Fat adaptation and glycogen restoration for prolonged cycling—recent studies from the Australian Institute of Sport

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Abstract Current sports nutrition strategies are based on the theory that body carbohydrate (CHO) stores are limited, and that extra CHO consumed before and during a workout will continue to make this important fuel available. These strategies have been shown to be effective in delaying the onset of fatigue and enhancing the performance of endurance sports (> 90 minutes duration). However, CHO consumed during prolonged exercise provides an additional fuel source for the muscle, rather than altering the rate of depletion of muscle glycogen stores. An alternative angle to enhance performance is to find a fuel source for exercise that could replace glycogen and slow its rate of use. The classical carbohydrate-loading studies of the 1960s found that short-term exposure to a high fat, low CHO diet caused dramatic reductions in muscle glycogen stores and decreased exercise capacity. By contrast, studies that lengthened the duration of this diet showed, with time, that the body adapts to CHO deprivation by increasing the utilisation of fat during exercise, and using the precious glycogen stores at a slower rate. These adaptations may occur after as little as five days of a high fat, low CHO diet. A recent Australian Institute of Sport study investigated whether fat-loading strategies provide additional benefits to the performance of endurance athletes. We exposed well trained athletes to five days of a high fat, low CHO diet, followed by one day of high CHO intake to refuel glycogen stores before a performance ride on day seven. The athletes also consumed a high CHO breakfast and drank sports drink throughout the performance test according to current sports nutrition guidelines. These strategies ensured that CHO availability was optimal. The fat adaptation diet caused major changes in fuel utilisation during sub-maximal exercise, with at least some of the adaptations persisting on day seven, even in the face of a plentiful CHO supply. As dramatic as these metabolic changes were, they failed to improve the performance of the cyclists' time trial. Together with other research, this study fails to find evidence that fat adaptation strategies offer any benefits for the endurance athlete. The only remaining question is whether there are any advantages for ultra-endurance athletes who compete in events undertaken at a lower intensity and for longer periods (e.g. four hours or more). For these athletes, fat is the predominant fuel source. **(Aust J Nutr Diet 2001;58 Suppl 1:S23–S27)**

Introduction

Studies show that a few days of eating a high fat, low carbohydrate (CHO) diet reduces resting muscle glycogen stores and impairs an athlete's capacity for prolonged (> 90 minutes), submaximal (approximately 70% $V0_2$ max) exercise (1–4). However, longer periods (more than seven days) on such diets are associated with metabolic adaptations that enhance fat oxidation during exercise and compensate for the reduced CHO availability (5,6). Although these 'fat adaptation' studies have reported reduced rates of muscle glycogen oxidation during exercise, this is not proof of glycogen sparing per se. These lower rates of glycogen utilisation during exercise might be explained by the low initial concentrations of muscle glycogen after high fat diets as much as by specific muscle adaptation (7). Nevertheless there is evidence of muscle adaptation after chronic exposure to high fat diets even in well trained subjects, with studies reporting an increase in some of the enzymes involved in fat transport and oxidation (8,9).

The effect of these metabolic adaptations on exercise capacity or performance during prolonged exercise is not clear. Although some studies have reported performance benefits (5,10), these results are not straightforward because of unusual features of the design of the study—for example the ordered allocation of treatments, or carry-over effects from various measurements of performance conducted in succession. Other studies have reported that long-term high fat diets produce no change in performance (6,11,12) or an impaired ability to adapt to a training program (13).

At the Australian Institute of Sport we recently have completed two studies of an alternative model involving fat adaptation. We were interested in the effects of a period of exposure to high fat, low CHO intake, followed by the restoration of muscle glycogen stores with a high CHO diet. Such 'dietary periodisation' aims to enhance the capacity of both glycolytic and lipolytic systems to oxidative metabolism during prolonged exercise, by increasing the contribution from fat to substrate metabolism while potentially sparing intact muscle glycogen stores. Although previous studies of fat adaptation have used two- to seven-week periods of adherence to high fat intakes (5,6,10,12,13), such diets are impractical for subjects to maintain and may interfere with an athlete's high intensity training program. However, a recent study indicated that significant increases in fat oxidation occurred after five days of a high fat, low CHO diet (11).

We decided to investigate this more fully, as five days is a more manageable period for radical dietary change and should minimise the potential health and training disadvantages arising from longer periods on high fat intakes. A one-day period of CHO recovery was chosen in an attempt to restore muscle glycogen concentrations without allowing a 'wash-out' of adaptations incurred by the high fat phase. We conducted two studies, both with the aim of investigating the effects of five days adaptation to a high fat diet followed by one day of CHO restoration,

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on metabolism and performance of endurance cycling (approximately 2.5 hours duration) on day seven.

The main differences between the studies were:

- study 1: to investigate baseline metabolic conditions and to provide an exercise situation that might benefit most from glycogen sparing, cyclists undertook the performance test on day seven without further CHO intake on the day (14); and,
- study 2: to investigate the nutrition strategies that are recommended and practised by most cyclists, subjects undertook the performance test after a high CHO breakfast and with the intake of a sports drink throughout the session.

Overview of methods

In both studies we recruited eight well-trained male cyclists or triathletes to undertake two separate trials. The study was undertaken with a counterbalanced cross-over design, and a two-week wash-out period between each trial. In each study we supplied all food to athletes, with at least one meal a day being eaten under supervision in the laboratory, and other meals or snacks eaten at home and recorded in a food diary. Training sessions also were prescribed for each athlete and undertaken under supervision. Each athlete completed the same training program in each of his trials.

Figure 1 summarises the dietary and training protocols undertaken in each of the studies.

On day six in each trial, after the testing session, subjects rested and consumed a high CHO diet [10 g/kg body mass (BM) per 24 hours] in preparation for testing on day seven. The two studies differed principally with regard to the nutrition on the performance day. In study 1, subjects fasted overnight and consumed water throughout the performance ride. In study 2, subjects consumed a CHO-rich breakfast (2 g CHO/kg BM) before the ride, then consumed a sports drink throughout the ride, providing a CHO intake of 0.8 g/kg BM/hour.

In the first study, the diets were presented openly to subjects. In our second study we attempted to blind the order of dietary treatments. We used three methods to disguise the diets in this study:

- we developed recipes to allow certain foods or dishes to be made in indistinguishable high fat or low fat forms. Muffins, curries, milk shakes and mousses were examples of foods that could be manipulated in this way;
- we added Polyjoule™ or fats (oils or cream) to dishes to modify the CHO or fat content;
- we used ‘decoy’ foods on each diet, placing perceived CHO-rich foods in conspicuous ways in high fat menus (e.g. bread, fruit, salads), and conversely, high fat foods (e.g. butter) were made obvious in the high CHO trial.

Figure 1. Overview of dietary and training protocols[a]

⇓ DAY 1	2	3	4	5	⇓ 6	7
High fat or high CHO	High fat or high CHO	High fat or high CHO	High fat or high CHO	High fat or high CHO	High CHO	Performance trial
20 min @ 70% $V0_2$ max + interval training	3–4 hour long slow ride	Hill ride 2–3 hours	Interval training	3–4 hour long slow ride	20 min @ 70% VO_2 max Rest	120 min @ 70% $V0_2$ max + 7kJ/kg time trial
⇓ Baseline					⇓ Adaptation	
Muscle biopsy Blood sample RER data					Muscle biopsy Blood samples RER data	

(a) CHO, carbohydrate; RER, respiratory exchange ratio. The dietary treatments were isocaloric [approximately 0.22 MJ/kg body mass (BM)] and provided the following macronutrient composition: high carbohydrate—9.6 g CHO/kg BM (74% of energy), 0.7 g fat/kg BM (13% of energy); fat adaptation—2.4 g CHO/kg (19% of energy), 4.0 g fat/kg (68% of energy)

At the end of each trial in study 2, subjects were asked to nominate which diet they had just received, when they were able to distinguish the diet, and which clues enabled them to make a definite decision.

We collected various metabolic and mechanistic data during each study, as outlined in Figures 1 and 2. In study 1, muscle glycogen concentrations were determined from biopsy samples collected before the first testing session on day one, and after five days of dietary adaptation, on the morning of day six. We collected additional biopsy samples before and after the two hours of steady state cycling on day seven, to determine the rate of muscle glycogen utilisation during this portion of the performance ride.

Blood samples and respiratory gas exchange data were collected during a 20-minute cycling session (70% $V0_2$ max) on the morning of days one and six of both studies. These measurements were also made at 20-minute intervals during the steady-state portion of the performance ride. From gas exchange data, the rates of fat and CHO oxidation during cycling were determined. In study 1 we attempted to further separate the contribution of substrates towards total fuel oxidation during the steady-state cycling bout on day seven. We used a continuous primed infusion of the stable tracer 6,6-^{2}H glucose to allow determination of plasma glucose kinetics, including whole body glucose disappearance (glucose Rd) during the ride. Muscle glycogen utilisation was then determined directly from changes in glycogen concentration in biopsy samples, and indirectly, by subtracting blood glucose Rd from total CHO oxidation.

The performance ride on day seven is described in detail in Figure 2. This ride consisted of a two-hour block of steady state cycling at 70% $V0_2$ max, during which metabolic measurements were made, followed by a self-paced time trial (time to complete 7 kJ/kg BM), undertaken on a Lode cycle ergometer (Lode Bike, Groningen, The Netherlands). No measurements other than time to complete each 10% of the set work were collected during the time trial, to minimise interference in the cyclists’ performance. Subjects were kept blind to their performance times until the completion of the entire study. During both studies, the researcher responsible for collection of the time trial data was kept blind to the dietary treatments to prevent observer bias.

Results and discussion

According to questionnaires completed each day, all subjects experienced symptoms of mild headaches, lethargy and increased fatigue during the fat adaptation treatment compared with the high CHO treatment. Although the full training program was completed, all subjects experienced difficulties in at least one training session on this trial, either complaining of increased perception of effort or difficulty in maintaining the training pace. The generalised symptoms appeared to decrease as the week progressed. In study 2, when subjects were blinded to the order of dietary treatment, seven of the eight subjects were able to correctly identify these diets. Decisions were made between the evening of day one and day four, and the most important clue was sense of well-being and exercise tolerance rather than any dietary observations.

Measurements undertaken in study 1 showed that muscle glycogen concentrations declined with five days of training and the high fat, low CHO intake such that day six concentrations following the fat adaptation treatment were lower than in the high CHO trial, and below levels determined on day one. Muscle glycogen concentrations were maintained throughout the training program by the CHO treatment. Regardless of previous dietary treatment, 24 hours of high CHO intake and rest rapidly restored muscle glycogen concentrations above day one values. Accordingly, subjects commenced the performance ride on day seven with similarly elevated muscle glycogen stores on both treatments. We assumed that muscle glycogen concentrations mirrored this pattern in study 2 since identical dietary and training programs were followed.

Rates of fat and CHO oxidation during submaximal cycling are summarised in Figure 3. This figure shows that the five-day adaptation to a high fat diet caused a significant decrease in the rates of CHO oxidation during exercise. Conversely, rates of fat oxidation almost doubled after this treatment. The restoration of muscle glycogen content, after one day of a high CHO diet, only partially reversed these changes. In study 1, fat oxidation in the fat adaptation trial during the first 20 minutes of the steady-state portion of the performance ride was reduced compared to day six rates, but remained elevated above baseline rates, and above the rates in the high CHO trial. Throughout the 120 minutes, rates of fat oxidation continued to rise in both trials, but remained significantly higher at all time points in the fat adaptation trial than in the high CHO trial. Mean values for total fat oxidation over the 120 minutes were calculated as (mean ± sd) 61 ± 5 g and 94 ± 6 g for the high CHO and fat adaptation trials respectively ($P < 0.05$). The CHO oxidation rates mirrored these changes: rates on day seven in the fat adaptation treatment increased above the day six values, but remained below rates seen at baseline or in the corresponding times in the high CHO trial. Throughout the ride, CHO oxidation rates declined in both trials, mirroring the gradual decline in blood glucose concentrations with time. Although blood glucose declined in both trials, concentrations were better preserved in the fat adaptation trial over the last 20 minutes of the steady state ride compared with the high CHO trial. Total CHO oxidation during the 120 minutes was 342 g versus 271 g for high CHO and fat adaptation trials. Tracer-derived estimates of blood glucose utilisation during the steady-state ride showed no differences between trials. Therefore, we estimated that CHO sparing as a result of the fat adaptation treatment was explained almost entirely by muscle glycogen sparing. Direct measurements of glycogen utilisation during the ride confirmed these findings.

In study 2, subjects consumed a high CHO meal before the ride, and drank a carbohydrate-containing sports drink throughout. These strategies increased CHO

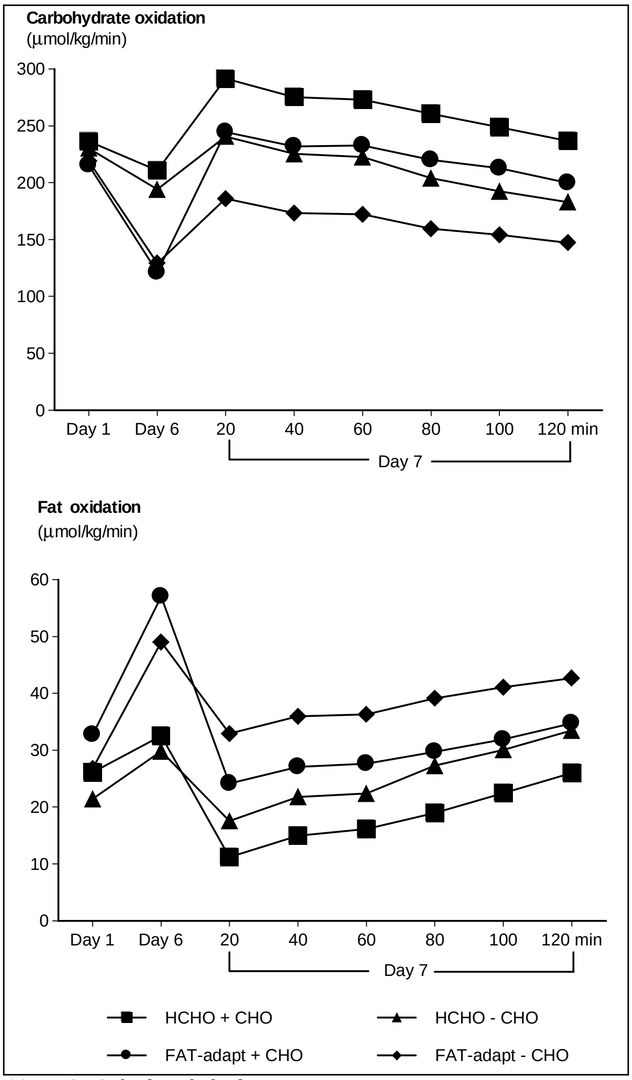
Figure 2. Overview of performance testing day (day 7) in studies[a]

Pre-event nutrition*	120 min cycling @ 70% $V0_2$max					Time trial (7 kJ/kg)	
Time (minutes)	0	20	40	60	80	100	120
Study 1 only Muscle biopsy	⇓						⇓
RER data Blood samples Fluid intake*		⇓	⇓	⇓	⇓	⇓	⇓

* Study 1: overnight fast, water intake during trial. Stabilisation of 6,6^{2}H-glucose infusion.
Study 2: pre-event CHO breakfast (2 g/kg CHO) and sports drink (0.8 g/kg/h CHO) during trial.

(a) RER, respiratory exchange ratio; CHO, carbohydrate.

Figure 3. Estimates of carbohydrate (CHO) oxidation and fat oxidation at 70% VO_2 max at baseline (day 1) and following five days of fat adaptation (FAT-adapt) (day 6) and glycogen restoration (day 7). In study 1 no additional CHO was consumed during the trial. In study 2 CHO was consumed before and during the trial[a]



(a) HCHO, high carbohydrate.

oxidation rates in both trials. However, the differences between the fat adaptation trial and the high CHO trial remained. Again, CHO sparing was seen, estimated at approximately 66 g over the 120 minutes. Figure 3 shows a clear hierarchy in rates of both CHO and fat oxidation between trials, and between the treatments in each study. This provides clear evidence that at least some of the adaptations caused by a five-day exposure to the high fat diet persisted and were independent of the availability of CHO substrates.

Performance outcomes, measured as the time to complete a time trial are summarised in Table 1. We did not find evidence of improved cycling performance in either study, despite observing marked changes in fuel utilisation during the steady state ride preceding the time trial. In study 1, time trial performances were (mean ± SEM) 30.73 ± 1.12 minutes versus 34.17 ± 2.62 minutes for fat adaptation and high CHO respectively ($P = 0.21$). While these differences are not significant, mean time trial time was 8% faster with the fat adaptation treatment compared to the high CHO trial [95% confidence interval (CI), –6 to 21%], while the difference in mean power output during the time trial was 21W [95% CI, –18 to 60 W]. However, these differences are largely explained by two individuals who performed substantially better following the fat adaptation treatment. More correctly, these subjects performed badly on the control (high CHO) trial due to the onset of severe symptoms of fatigue at the end of the steady-state ride and during the time trial, in conjunction with lower blood glucose concentrations. It appears that fat adaptation may be of benefit to individuals who are at risk of developing symptomatic hypoglycaemia during prolonged exercise when deprived of CHO, in so far that it may allow better maintenance of blood glucose concentration. However, these subjects also will benefit from strategies to consume CHO during prolonged exercise; practices that are commonly recommended and easier to achieve than five days of extreme dietary change. Indeed, in study 2, subjects performed almost identical times for the time trial in both trials (mean ± sem, 25.45 ± 0.96 minutes versus 25.53 ± 0.67 minutes, $P = 0.86$). Overall, despite higher muscle glycogen stores and an enhanced capacity for fat utilisation at the onset of the time trial, subjects did not show a clear performance benefit following the fat adaptation treatment in either study.

It is not clear whether the metabolic changes observed following fat adaptation strategies in our studies involve up-regulation of fat oxidation during exercise, a down-regulation of CHO oxidation or a combination of both. Other human studies have reported changes in the activity of enzymes such as beta-hydroxyacyl- CoA-dehydrogenase (8), carnitine palmitoyl transferase-I (7) and pyruvate dehydrogenase (15) following several days or several weeks of high fat, low CHO intake. Whether these or other changes occur over the period of fat exposure used in our study, or in highly trained athletes who continue to undertake extensive training programs, needs to be investigated. Other studies have found that exposure of trained subjects to one to 28 days of a high fat intake increases muscle triglyceride stores (4,16). It is possible that this might provide an additional substrate pool to account for some or all of the additional fat utilised after the fat adaptation treatment. Nevertheless, the precise mechanism to explain the preferential use of this or other fat substrates in the face of plentiful glycogen stores remains to be elucidated.

We were interested in the effect of these metabolic adaptations on the performance of endurance cyclists, since fat adaptation has recently been promoted in some sporting circles as an ergogenic strategy for competition preparation. Previous studies of long-term fat adaptation have reported conflicting results with respect to performance benefits. When undertaken by trained subjects, fat adaptation has been reported both to enhance (5,10) or to fail to alter (6,11) exercise capacity. Alternatively when sedentary subjects are exposed to high fat diets while commencing a training program, gains in endurance are either impaired (13) or unchanged (12) compared with a control group consuming a high CHO diet. The literature is difficult to review because of the unorthodox design of some studies (5,10), small subject numbers (5,6) and lack of relevance of performance measurements to outcomes in competitive sport (5,6). We chose a fixed work time trial as the outcome measure of this study because of its application to sport and its documented reliability even when undertaken after a steady-state exercise pre-load (coefficient of variation = approximately 3.5%) (17). In the initial study, cyclists performed after an overnight fast and consumed water during their prolonged exercise bout. While this is not representative of the real life nutritional practices recommended for endurance athletes, the withholding of CHO intake immediately before and during the performance ride provided baseline metabolic conditions. In the second study, metabolism and performance were compared following the recommended strategies of a pre-event meal and CHO intake during the

Table 1. **Effects of five days of fat adaptation (FAT-adapt) and glycogen restoration on performance of time trial (7 kJ/kg body mass) after 120 minutes of cycling**

	Performance on fat adaptation (minutes)	*Performance on high carbohydrate (minutes)*
Study 1		
Individual subjects	26.75	25.57
	32.68	32.98
	27.93	32.12
	30.18	28.88
	35.03	37.65
	33.00	34.73
	29.57	47.18
Mean ± SEM	30.73 ± 1.12	34.17 ± 2.62
Study 2		
Individual subjects	26.4	26.9
	24.6	22.5
	23.8	24.7
	24.9	22.6
	28.4	29.7
	22.6	22.9
	26.2	26.4
	27.4	27.9
Mean ± SEM	25.52 ± 0.67	25.45 ± 0.96

ride. We found no evidence of clear performance advantages in either study.

In summary, five days of exposure to a high fat, low CHO diet caused substantial changes in fuel substrate utilisation during submaximal exercise. At least some of these changes were independent of CHO availability, since enhanced capacity for fat oxidation persisted despite restoration of muscle glycogen stores. Despite promoting glycogen sparing during prolonged exercise, fat adaptation strategies did not provide a clear benefit to the subsequent performance of a time trial lasting approximately 25 to 30 minutes. The results of these studies do not support the practice of fat adaptation strategies by endurance athletes competing in events of two to three hours duration. The only remaining question is whether there are any advantages for ultra-endurance athletes who compete in events undertaken at a lower intensity and for longer periods (e.g. four hours or more). For these athletes, fat is the predominant fuel source.

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References

1. Bergstrom JL, Hermansen E, Hultman E, Saltin B. Diet, muscle glycogen and physical performance. Acta Physiol Scand 1967;71:140–50.

2. Christensen EH, Hansen O. Zur methodik der respiratorischen quotienbestimmung in ruhe and bei arbeit. III: Arbeirsfahigkeit und ernahrung. Scand Arch Physiol 1939;81:160–71.

3. Galbo H, Holst JJ, Christensen NJ. The effect of different diets and of insulin on the hormonal response to prolonged exercise. Acta Physiol Scand 1979;107:19–32.

4. Starling RD, Trappe TA, Parcell AC, Kerr CG, Fink WJ, Costill DL. Effect of diet on muscle glycogen and endurance performance. J Appl Physiol 1997;82:1185–9.

5. Lambert EV, Speechly DP, Dennis SC, Noakes TD. Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet. Eur J Appl Physiol 1994;69:287–93.

6. Phinney SD, Bistrian BR, Evans WJ, Gervino E, Blackburn GL. The human metabolic response to chronic ketosis without caloric restriction: preservation of submaximal exercise capacity with reduced carbohydrate oxidation. Metabolism 1983;32:769–76.

7. Hargreaves M, McConell G, Proietto J. Influence of muscle glycogen on glycogenolysis and glucose uptake during exercise in humans. J Appl Physiol 1995;78:288–92.

8. Fisher EC, Evans WJ, Phinney SD, Blackburn GL, Bistrian BR, Young VR. Changes in skeletal muscle metabolism induced by a eucaloric ketogenic diet. In: Knuttgen HG, Vogel JA, Poortmans J, editors. Biochemistry of exercise. Champaign, Illinois: Human Kinetics; 1983. p. 497–501.

9. Helge JW, Kiens B. Muscle enzyme activity in humans: role of substrate availability and training. Am J Physiol 1997;272:R1620–4.

10. Muoio DM, Leddy JJ, Horvath PJ, Pendergast DR. Effect of dietary fat on metabolic adjustments to maximal VO_2 and endurance in runners. Med Sci Sports Exerc 1994;25:81–8.

11. Goedecke JH, Christie C, Wilson G, Dennis SC, Noakes TD, Hopkins W, et al. Metabolic adaptations to a high-fat diet in endurance cyclists. Metabolism 1999;48:1509–17.

12. Helge JW, Wulff B, Kiens B. Impact of a fat-rich diet on endurance in man: role of the dietary period. Med Sci Sports Exerc 1998;30:456–61.

13. Helge JW, Richter EA, Kiens B. Interaction of training and diet on metabolism and endurance during exercise in man. J Physiol (Lond) 1996;492:293–306.

14. Burke LM, Angus DJ, Cox GR, Cummings NK, Febbraio MA, Gawthorn K, et al. Effect of fat adaptation and carbohydrate restoration on metabolism and performance during prolonged cycling. J Appl Physiol (in press).

15. Putman CT, Spriet LL, Hultman E, Lindinger MI, Lands LC, McKelvie RS, et al. Pyruvate dehydrogenase activity and acetyl group accumulation during exercise after different diets. Am J Physiol 1993;265:E752–60.

16. Janssen E, Kaijser L. Effect of diet on the utilization of blood-borne and intramuscular substrates during exercise in man. Acta Physiol Scand 1982;115:19–20.

17. Jeukendrup A, Saris WHM, Brouns F, Kester ADM. A new validated endurance performance test. Med Sci Sports Exerc 1996;28:266–70.