ORIGINAL ARTICLE

Objective evaluation of small bowel and colonic transit time using pH telemetry in athletes with gastrointestinal symptoms

K A Rao, E Yazaki, D F Evans, R Carbon

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See end of article for authors' affiliations

Correspondence to: Dr K Arjun Rao, Specialist Sportscare WA, Sports Medicine, 220 Whatley Crescent, Maylands, WA 6051, Australia; a.rao@bigpond.net.au

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Background: Gastrointestinal (GI) disturbances are often reported by long distance runners and are more common in women, particularly after prolonged high intensity exercise.
Objectives: To determine whether these symptoms could be associated with alterations in GI motility.
Methods: Small bowel and colonic transit were measured using pH telemetry in a group of 11 female athletes (age 22 to 53 years), six of whom experienced lower GI symptoms during exercise. Subjects participated in two experimental sessions: a control measurement, where small bowel transit was estimated during a rest period (R) of six hours; and an exercise session (E), where small bowel transit was measured during a one hour period of high intensity exercise (cross country running) at >70% $\dot{V}O_2$max. Colonic transit was estimated indirectly from determinations of whole gut transit time by radio-opaque marker.
Results: Small bowel transit time was 3.5 to 10.6 h (R) and 3.0 to 8.7 h (E) in asymptomatic athletes, versus 4.0 to 6.6 h (R) and 4.6 to 7.3 h (E) in symptomatic athletes (NS). Colonic transit time was 35.0 to 62.5 h (R) and 30.5 to 70.9 h (E) in asymptomatic athletes versus 20.4 to 42.9 h (R) and 21.5 to 67.2 h (E) in symptomatic athletes (NS).
Conclusions: Small bowel and colonic transit times were similar in the two groups in the rest and exercise sessions. The diarrhoea seen in this study did not result from accelerated colonic transit. Other mechanisms must be sought.

Lower gastrointestinal (GI) tract symptoms during running (lower abdominal cramps, urge to defecate, the need to stop for a bowel movement, diarrhoea, rectal incontinence and bleeding) tend to occur more often in women and after high intensity running exercise.[1 2] However, the reasons for such symptoms remain uncertain. In addition there may well be a ''conditioning'' effect of training on the gut with symptoms settling after an initial period of training or after incremental training loads, and possibly an inverse relation with age.[1 2]

Several aetiological mechanisms have been proposed to account for the development of lower GI tract symptoms during exercise. These include intestinal ischaemia, mechanical trauma, stress, alterations in GI absorption or permeability, and neuroendocrine changes.

It has been speculated that alterations in GI motility may be primarily responsible for these problems, particularly runner's diarrhoea or ''trots.''[3] Evans *et al* reported that the migrating myoelectric complexes (MMC) of the small bowel were abolished or significantly decreased during three to four hours of moderate exercise (a 19 km walk).[4] As actual measurements of motility require sophisticated technological equipment together with appropriate expertise, the emphasis has been placed more on indirect methods of assessment—that is, the estimation of GI transit time. Read *et al* investigated the relation between the motor activity of the small bowel and the transit time.[5] They showed that GI transit of a solid meal was related to both fed and fasted intraluminal pressure activity in the bowel. In addition there was a significant negative correlation between contractile activity and transit time. Physical exercise is often recommended for the treatment of chronic constipation.[6] As diarrhoea and bowel irritability are common complaints among long distance runners, this suggests that exercise shortens GI transit time, perhaps in association with a general increase in motility. However, there are difficulties in relating gut contractility to propulsion.

Clinically, the consequences of a significant acceleration or delay in small intestinal transit are likely to be as follows. A rapid rise in transit may lead to incomplete absorption of available small intestinal nutrients with loss of the essential circulatory metabolites required to fuel exercising muscles. A secondary effect may be the dumping of small intestinal contents into the proximal colon, which, together with bile salts (which are known to be cathartics), may give rise to diarrhoea. This is therefore a possible culprit for ''jogger's trots.''[3] Alternatively, the presence of luminal content within the small intestine may give rise to symptoms of bloating and abdominal pain, which are commonly experienced by athletes undergoing prolonged intensive exercise. A significant delay in small intestinal transit could give rise to another problem—stasis of chyme may stimulate splanchnic blood flow, thereby depriving the somatic circulation and causing loss of performance.

Despite technical advances in the past decade, comparatively little is known about the effect of exercise on transit time through the GI tract. There are conflicting data on small bowel transit, with studies that show acceleration, delay, or no change during various exercise regimens.[6–8] This uncertainty could reflect physiological differences in the subjects and differences in measurement technique. For example, with the breath hydrogen (H_2) method,[6–8] lactulose is used as a liquid meal, and this hypertonic polysaccharide accelerates small bowel transit time.[9] A further confusing factor is that

Abbreviations: MATT, mouth to anus transit time; MMC, migrating myoelectric complex; RTC, radio-telemetry capsule; $\dot{V}O_2$max, maximum oxygen consumption

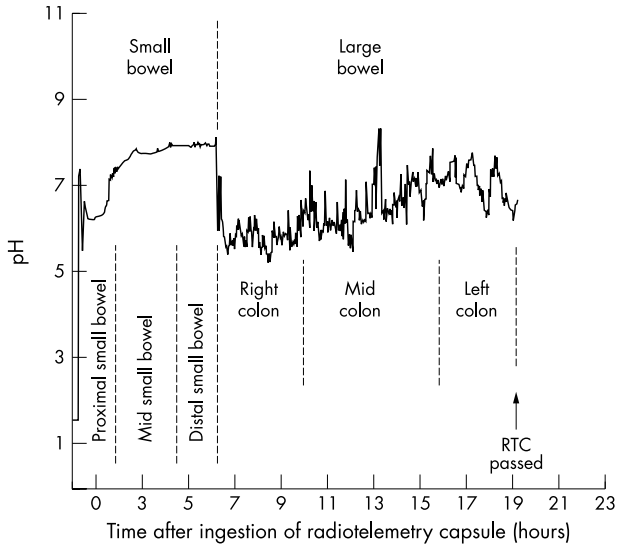

Figure 1 The gastrointestinal pH profile method using a radio-telemetry capsule.

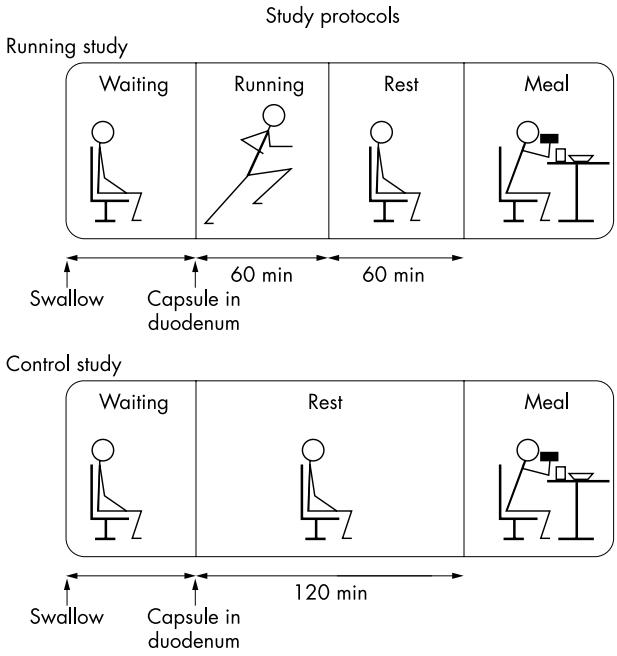

Figure 2 Study protocols.

exercise in itself causes a reduction in breath H_2 concentration.[10] Also, variability in gastric transit may reduce the sensitivity of the test.

Other techniques have been developed including the pH profile method,[11] which may be a more accurate way of assessing GI transit. The pH profile method has the advantage that subjects can be freely ambulant during

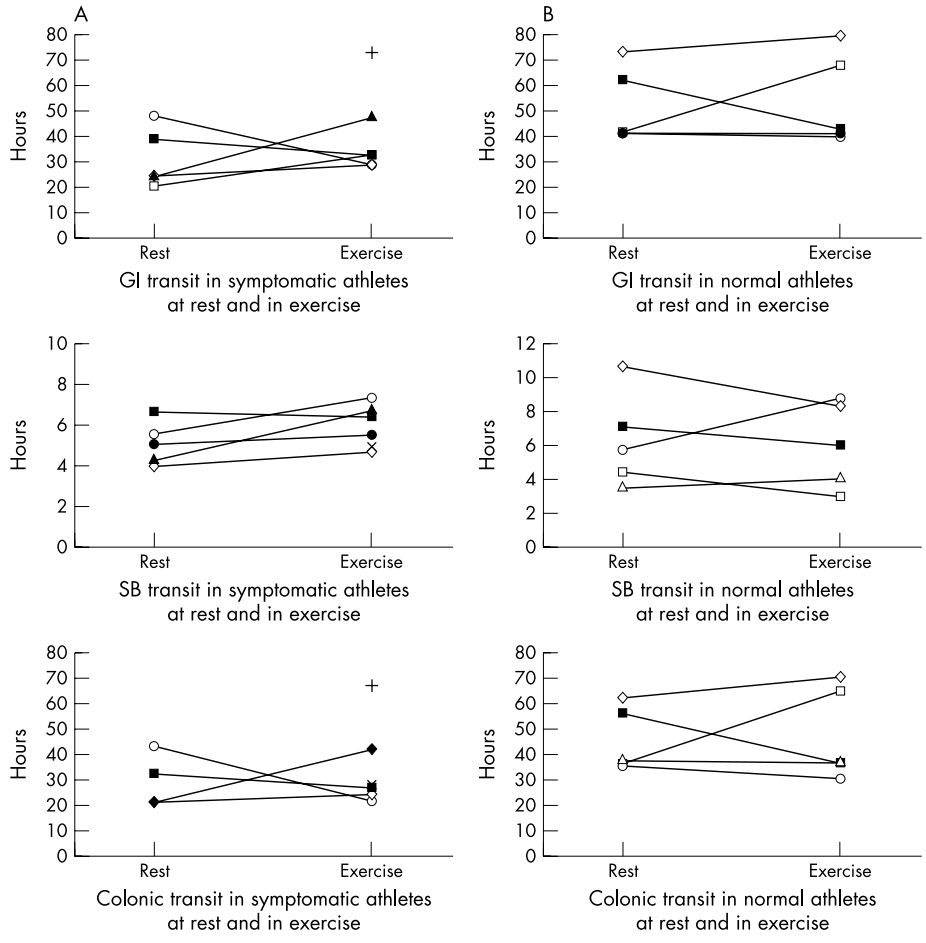

Figure 3 Gut transit values for symptomatic athletes (A) and control (''normal'') athletes (B), at rest and with exercise. Top panels: whole gut (GI) transit; middle panels: small bowel (SB) transit; bottom panels: colonic transit.

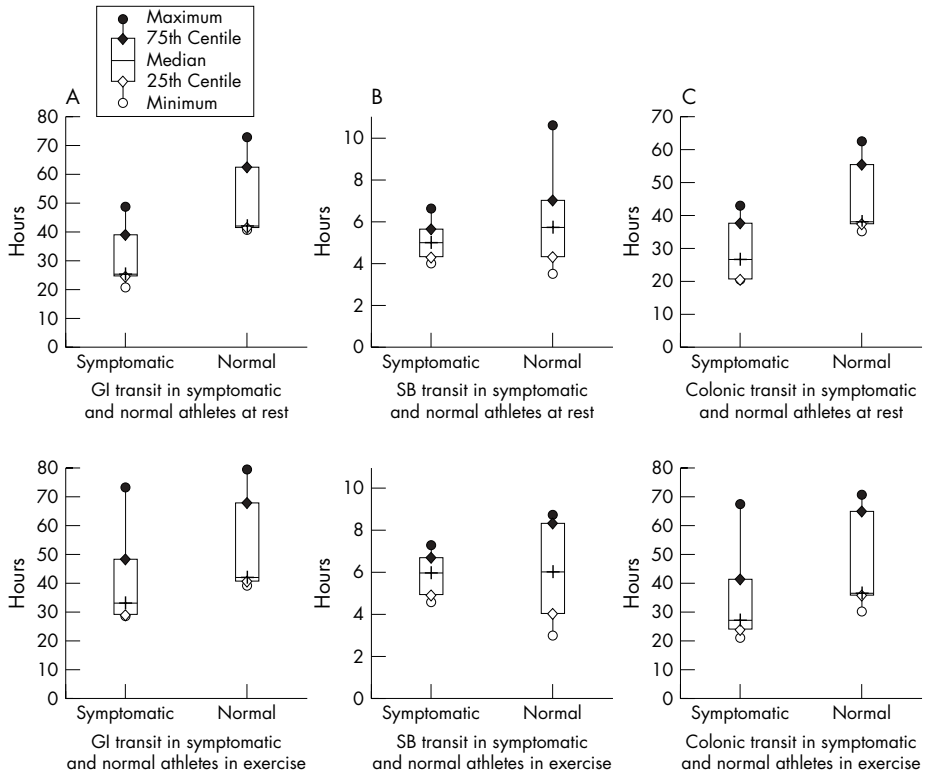

Figure 4 Box and whisker plots of gut transit values during rest sessions (top panels) and exercise sessions (bottom panels) in symptomatic and control (''normal'') athletes. (A) Whole gut (GI) transit. (B) Small bowel (SB) transit. (C) Colonic transit.

studies. Additional information concerning the colon and rectum can also be attained by following the passage of the pH capsules beyond the terminal ileum.[11] It is therefore possible to measure small bowel transit time under real exercise conditions, without the additional problem of unknown gastric residence time that is encountered during the breath H_2 technique.

With regard to the colon even less is known about the relation between colonic motility and exercise. Again, the results of the few existing studies are conflicting: both orocaecal (breath H_2 method) and whole gut transit time have been shown to be unchanged, accelerated, or delayed.[12–14] Uncontrolled variables have included the population recruited, the methods used, and the type and intensity of the exercise and diet.

In view of the paucity of data regarding GI function and exercise, the major aims of this study were:

- to investigate the effect of exercise on small bowel and colonic transit time under fasted and normal ambulatory conditions, using a combination of the pH profile method and radio-opaque marker techniques;
- to compare GI transit time from two subject groups: a group of athletes complaining of bowel related symptoms during exercise; and a normal healthy age and sex matched control group.

METHODS

Subjects

The two groups of subjects were as follows. The index group consisted of seven female runners, aged between 34 and 53 years (mean 40 years), who experienced lower GI symptoms—for example, lower abdominal cramping, urge to defecate, increased frequency of bowel movements, diarrhoea and rectal bleeding—during or shortly after exercise. The control group consisted of six female runners aged between 22 and 51 years (mean 31 years), and who experienced no GI symptoms during either training or competition.

All subjects were healthy and non-pregnant: Those not currently on oral contraception had a negative pregnancy test, and the time of their last menstrual period was recorded.

As the menstrual cycle plays an important role in determining the small bowel transit time in normally menstruating women,[15] the subjects participated in the study during the same phase of their individual menstrual cycles.

The experimental protocol described in this study was approved by the Tower Hamlets district ethics committee, and written informed consent was obtained from each subject before the experiments were carried out.

Study design

Each subject took part in two separate studies: an exercise study and a resting study. The exercise study comprised a one hour run followed by a one hour rest. The resting study involved resting throughout the study period. Subjects were not told whether they would run or rest until the point at which the study began.

GI transit time determination: pH profile method

Transit along the small intestine was measured using a pH sensitive radio-telemetry capsule (RTC). The RTC consists of a glass electrode with an integral reference cap and battery. Radiotransmissions from the capsule are detected by an aerial belt worn around the subject's abdomen and transmitted to a portable solid state receiver. The recorder samples the pH from the capsule at six second intervals, thus enabling continuous ambulatory monitoring for up to 24 hours.

A previously calibrated pH sensitive radio-pill (type 7006 Remote Control Systems, London, UK) was swallowed by the subject (the pill moves freely through the GI tract). For a

period of 24 hours the pH signal was continuously monitored during exercise and during normal ambulant activities with an aerial belt attached to a solid state recorder (Medilog 1000, Oakfield Instruments Ltd, Oxford, UK). The data were analysed by a computer with dedicated software.

Evans *et al* reported the characteristics of the human gut profile using a pH sensitive radio-pill.[11] A sharp rise in pH value, from around pH 2 to pH 6 or more, indicates when the radio-pill moves into the duodenum from the stomach (fig 1). The pH value progressively rises to, and stabilises at, approximately pH 8 in the terminal ileum. When the radio-pill moves into the caecum there is a sharp fall in pH from pH 8 to pH 6 or below depending on the degree of fermentable carbohydrate in the luminal content of the caecum. Thus small bowel transit time can be identified with these changes in pH. Colonic transit can also be plotted by following the RTC around the colon until it is passed in the faeces.

The method requires the collection of faecal specimens until the radio-pill is retrieved. It is then cleaned and re-sterilised after checking for pH drift in calibration buffers (pH 4 and pH 9.2 at 37℃).

Whole gut transit

Mouth to anus transit time (MATT) was determined by modification of the Hinton/Metcalfe method.[16 17] Three different types of radio-opaque marker (squares, spicules, and circles) were used to determine MATT in control and exercise subjects. Subjects ingested 20 of these markers at predetermined times: minus 18 hours (squares), minus 12 hours (spicules), and 0 hours (circles) (the times relate to the start of the study). A diary was kept of the times the markers were taken and the times of faecal collection. All faecal specimens were collected into suitable sealed containers, then labelled with the subject's name, time, and date and stored in a cold room.

Each stool was flattened (to avoid one marker obscuring another) and then *x* rayed to determine marker content. The number of markers recovered was counted by two observers independently, and a mean value obtained. The marker content of each stool, together with the times the markers were taken, was used to calculate the transit time, as described by Arhan *et al*.[18]

$$\delta_t = \frac{\sum_{n=1}^{n} x_i \cdot t_i}{\sum_{n=1}^{n} x_i}$$

where δ_t = mean mouth to anus transit time, x_i = number of markers of type 1 (squares), type 2 (spicules), or type 3 (circles) passed at time t_i, and n is the total number of stools. The average mean mouth to anus transit time, from the individual transit times of the three types of marker in each person, was calculated using the following formula:

$$(average)\ \overline{\delta_t} = \frac{\overline{\delta_{t1}} + \overline{\delta_{t2}} + \overline{\delta_{t3}}}{3}$$

where δ_{t1}, δ_{t2}, and δ_{t3} are the mean mouth to anus transit times for the type 1, type 2, and type 3 markers, respectively.

From knowledge of both small bowel transit time and mouth to anus transit time, colonic transit time can be estimated.

Experimental protocols

The experimental protocols are illustrated in fig 2. Following an overnight fast and ingestion of the first two batches of radio-opaque markers (squares and spicules), the subject swallowed a previously calibrated radio-pill with up to 500 ml of water. The aerial belt and recorder were then applied and the subject sat on a chair or lay on a bed on the right side in order to encourage transit of the pill into the duodenum. The third batch of radio-opaque markers (circles) was also taken at this time. At this point some subjects were asked to rest while continuous measurements of pH were made. Two hours after the pill passed into the duodenum, each subject was given a normal but standardised meal comprising two sandwiches and a soft drink. This was to encourage movement of the radio-pill along the small bowel, as it is known that the radio-pill occasionally remains stationary in the terminal ileum.[11] Another group of subjects was asked to run for 60 minutes at an intensity of approximately 70% of maximum oxygen consumption ($\dot{V}O_2$max) while continuous measurements of pH were made. On completion of the run, the subject rested for an hour and was allowed to drink water ad libitum. After this, the subject was given the same standard meal as in the rest session.

Subjects were then allowed to leave the laboratory, and pH measurements were continued overnight. Subjects resumed their normal ambulant activities except that they were not allowed to undergo any training during this period. After 24 hours they removed the recorder but were asked to collect all stools until the radio-pill was recovered. They were also required to collect an additional faecal specimen soon after this to ensure retrieval of most of the markers. Normal training could now be resumed.

Statistical analysis

Statistical analysis was done using Wilcoxon rank sum and Mann–Whitney U tests, with the level of significance set at $p<0.05$.

RESULTS

Lower GI symptoms, particularly diarrhoea and an urge to defecate, were reproduced and thereby confirmed in five of the six subjects in the symptomatic group during the exercise sessions, with no symptoms during the rest sessions (two actually defecated during the run and three shortly afterwards). Three of the six subjects in the symptomatic group also experienced rectal bleeding and lower abdominal pain during the exercise session.

Individual measurements of transit time for the whole gut, the small bowel, and the colon are shown in fig 3 for symptomatic athletes (A) and control (asymptomatic) athletes (B) during the rest period and the exercise session. Comparative box and whisker plots in fig 4 illustrate the range of values for whole gut transit, small bowel transit, and colonic transit between the symptomatic and control athletes during the rest period and in the exercise session. The data are expressed as median, 25th, and 75th centiles, with ranges for the two groups. The probabilities (p values) indicated for each group in the transit studies were obtained using the Wilcoxon rank sum test and the Mann-Whitney test to determine significance levels from the appropriate statistical tables.

Transit studies

One of the symptomatic subjects failed to complete the rest period because the RTC did not enter the duodenum within the normal time frame (+ symbol in fig 3). The data from one of the athletes in the symptomatic group were also not included because of failure to retrieve the radio-pill and inappropriate collection of faecal specimens.

Small bowel transit

Small bowel transit varied between 3.5 and 10.6 hours in both study groups. The measured small bowel transit variation in control athletes during the rest period was 3.5 to 10.6 hours, and with exercise it was 3.0 to 8.7 hours ($p = 0.91$); the corresponding transit times in the symptomatic athletes were 4.0 to 6.6 hours during the rest period, and 4.6 to 7.3 hours with exercise ($p = 0.27$).

Colonic transit

There was no significant difference in colonic transit in the symptomatic athletes during the rest or exercise sessions. In control athletes the estimated colonic transit times were 35.0 to 62.5 hours during the rest period, and 30.5 to 70.9 hours with exercise ($p = 0.91$); the corresponding transit times in the symptomatic athletes were 20.4 to 42.9 hours during rest period and 21.5 to 67.2 hours with exercise ($p = 0.52$).

DISCUSSION

Our aim in this study was to determine whether significant alterations in either small bowel or colonic transit during dynamic exercise might account for the increased bowel movement and pain reported by long distance runners. Our main finding was GI transit times were similar in the five control athletes and the six symptomatic athletes, five of whom had lower GI symptoms during their exercise.

Davis *et al* have shown that small bowel transit using radiotelemetry capsules (RTCs) should usually be between three and six hours (maximum) in normal fasted subjects.[19] Our results suggest that, using the Davis criteria, small bowel transit time was unaffected by exercise in the symptomatic group. Small bowel transit in the control group was more variable. Both physiological differences and technique may explain the conflicting results. Comparative studies of small bowel transit times during exercise are as conflicting as gastric emptying, with equal numbers of studies showing acceleration, delay, or no change in transit during various exercise regimens.[20]

The relation between small bowel transit and motility remains complex. Motility of the stomach and small intestine is governed by intrinsic control mechanisms in the myenteric plexus within the gut wall, which can be modulated by local intrinsic or extrinsic stimuli or by the central nervous system (CNS). Motility patterns are governed by food state and are now well understood. In the fasting state the stomach and small intestine are controlled by a cyclical pattern of contractile activity, the inter-digestive MMC, and this is abolished and replaced by a more random but continuous contractile pattern shortly after ingestion of a meal. The fed pattern continues for a period of two to six hours in the human depending on the volume, content, and energy density of the meal.

Evans *et al*,[4] using a pressure sensitive radio-telemetry capsule, reported a significant decrease or abolition of the MMC during a strenuous 12 mile walk. More recently Soffer *et al*,[21] using micro-transducers and a digital data logger, reported that cycling exercise altered the normal fed/fasting cycle of small intestinal motor activity in some subjects but only if exercise was intense (80% $\dot{V}O_2$max). However, switching to a fed cycle would imply an acceleration of transit time, which we did not demonstrate in this study. Clearly there are compounding factors which remain unknown, but it is likely that there are no major differences in small bowel transit to explain the accelerated whole gut transit in our symptomatic group.

The predominance of diarrhoea in runners and other athletes undertaking prolonged intense activity would place a colorectal cause high on the list of possible GI involvement. However, we must be cautious in making assumptions about organ related associations, as it is known that there are many non-colonic causes of diarrhoea. These may reflect dysfunction higher in the GI tract or may even be non-GI in nature. Two examples are the diarrhoea seen in ''dumping'' syndrome, a sequel of vagotomy, and the diarrhoea and the urgency to defecate during stressful events such as examinations or public speaking. In the second example, although a neuroendocrine cause could be postulated, a direct CNS effect is also highly likely.

Current opinion would suggest an alteration in colonic motor function as the main factor responsible for runner's diarrhoea. Studies of colonic function during exercise, as with the upper GI tract, are related to motility and transit. In a study by Bingham *et al*,[22] colonic transit, as measured by radio-opaque markers, was not altered by a moderate training schedule involving up to one hour of jogging a day for seven to nine weeks. Our findings in the present study support this.

As with small bowel transit, colonic transit studies have been conflicting, some showing an acceleration and others a delay or no change in colonic transit.[23] Variables that might be relevant include the methods used, the population recruited, and the type and intensity of exercise and diet. In the present study, diarrhoea did not appear to be related to alterations in measured GI transit. However, limitations of the study included the indirect measurement of colonic transit and no measurement of colonic motility, particularly at the time of symptom onset.

Alterations in colonic motility have been reported in response to a one hour jogging schedule to 80% $\dot{V}O_2$max in regularly exercising volunteers.[24] The investigators documented an increase in the dominant frequency and propagation of colonic contractions using microtransducers sited in the sigmoid colon, and this was related to the incidence of diarrhoea in some subjects. In dogs, a similar increase in colonic motility and propagation was reported by Dapoigny and Sarna,[25] and these investigators also found an increase in the defecation rate after exercise.

The overall effect of exercise on colonic motility and transit may be a general rise in intraluminal movement of colonic content giving rise to stimulation of propagated motility, caudal movement, and defecation as a result of rapid rectal filling. A colonic cause for runner's diarrhoea is therefore a likely candidate, and possible treatment may be to inhibit motility pharmacologically or decrease the luminal content with a low residue diet. It is likely that an interaction of different mechanisms affects colonic motility, thus producing GI disturbances. The mechanical action of running must also be involved, as these symptoms are not seen in non-weight-bearing exercise—for example, swimming and cycling.[26]

Reduction of splanchnic blood flow has been widely hypothesised as the main cause of symptoms. However, this is questionable, particularly as GI disturbances in the higher intensity sports and events such as middle distance races—where the athletes reach higher heart rates and static oxygen consumption—are far less common. Prostaglandins and other GI hormones may also be involved. Other features of the GI tract, such as absorption and permeability, may be affected by exercise, but their role remains speculative.

The exact roles of the various pathophysiological mechanisms remain elusive. More research is needed to improve our understanding of such a common complaint.

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Authors' affiliations

K A Rao, R Carbon, Department of Sports Medicine, Queen Mary and Westfield College, London E1, UK
E Yazaki, D F Evans, Wingate Institute, Barts and London School of Medicine, London E1, UK

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