Changes in blood glucose levels during a 1005-km running race: a case study

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The blood glucose response of a male ultramarathon runner was monitored throughout a 1005-km race. Before the race the runner had a fasting blood glucose concentration of 5.1 mM. At no stage during the race were his mean blood glucose levels less than 5.8 mM. This was partially attributed to the eating patterns of the athlete, the times at which blood samples were taken, the glycaemic index of food ingested and hyperglycaemia. While there was no evidence of glucosuria, ketones were present in the urine on one day of the event. There were other signs suggesting that at various stages of the event the runner had a metabolic acidosis. Possible reasons for this are discussed.

Keywords: Ultramarathon, glucose, hypoglycaemia, hyperglycaemia, ketones

Competing in the annual Sydney to Melbourne (Australia) running race (1005 km in 8.5 days) presents several metabolic challenges – there is a need to ingest a large amount of food as endogenous substrates may be insufficient to complete the event; there is also a need to maintain appropriate blood glucose levels. Central to both these metabolic challenges is the consumption of a diet rich in carbohydrate during the race[1, 2].

Not all individuals appear to benefit from carbohydrate ingested while exercising[3]. Coyle et al.[4] suggested that an athlete's ability to utilize carbohydrate ingested during endurance exercise may be dependent on the oxidative potential of the skeletal muscle tissue (i.e. level of capillarization and oxidative enzyme adaptation). Only when such potential is well developed will carbohydrate ingested during exercise be utilized. To our knowledge, no skeletal muscle analyses have been completed on ultraendurance athletes; however, the training practices of these athletes are consistent with the enhancement of oxidative enzyme activity and muscle capillarization[5]. Thus, one would expect that ultraendurance athletes would be able to metabolize carbohydrate consumed during exercise.

An appropriate carbohydrate intake can produce elevations in both blood glucose and insulin levels which may enhance blood glucose utilization and

spare endogenous substrates, particularly lipid[1, 6, 7]. Reductions in blood glucose levels have been associated with fatigue[4, 8] and hypoglycaemia[3]. Rontoyannis et al.[9] estimated that the daily running pace of the winner in the 1985 Sydney–Melbourne race corresponded with a mean(s.d.) $\dot{V}_{O_2 max}$ of 42.0(9.8) (range 30–57)%. An oxygen consumption of this magnitude represents a low work intensity[1]. Gluconeogenesis has been shown to be sufficient to prevent hypoglycaemia during low intensity work (40% $\dot{V}_{O_2 max}$) lasting 4 h when there was no food ingested[6, 10]. In addition, blood glucose levels were enhanced and endogenous lipid substrate spared by the consumption of carbohydrate during low intensity exercise[6, 7]. Consequently, when carbohydrate is ingested, hypoglycaemia is unlikely during several hours of low intensity exercise. However, it is not yet known if this continues to be the case during low intensity exercise which is maintained over several days. In addition, Coyle and Coggan[1] suggest that when blood glucose levels are elevated in the absence of increments in insulin levels, endogenous substrate may not be spared. This scenario may increase the possibility of hyperglycaemia. Sleep deprivation, which is common in ultraendurance events[9], may produce a relative insulin insufficiency[11]. This, in combination with the regular ingestion of carbohydrate by an ultraendurance athlete, may produce elevations in blood glucose in the absence of hyperinsulinaemia. The purpose of this case study was to determine the blood glucose response of a runner consuming food rich in carbohydrate during the 1005-km Sydney–Melbourne race.

Case study

A 38-year-old male novice runner (weight 55.5 kg; 5.6% body fat; height 171.5 cm) with a $\dot{V}_{O_2 max}$ of 70.6 ml kg^{-1} min^{-1} was monitored throughout the 1990 Sydney–Melbourne race. He had an extensive preparation for the event, with training loads for the 6 months preceding the event averaging 240–320 km per week. Blood glucose levels were determined 3 days before and approximately every 8 h during the event (Glucometer, Ames, Australia). Capillary blood was drawn from the finger for all analyses. Blood was taken for analysis before large meals; however, the athlete was drinking approximately 600 ml of a glucose polymer (Maximum, Bio-Organics, Australia; 70 g carbohydrate l^{-1}) every hour during exercise. Urine samples were analysed to determine glucose,

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ketone and pH levels at 12-hourly intervals during the event (Multistix Reagent Strips, Ames, Australia). The amount of sleep was monitored. While the dietary regimen for the run was preplanned, macronutrient dietary analysis was conducted on the actual intake at the completion of the event.

The runner's fasting blood glucose level 3 days before the event was 5.1 mM. The athlete completed the event in 199 h at a mean(s.d.) speed of 6.04(0.85) (range 4.60–7.63) km h^{-1}. The oxygen consumption associated with the mean running pace was estimated to be 34% $\dot{V}O_{2max}$. Over the course of the event the runner consumed 220.2 MJ of which 65% was obtained from carbohydrate, 26% from fat and 9% from protein.

Results presented in Table 1 indicate that at no stage during the event was the runner hypoglycaemic (frank hypoglycaemia <2.5 mM[1]). Mean blood glucose levels at all times exceeded the normal range associated with fasting (3.6–5.9 mM). This was not surprising, as previous investigations have demonstrated that the consumption of carbohydrate during low and moderate intensity exercise increased blood glucose levels[4,6,7]. However, some of the increase in blood glucose levels was attributed to the imprecise relationship between the times at which samples were taken and food ingested. It was virtually impossible to avoid such imprecision as the athlete was eating continually throughout the event. Ahlborg and Felig[6] reported that 50 min after the ingestion of 200 g glucose during exercise (30% $\dot{V}O_{2max}$), blood glucose levels increased from 4.5 to 6.3 mM. On the fourth, sixth and eighth days of the event solid foods were ingested less than 30 min before the measurement of blood glucose levels. The ingestion of these foods probably distorted the blood glucose values. Mean blood glucose scores were corrected for this sampling error by removing the distorting values from the analysis (Table 1). These adjusted values may more accurately portray the average blood glucose concentration on any given day, and indicate that blood glucose was maintained throughout the 199 h of the event at levels greater than those associated with fatigue in previous research in which subjects received carbohydrate supplementation[4,7]. In addition, some of the diurnal variation in blood glucose levels was attributed to the

different glycaemic indices of foods eaten. Differences in the glycaemic index of foods ingested during the event was illustrated by blood glucose levels of 15.9 and 18.4 mM shortly after the ingestion of lentil soup, boiled potatoes and a commercial hamburger, respectively.

On day 5 of the event, a small level of ketonuria was detected (Table 1). This may have been due to lipid and/or protein metabolism; however, amino acid degradation appeared to be the less likely explanation, as the protein degradation throughout the race was estimated to be between 78.0 and 88.0 g day^{-1}, with protein degradation on day 5 estimated to be 78.0 g (estimates based on data presented by Viru[12]). Consequently, had protein degradation been the source of ketone bodies, one would have expected ketonuria to be evident on additional occasions. As ketonuria was transient, it was thought that hyperglycaemia was a more likely explanation.

Despite ketonuria being attributed to hyperglycaemia, there was no evidence of glucosuria (Table 1). Interestingly, ketonuria did not coincide with the highest blood glucose levels. This could be explained in three ways: first, ketone body formation was not abnormal on the days that blood glucose levels were highest; second, the threshold for ketone body excretion was not attained on these occasions; and third, ketonuria only occurred when blood glucose values were elevated for the preceding 24 h (see unadjusted values Table 1). This was the case on day 5.

Excessive ketogenesis can result in metabolic acidosis, which in turn can increase the rate of ventilation[13]. The runner's support crew reported that the ventilation rate of the athlete was disproportionately high for his pace on several days. This may have been indicative of respiratory compensation for a metabolic acidosis caused by a relative insulin insufficiency. Sleep deprivation may have provided the trigger for such an insulin insufficiency[11] because the runner had only 31.5 h sleep during the event.

The results of this case study indicated that a trained ultramarathoner running at 34% $\dot{V}O_{2max}$ for 8 days did not become hypoglycaemic when ingesting food rich in carbohydrate. Mean blood glucose levels were at all times in excess of 5.8 mM (adjusted values). The elevated blood glucose levels during the

Table 1. A daily summary of the blood glucose, urinary glucose, urinary ketone body and urinary pH values obtained over the course of the Sydney to Melbourne ultramarathon. Unadjusted blood glucose values were on occasion distorted by meals ingested less than 30 minutes before sampling. The adjusted mean blood glucose scores were determined after these distorting values were removed from the analysis

Day	Unadjusted blood glucose (mM)	Adjusted blood glucose (mM)	Urinary glucose (mM)	Urinary pH	Urinary ketones (mM)
1	6.0 (2.3)	6.0	0.0*	7.5*	0.0*
2	7.0 (1.4)	7.0	0.0*	6.5*	0.0*
3	6.2 (2.5)	6.2	0.0*	6.5*	0.0*
4	11.3 (5.3)	7.9	0.0*	6.8 (1.1)	0.0*
5	6.5 (1.0)	6.5	0.0*	5.5 (0.7)	0.8 (1.1)
6	6.6 (1.7)	5.8	0.0*	5.5 (0.7)	0.0*
7	7.5 (4.4)	7.5	0.0*	6.3 (0.4)	0.0*
8	12.9 (7.8)	7.3	0.0*	6.0*	0.0*
9	7.4	7.4	0.0*	6.0*	0.0*

Values are mean(s.d.); *s.d. = 0.0

event were partially attributed to: the athlete eating continually throughout the event; the times at which samples were taken; and the glycaemic index of food. There was also some evidence to suggest that a fourth variable, hyperglycaemia, may have influenced blood glucose levels. The relative importance of each of these four variables in determining the blood glucose scores at various stages in the event could not be determined.

Acknowledgements

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