competition. Ideally, the correct hypothesis should explain the greater prevalence of problems in women and younger athletes, the effects of the intensity of effort, and the lessening of difficulties as training is increased. Points can be advanced for and against each of the proposed explanations, and the aetiology may ultimately prove to involve several of these factors.

**Dietary influences.** One immediate potential issue is that some distance runners adopt a specific lifestyle and dietary habits. A comparison of 93 randomly selected runners with 95 controls found the runners ate more

Health & Fitness Journal of Canada, ISSN 1920-6216, Vol. 8, No. 1 · January 30, 2015 · 36
dietary fibre, and had more frequent bowel movements, often loose and urgent (Sullivan et al., 1994). Athletes are also likely to eat a greater bulk of food than sedentary people, and many consume large quantities of vitamins and other supplements that affect intestinal motility. Offering some support of the notion of a "runner's diet," Bingham and Cummings (1989) noted that the allocation of subjects to a moderate exercise programme (jogging an hour per day) produced no consistent change in large bowel function as traced by radioactive markers, provided that the active subjects continued to eat a similar diet to sedentary individuals (as assured by residence in a nutritional laboratory). Dietary fibre certainly increases the bulk of the colonic contents and thus tends to speed transit. Moreover, the faster transit decreases water absorption, and this further increases the likelihood of defaecation (Brouns and Becker, 1993). However, one investigation suggested that athletes who develop lower gastrointestinal problems during exercise did not distinguish themselves from their peers in terms of their intake of either dietary fibre or milk (Sullivan et al., 1994).

Many athletes and health-conscious members of exercise clubs have intakes of vitamin and other nutritional supplements that are substantially above recommended daily limits, and this can predispose to both nausea and diarrhoea (Hoyt 1980; Lanham-New et al., 2011; Ostojic and Ahmetovic, 2008; Worme et al., 1990). Doses of vitamin A >3000 mg-day⁻¹, of vitamin C >2000 mg-day⁻¹, of creatine >10 g per serving, and excessive intakes of Mg, zinc, iron, bicarbonate, and cysteine can all cause gastrointestinal problems. A large intake of caffeine, possibly taken as an ergogenic aid, can also have a laxative effect (Putukian, 1997).

Sometimes, vigorous exercise can also slow the absorption of water, minerals and sugars, with an active secretion of fluids into the gut. Fordtran and Saltin (1967) reported that 1 hour of exercise at 70% of maximal oxygen intake left gastric emptying unchanged, and caused no impairment of jejunal or ileal absorption that could increase the osmotic load in the intestines and predispose to diarrhoea. In contrast, another investigation found that cycle ergometer exercise at only 45-50% of maximal oxygen intake halved the jejunal intake of water, sodium, potassium and chlorine (Barclay and Turnbereg, 1988). Likewise, Maughan et al., (1990) demonstrated a slowing in the absorption of deuterated water when exercising at 80% of maximal oxygen intake relative to exercise at 42% or 61% of maximal aerobic power, although their study failed to distinguish between effects from a slowing of gastric emptying and an action of exercise upon jejunal absorption. Isaacs (1984) made direct observations on sodium absorption in five patients with ileostomies; he found evidence of sodium retention when his subjects were running a marathon.

Several authors have linked the urge to defaecate to a malabsorption of carbohydrate and resulting changes in the volume of fluid in the gut (Peters et al., 1993; Pribe and Pribe, 1984; Rehrer et al., 1989; Williams et al., 1964), and also fermentation of carbohydrates in the large intestine (Lanham-New et al., 2011). Pribe and Pribe (1984) noted that nervous diarrhoea immediately before competition was often associated with lactose intolerance. Williams and associates had 6 healthy subjects walk at 4.8 km·hr⁻¹ for 4.5 hr at a temperature of 38°C; this caused no change of xylose
absorption, but it did result in a slowing in the absorption and excretion of 3-o-methyl glucose (Williams et al., 1964). Rehrer et al. (1989) pointed out that sports drinks with >7-10% carbohydrate can impair osmotic water absorption and even draw water into the intestines. Foods with a high fibre content or a high glycaemic index can have the same effect (Rehrer et al., 1989).

**Accelerated gastrointestinal transit.**
A general exercise-induced speeding of gastrointestinal transit would reduce the absorption of water from the gut, and thus would likely predispose to diarrhoea; more specific effects might be linked to increases of colonic motility and expulsive movements.

Empirical data on exercise and gastrointestinal motility show substantial variation from one survey to another (Table 2), reflecting differences of methodology, differences in the reported data (oro-anal, oro-caecal or colonic transit), differences in the intensity of exercise performed, and studies of the acute versus the chronic effects of physical activity. Harrison, Leeds, Bolster and Judd (1980) suggested that the habitual gastrointestinal motility also influenced the response to physical activity, with exercise speeding transit in individuals where bowel movements were normally slow, and having the opposite effect in individuals where bowel movements were normally rapid. Many studies have looked at the effects of exercise upon gastric and small intestinal motility, using a breath hydrogen estimate of oro-caecal transit. But from the viewpoint of exercise-induced diarrhoea, a more important factor is the time that food takes to pass through the colon, usually assessed by the use of radio-opaque or radioactive markers such as $^{51}$Cr or $^{99}$Tc.

**Acute effects of exercise on oro-caecal transit.** Six reports have examined the acute effects of moderate and vigorous aerobic exercise upon the oro-caecal transit time. Three found a slowing of transit (Meshkinpour et al., 1989; Moses et al. 1988; van Nieuwenhoven et al. 2004) one a speeding of gastric emptying, but no change in small intestinal behaviour (Cammack et al. 1982), one no effect (Ollerenshaw et al., 1987), and one a speeding of transit (Keeling and Martin 1987) (although this last outcome was possibly attributable to more rapid gastric emptying).

Meshkinpour et al. (1989) reported that 60 min of treadmill walking at 4.5 km-h$^{-1}$ slowed the mouth to caecal transit time for water/lactulose from 55 min at rest to 89 min during exercise. Moses et al. (1988) found that more vigorous effort, a 2-hour treadmill run at 65% of maximal oxygen intake, also delayed the transit of both water and a glucose polymer solution relative to resting conditions. Likewise, van Nieuwenhoven et al. (2004) noted that 90 min of cycling or running at 70% of aerobic power increased oro-caecal transit time and increased intestinal permeability, changes being larger with running than with cycling. Ollerenshaw et al. (1987) studied the effects of 3 differing patterns of exercise upon small intestinal transit, using $^{99}$Tc labelled resin beads. The most vigorous activity in this study involved three 20-min periods of cycling at a heart rate of 160 bpm, interspersed with five 20-min periods of walking. Small intestinal transit times did not differ between minimal, moderate and "strenuous" patterns of exercise.
Table 2: Influence of vigorous exercise upon intestinal transit.

<table>
<thead>
<tr>
<th>Author</th>
<th>Subjects</th>
<th>Methodology</th>
<th>Exercise</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cammack et al. 1982</td>
<td>1 M, 6 F</td>
<td>Breath hydrogen analysis and $^{99}$Tc</td>
<td>60 min cycle ergometry at heart rate of 117 bpm</td>
<td>Gastric emptying speed, but no change of small intestinal transit time for solid meal (300 min)</td>
</tr>
<tr>
<td>Cheskin et al. 1992</td>
<td>5 M, 5 F</td>
<td>Pressure sensitive catheter in colon</td>
<td>1 h jogging at 70-80% of maximal heart rate</td>
<td>Colonic contractions increased in 6/10 subjects, including 5 with exercise-induced diarrhoea</td>
</tr>
<tr>
<td>Dapoigny and Sarna, 1991</td>
<td>6 dogs</td>
<td>9 strain gauge transducers</td>
<td>1 h treadmill run, 5 km·hr$^{-1}$, 5% slope</td>
<td>Exercise decreased colonic migrating motor complexes, but at beginning of exercise giant migrating complexes followed by defeacation</td>
</tr>
<tr>
<td>Evans et al. 1984</td>
<td>20 healthy volunteers (10 exercise, 10 controls)</td>
<td>Pressure sensitive radio-telemetry capsule in proximal jejunum</td>
<td>4 hr, 12 mile walk</td>
<td>Decreased migrating motor complex contractions by about a half</td>
</tr>
<tr>
<td>Holdstock et al. 1970</td>
<td>13 M, 14 F (19/27 had irritable colon syndrome)</td>
<td>$^{51}$Cr capsules in colon</td>
<td>Modest walking vs. sitting</td>
<td>Exercise increases mass movements of colon and urge to defeacate</td>
</tr>
<tr>
<td>Kayaleh et al. 1996</td>
<td>8 M runners</td>
<td>Breath hydrogen analysis</td>
<td>1 hr run (9.6 km) vs. rest</td>
<td>No change in mouth to caecum transit time</td>
</tr>
<tr>
<td>Keeling and Martin, 1987</td>
<td>12 M</td>
<td>Breath hydrogen analysis</td>
<td>120 min treadmill walk at 5.6 km·hr$^{-1}$ up 2% grade vs. sitting</td>
<td>Liquid meal, decrease of oro-caecal transit from 66 to 44 min with exercise</td>
</tr>
<tr>
<td>Meshkinpour et al. 1989</td>
<td>7 M 14 F</td>
<td>Breath hydrogen analysis</td>
<td>60 min treadmill walk at 4.5 km·hr$^{-1}$</td>
<td>Liquid meal. Increase of oro-caecal transit time 58 to 89 min with exercise</td>
</tr>
<tr>
<td>Moses et al. 1988</td>
<td>10 M</td>
<td>Breath hydrogen analysis</td>
<td>120 min treadmill run at 65% maximal oxygen intake</td>
<td>Oral-caecal transit delayed relative to rest with either water or glucose/polymer meal</td>
</tr>
<tr>
<td>Ollerenshaw et al. 1987</td>
<td>9 M</td>
<td>$^{99}$Tc-tagged resin beads</td>
<td>Comparison of mild, moderate and strenuous exercise (5 hr, at heart rate to 160 bpm)</td>
<td>Water + lunch, small intestinal transit 240-300 min, no significant difference between 3 exercise patterns</td>
</tr>
<tr>
<td>Rao et al. 1999</td>
<td>Untrained subjects (6 M, 6 F)</td>
<td>Solid state probe placed by colonoscopy</td>
<td>Graded cycle ergometer exercise to 75% maximal aerobic power</td>
<td>Colonic phasic activity decreased during exercise, (intensity-dependent response) increase of propagated activity after exercise</td>
</tr>
<tr>
<td>van Nieuwenhoven et al. 2004</td>
<td>10 symptomatic, 10 asymptomatic athletes</td>
<td>Breath hydrogen</td>
<td>90 min of cycling or running at 70% of maximal power</td>
<td>Exercise increased oro-caecal transit time, greater effect for running than cycling</td>
</tr>
</tbody>
</table>
Holdstock et al. (1970) noted that mass movements in the colon were increased and a call to defaecate was initiated by a 2% grade at 5.6 km·hr⁻¹ vs. sitting; there was a decrease in transit time with exercise, greatest amongst those with the slowest resting transit time; however, a speeding of gastric emptying may have contributed to this finding.

**Acute effects of exercise upon the colon.**

Cammack et al. (1982) used both radioactive technetium (gastric emptying) and breath hydrogen excretion (small intestine) to study the passage of a solid meal; cycle ergometer exercise at a pulse rate of 120 bpm for 6 hr cut the half-time of gastric emptying from 1.5 to 1.2 hr, but small intestinal transit remained unchanged. The one discordant result was that of Keeling and Martin (1987); they looked at a breath hydrogen estimates of the oro-caecal transit time when walking up a 2% grade at 5.6 km·hr⁻¹ vs. sitting; there was a decrease in transit time with exercise, greatest amongst those with the slowest resting transit time; however, a speeding of gastric emptying may have contributed to this finding.
combination of eating breakfast, a change of posture, and modest walking around the hospital.

Transit through the gut is stimulated by migrating motor complex contractions. In dogs, an hour of treadmill running decreased colonic migrating motor complexes, but at the beginning of exercise a giant migrating complex often initiated defaecation (Dapoigny and Sarna, 1991). In a human radiotelemetry study, Evans et al. (1984) compared 10 exercisers with 10 controls, finding that contractions in the proximal jejunum were roughly halved in those taking a 4-hour, twelve-mile walk. On the other hand, 1 hr of treadmill running at 70-80% of maximal heart rate for 1 hr increased colonic contractions, as measured by a catheter containing a pressure transducer, from an irregular 1-4 cycles per min at rest to a regular 3-9 cycles per min in 6 of 10 volunteers during exercise; 5 of these 6 individuals who showed increased colonic activity were susceptible to athlete’s diarrhoea (Cheskin et al., 1992). Another report noted that during graded cycle ergometer exercise to 75% of maximal oxygen intake, colonic phasic activity was decreased, reducing resistance to colonic flow, and that following such exercise an increase of propagated activity led to expulsion of the colonic contents (Rao et al., 1999).

**Chronic exercise and gastrointestinal transit.** Of 10 published reports, 5 found no effect of chronic exercise upon gastrointestinal transit, but 5 other investigations observed a speeding of transit. Thus, Coenen et al. (1992) found no difference in oro-anal transit when 3 days of moderate jogging were compared with 3 days of sedentary living, although the weight of stools was increased when exercising. Likewise, moderate exercise (1 hr of treadmill walking at 4.5 km-hr⁻¹ on 3 days) had no significant effect on either total or colonic transit time in sedentary men (Robertson et al., 1993). Bingham and Cummings (1989) again found no changes in colonic or overall intestinal transit time when sedentary subjects took 6-9 wk of progressive aerobic training. Further, Kayaleh et al. (1996) observed no difference from resting oro-caecal transit time when 8 trained runners each ran a distance of 9.6 km in one hour. Finally, Seboué et al. (1995) observed no difference of colonic transit time between 11 soccer players who were training 15 hr wk⁻¹ and also paying at least one soccer match and 9 relatively inactive radiology student technicians.

In contrast, Cordain et al. (1986), using the less reliable carmine dye estimate of GI transit time, found a speeding of transit with 6 weeks of aerobic running. Likewise, the colonic transit time of elderly men was almost doubled when recreationally active individuals were asked to remain temporarily inactive (Liu, et al., 1993); this response was due largely to a slowing of passage through right and left segments of colon. Song et al., (2012) used accelerometers to make a 3-level classification of habitual physical activity; in women, but not in men, moderate and high levels of physical activity were associated with a faster colon transit time than that seen in the least active group. Again, De Schryver et al. (2005) had half of a group of middle-aged and previously inactive subjects with tendency to constipation engage in a daily programme that included a 30 min daily walk and 11 min of home-based exercises; this amount of exercise was sufficient to halve their colonic transit.
How fast must you trot? Vigorous exercise and diarrhoea.

In support of the visceral ischaemia hypothesis, training increase the visceral blood flow at any given absolute intensity of exercise (Clausen, 1977) and this could explain why the risk of exercise-induced diarrhoea is decreased with training and/or a decrease in the relative intensity of effort (Fogoros, 1980). However, several factors argue against ischaemic damage to the gut being the primary cause of exercise-induced diarrhoea. Defaecation often occurs before the runner is exhausted. Only a small proportion of those affected report bloody stools, and in some instances the runner completes the event despite the unwanted defaecation. Nevertheless, a lesser degree of visceral hypoxia may be a contributing factor. The decrease in visceral blood flow reduces absorption of fluids and carbohydrates such as methyl glucose and xylose, allowing such substances to accumulate in the colon, and problems may be exacerbated by reperfusion damage and a leaky intestinal mucosa (Ter Steege et al., 2008).

Visceral ischaemia. During prolonged and vigorous exercise, particularly in a warm environment, increased activity of the sympathetic nervous system diverts up to 80% of the visceral blood flow to the skin and working muscles (Clausen, 1977; Qamar and Read, 1987; Rowell et al., 1964; Schaub et al., 1985; Sullivan, 1984). The risk of visceral ischaemia is increased if an athlete incurs a fluid loss equivalent to more than 4% of body mass (Rehrer et al., 1989), and this trend is exacerbated by changes in erythrocyte deformability, blood viscosity and increasing platelet aggregation (Heer et al., 1987; Vandewalle et al., 1988). Intestinal damage, with pain, diarrhoea and rectal bleeding may result (Moses, 2005).

Mechanical factors. A mechanical contribution to exercise-induced diarrhoea is suggested by the greater prevalence of the problem in running than in cycling or swimming (Sullivan and Wong, 1992), the greater speeding of intestinal transit with running than with other forms of exercise (Oettlé, 1991), and the greater movement of the abdomen during running, as sensed by an accelerometer (Rehrer and Meijer, 1991).

A sympathetically-induced relaxation of the colon allows the passage of its contents into the rectum, particularly when visceral movements are induced by running (Cordain et al., 1986). Mechanical stimulation of the intestines may also lead to the release of hormones such as prostaglandins and vasoactive

time. Further, although a 13-week strength training programme that resulted in 41-45% increases in muscle strength had no effect upon the oro-caecal transit time, it doubled the speed of whole bowel transit (Koffler et al., 1992).

Summary. An acute bout of exercise generally leads to a slowing or oro-caecal transit. On the other hand, the colon is stimulated by acute exercvise, and the initiation of migrating motor complex contractions may provoke defaecation. Opinions on the effect of chronic exercise are divided, with a half of investigations showing no effect, and the remainder a speeding of gastrointestinal transit. Although such changes could possibly contribute to exercise-induced diarrhoea, a telling argument against this viewpoint is the absence of any difference in either small intestinal or colonic transit times between athletes with exercise-induced diarrhoea and those without such problems (Rao et al., 2004).

Mechanical factors. A mechanical contribution to exercise-induced diarrhoea is suggested by the greater prevalence of the problem in running than in cycling or swimming (Sullivan and Wong, 1992), the greater speeding of intestinal transit with running than with other forms of exercise (Oettlé, 1991), and the greater movement of the abdomen during running, as sensed by an accelerometer (Rehrer and Meijer, 1991).
intestinal polypeptide (VIP), and the resulting secretions may increase the propensity to a watery diarrhea (Fahrenkrug et al., 1978; Hosssdorf et al., 1982; Hubel, 1985).

**Hormonal changes.** The circulating concentrations of various hormones that affect colonic motility and local blood flow (gastrin, secretin, pancreatic polypeptide, pancreastatin, VIP, neurokinin A, motilin, prostanlandins, and peptide YY) are all modified by exercise (Banks et al., 1985; Bunt, 1986; Greenberg et al., 1986; Hilsted et al. 1980; Martins et al., 2007; O'Connor et al., 1995; Øktedalen et al., 1992; Rehrer, 1990; Sullivan et al., 1984), partly in response to the mechanical stimulation discussed above, and partly as a consequence of visceral ischaemia (see Table 3). It is less clear how far these and other exercise-induced hormonal changes contribute to runner's diarrhoea (Peters et al., 2001), although a number of mechanisms can be envisaged.

A release of opioids with prolonged aerobic exercise can act on gut opioid receptors. In general, this slows intestinal transit (Cammack et al., 1982; Chapman et al., 1950), but the opioids can enhance antral contractions and thus oro-caecal transit (Neely, 1969).

Gastrin and motilin concentrations are increased during exercise (Sullivan, Wong and Heidenheim 1984). Gastrin primarily controls gastric emptying, but it also influences the action of the ileocaecal valve. Mechanical stimuli cause the release of VIP, and concentrations can rise to high levels if endurance exercise is combined with dehydration. VIP contributes to watery diarrhoea by decreasing the small intestinal absorption of fluids and sodium, and by stimulating the intestinal secretion of water, sodium, chloride, and possibly bicarbonate into the gut (Kane et al., 1983; Krejs et al., 1980; Mailman, 1978), and increasing colonic contraction (Eklund et al., 1979; Mailman, 1978). VIP and peptide histidine isoleucine also decrease internal anal sphincter pressure (Nurko et al., 1989). Secretin controls water balance, provoking the release of a watery secretion from the pancreatic and bile duct epithelia. Pancreatic polypeptide also modifies intestinal secretions. Neurokinin A stimulates smooth muscle. Motilin stimulates migratory motor complexes and contraction of the intestinal muscle (Rennie et al., 1979). Colonic relaxation is stimulated by an exercise-induced fall in insulin levels, and by increased pancreatic polypeptide (Galbo, 1983; Tache, 1984). Increases of prostaglandin (PG)E1, PGF1 and 6-keto PGF are found after running a marathon (Demers et al., 1981). Prostaglandins are released by weak mechanical stimulation of the jejunal mucosa, with a transmucosal shift of tritiated water (Beubler and Juan, 1978). PGE and PGF accelerate intestinal transit and decrease colonic contraction. PGE2 also initiates giant migrating contractions that are often associated with defaecation (Staumont et al., 1988). Splanchnic vasoconstriction also increases the production of peptide YY, which increases small intestine motility and contraction in the dog (Buell and Harding, 1989).

**Electrolyte imbalance.** Changes of electrolyte balance associated with the heavy sweating of prolonged endurance activity may irritate the colon and increase its motility (Keeffe et al., 1984).
How fast must you trot? Vigorous exercise and diarrhoea.

Table 3: Changes in hormone concentrations during vigorous aerobic exercise, and their effects upon the gastrointestinal tract.

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrin</td>
<td>Controls gastric emptying and modulates action of ileocaecal valve</td>
</tr>
<tr>
<td>Motilin</td>
<td>Stimulates migratory motor complexes and contraction of intestinal muscle</td>
</tr>
<tr>
<td>Neurokinin A</td>
<td>Stimulates smooth muscle</td>
</tr>
<tr>
<td>Opioids</td>
<td>Slows intestinal transit, but stimulates gastric antrum</td>
</tr>
<tr>
<td>Pancreatic polypeptide</td>
<td>Modifies intestinal secretions, causes colonic relaxation</td>
</tr>
<tr>
<td>Peptide YY</td>
<td>Increases small intestine motility and contraction</td>
</tr>
<tr>
<td>Prostaglandins</td>
<td>PGE and PGF speed intestinal transit but decrease colonic contractions; PGE2 initiates giant migrating contractions associated with defaecation</td>
</tr>
<tr>
<td>Secretin</td>
<td>Provokes watery secretion from pancreas and bile duct</td>
</tr>
<tr>
<td>Vasoactive intestinal polypeptide (VIP)</td>
<td>Decreases intestinal absorption of fluids and sodium, stimulates secretion of water, sodium, chloride and possibly bicarbonate into the gut, increases colonic contraction, decrease internal anal sphincter pressure</td>
</tr>
</tbody>
</table>

Stress and autonomic factors. The effects of anxiety and mental stress upon bowel function are well recognized. In general, stress delays oro-caecal transit (O'Brien et al., 1985), but it also increases colonic motility (Barone et al., 1986). Thus, in rats, restraint stress slows passage through the small intestine, but it speeds movement through the large intestine, and also initiates gastrointestinal secretory responses (Lenz et al., 1988). This reaction may be initiated by corticorelin (corticotropin releasing factor). Anxiety can also exacerbate the shift of blood flow away from the intestines. Many athletes develop symptoms before they begin to exercise, and a third of them report similar symptoms when they are emotionally distressed (Sullivan et al., 1994).

Moderate exercise increases parasympathetic nerve activity, stimulating colonic motor activity; however, problems of exercise-induced diarrhoea generally appear at higher intensities of effort, when sympathetic nerve involvement is dominant (Cammack et al., 1982; Read and Houghton, 1989). The combination of exercise and stress decreases parasympathetic tone, accelerating passage of the colonic contents. In support of the involvement of autonomic factors, sympathetic nerve activity is less at any given intensity of effort after training, and could thus explain the reduction of symptoms with training. On the other hand, emotional stress can hardly explain the diarrhoea that some runners encounter during normal training sessions.

Differential diagnosis

Before dismissing diarrhoea as a side effect of prolonged endurance exercise, it is important to eliminate other more serious pathologies. Athlete's diarrhoea is essentially a diagnosis of exclusion (Table 4). Particularly if the diarrhoea is a unique event of recent onset, it is likely to have been incurred through the ingestion of contaminated water or food. Indeed, this is one of the commonest issues confronting sports physicians at international events (DuPont et al., 2009; Karageanes, 2007; Shephard, 2000; Tillett and Loosemore, 2009). Problems may also arise from swimming in infected water (van Asperen et al., 1998; Weber,
How fast must you trot? Vigorous exercise and diarrhoea.

2005), or being splashed with contaminated mud (Griffiths et al., 2010; Mexia et al., 2013; Stuart et al. 2010).

Table 4: Conditions to be considered in the differential diagnosis of exercise-induced diarrhoea.

- Ingestion of contaminated food or water
- Swimming in infected water
- Splashing with infected mud
- Atherosclerosis or infarction of mesenteric artery
- Mesenteric thrombosis
- Haemorrhoids, anal fissures and fistulae
- Inflammatory bowel disease
- Malabsorption syndromes
- Biliary and pancreatic disorders
- Irritable bowel syndrome
- Pseudomembranous colitis
- Colon cancer
- Traveller’s diarrhoea
- General infections such as influenza
- Inappropriate diet, medications and supplements
- Thyrotoxicosis

Ho (2009) has published an algorithm to help in differential diagnosis. In addition to exercise, visceral ischaemia can reflect infarction or atherosclerosis of the mesenteric artery or venous thrombosis. Diarrhoea and rectal bleeding may be caused by peri-anal disease (haemorrhoids, anal fissures and fistulae), inflammatory bowel disease, various malabsorption syndromes, biliary and pancreatic disorders, irritable bowel syndrome, pseudomembranous colitis, colon cancer, traveller's diarrhea, and other infections such as influenza, an inappropriate diet, medications and dietary supplements, and even systemic diseases such as thyrotoxicosis must all be included in the differential diagnosis.

It is also important to recognize that participating in high intensity exercise can exacerbate conditions such as inflammatory bowel disease and post-radiation colitis.

Complications and counter-measures

Exercise-induced diarrhoea is in general more of a nuisance than a life-threatening condition. Nevertheless, it can have an adverse impact upon physical performance and motivation, and if severe and accompanied by substantial bleeding, it can contribute to athlete’s anaemia, dehydration, heat injury, rhabdomyolysis, and acute tubular necrosis (Fogoros, 1980). After addressing any obvious risk factors, countermeasures include organizational preparations, modifications of training and diet, and medical measures.

Organizational preparations.

Perhaps the most important action of the race organizers is to ensure that there are an adequate number of toilet facilities along the race route. Provision of adequate opportunities for rehydration may also reduce the risk of visceral ischaemia.

Training. If exercise is provoking diarrhoea, the immediate intensity of training should be reduced for 1-2 weeks, while an attempt is made to allow the body to adapt to the demands of competition (Brouns and Becker, 1993; Butcher, 1993; Murray, 2006). Cross-training may be helpful in building up physical condition; for example, a runner may add cycling, swimming or rowing to the conditioning regimen. Heat acclimation may also be helpful.

Diarrhoea may be precipitated by contraction of the abdominal muscles, for example when climbing a steep slope. Clothing that is too tight around the waist may also increase the risk of diarrhoea.
How fast must you trot? Vigorous exercise and diarrhoea.

**Diet.** Often, simple dietary changes can do much to avoid exercise-induced diarrhoea. A careful review of the runner’s diet should look for an excessive intake of fibre and the use of dietary supplements that are liable to provoke diarrhoea (above). There may be an advantage in eating a semi-hydrolyzed diet, particularly if the intestinal epithelium has been compromised by frequent bouts of visceral ischaemia (Bounos and McArdle, 1990). If there is evidence of lactose intolerance, it may be helpful to switch to lactose-reduced or lactose-free milk and milk products.

Food intake should be limited, and caffeine avoided in immediate preparation for an event (Murray, 2006). Runners typically opt for a low fibre, low residue diet, and omit gas-producing food items such as beans for 2-3 days before competition (Ho, 2009). At least one day before an event it is also wise to restrict the intake of the sugar alcohols often found in sugar-free candies, gum and ice cream. All solid food should be avoided for 2-5 hours before competition (Keeffe et al., 1984), and it is wise to empty the colon shortly before competition.

Since feeding normally increases gut flow, the ingestion of small amounts of fluid during an event may help to avert visceral ischaemia. Dehydration can exacerbate diarrhoea. If there is a history of diarrhoea, it is advisable to choose replacement fluids that will match mineral losses in the faeces rather than in sweat. Before, during and after running, the intake of fluids should be sufficient to maintain hydration. Energy gels and energy bars should be used cautiously while running, as these appear to produce diarrhoea in some people.

Any desired consumption of high-fibre and gas-producing foods such as beans, bran, fruit and salad should be deferred until after completion of the race.

**Medical measures.** In addition to undertaking a careful differential diagnosis, the physician should look for predisposing factors such as inflammatory bowel disease, irritable bowel syndrome or abdominal irradiation. If those with a history of inflammatory bowel disease, heavy exercise should be avoided when the disease is active (Ho, 2009). The anti-androgen drug flutamide currently used in treating prostate cancer induces diarrhoea, in part through a lactose intolerance mechanism. Abdominal irradiation may also induce a colitis. Irradiation damage can be minimized, at least in rats, by a high intake of glutamine and arginine both before and after irradiation (Ersin et al., 2000).

If the exercise-induced diarrhoea is severe, the stools should be checked for occult blood, and the haemoglobin level examined for signs of athletes' anaemia. The use of NSAIDS such as ibuprofen also needs to be curbed, particularly if the diarrhoea is bloody.

Self-medication must be evaluated carefully. Riddoch and Trinick (1988) found runners engaging in various forms of self-medication, including the use of kaolin, morphine, codeine phosphate, antacid tablets, and anti-diarrhoea tablets.

Some medically prescribed antispasmodic preparations such as Lomotil (diphenoxylate with atropine) have anticholinergic effects that inhibit sweating, thus increasing the risk of heat stress (Priebe and Priebe, 1984). Most of the adverse effects of VIP release can certainly be countered by administering atropine (Mailman, 1978), but use of this
drug is unwise, because it blocks sweating.

Low doses of the piperidine derivative Loperamide (Imodium) are effective in countering prostaglandin-induced diarrhoea in both acute and chronic cases (Lange et al., 1977). Loperamide is a peripheral opioid agonist, but it does not affect the central nervous system; it blocks the myenteric plexus, decreasing the tone of the intestinal wall, slowing gastrointestinal transit and allowing more water absorption. It also suppresses colic mass movements and the gastro-colic reflex. At higher doses, it also blocks calmodulin, a calcium binding messenger protein, with functions that include inflammation, metabolism, apoptosis, smooth muscle contraction, and the intracellular movement of minerals. Unlike other opioids, there is no evidence that tolerance develops with prolonged use of loperamide. A new calmodulin antagonist, zaldaride malate, is currently undergoing evaluation. Histamine H₂ receptor antagonists and proton pump inhibitors may be effective in athletes with bloody diarrhea caused by hemorrhagic gastritis (Baska et al., 1990).

**Practical implications and conclusions**

Exercise-induced diarrhoea is a potential problem for many active individuals, both dedicated long-distance runners and those with clinical issues such as inflammatory bowel disease, the irritable bowel syndrome or the after-effects of treatment for prostate cancer. Although the condition usually does not have major consequences for health, it can predispose to fluid and mineral imbalance and cause athlete’s anaemia. Moreover, the resulting social embarrassment may have a strongly negative impact upon motivation to exercise in the person concerned. Factors contributing to diarrhoea include an inappropriate choice of diet, mechanical stimulation of the colon, visceral ischaemia, exercise-induced hormonal changes, and pre-race stress and anxiety. After excluding more serious underlying pathologies, the management of exercise-induced diarrhoea stresses optimization of diet, enhancement of physical condition and heat acclimation by cross-training, emptying of the colon before competition, maintenance of hydration, and provision of toilet facilities along the race route. Moderate doses of loperamide may be considered for individuals with persistent problems, but it is important to avoid medications that reduce either brain function (opioids) or sweating (atropine preparations).

**Author's Qualifications**

The author’s qualifications are Roy J. Shephard, C.M., M.B.B.S., M.D.[Lond.], Ph.D., D.P.E., LL.D., D.Sc.

**References**


How fast must you trot? Vigorous exercise and diarrhoea.


How fast must you trot? Vigorous exercise and diarrhoea.


How fast must you trot? Vigorous exercise and diarrhoea.


