BIOCHEMICAL CHANGES IN ATHLETES DURING MARATHON AND ULTRA-MARATHON RACES, WITH SPECIAL REFERENCE TO THE INCIDENCE AND PREVENTION OF HYPOGLYCAEMIA

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INDEX

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Finally, as a dedication to my mother, who always encouraged learning and always made it possible, I present this thesis.

Penelope S. McArthur
August 1984

DECLARATION

I, Penelope McArthur, declare that the work on which this thesis is based is original and that neither the whole work nor any part of it has been, or is to be submitted for another degree in this or any other University.

I empower the University to re-produce for the purpose of research either the whole or any portion of the content of this thesis, in any manner whatsoever.

Penelope McArthur

Cape Town

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ABSTRACT

Fats and carbohydrates are the major fuels utilized during exercise and it has been suggested that carbohydrate depletion is the cause of exhaustion during prolonged exercise lasting more than two hours. However, there is some disagreement in the literature as to whether this exhaustion is due either to muscle glycogen depletion or to hypoglycaemia secondary to liver glycogen depletion.

I therefore undertook three studies to determine the roles of hypoglycaemia in explaining fatigue in marathon and ultra-marathon runners.

In the first study, blood samples were drawn from 256 competitors at the end of three premier South African races of 42-, 56- and 88-km. The blood was analysed for its glucose, free fatty acid, insulin, glucagon, glycerol, alanine and cortisol concentrations.

In the second study, respiratory gas samples were collected from a group of 8 runners during the same 42- and 56-km marathons the following year, and analysed for O_2 and CO_2 content for the calculation of Respiratory Quotient. Muscle biopsies performed on the same runners at the end of the 42- and 56-km marathons and at the end of a 91-km marathon were

analysed for their glycogen content.

In the final study, three groups of 6 equally-matched runners (n = 18) were studied before, during and for 3 days after a 42-km standard marathon. During the race each group ingested different carbohydrate-containing drinks; one group drank a 2% glucose and water solution, the second group drank an 8% fructose and water solution and the third group drank an 8% glucose polymer and water solution. Blood samples were drawn before and at 10-, 21-, 32- and 42-km and muscle biopsies were performed before, immediately after and again 2 days after the race. The blood samples were analysed for glucose, free fatty acid and insulin levels, and the muscle biopsy samples for glycogen content.

The principal finding of the first study was that the majority of samples drawn after marathon races of 42-, 56- and 88-km, showed hyperglycaemia (blood glucose levels >5 mmoles ℓ^{-1}) but blood glucose levels fell with increasing race distance. 12% of runners had blood glucose levels below 4 mmoles ℓ^{-1} after the 88-km race as well as complaining of hypoglycaemic symptoms. The lowest blood glucose level measured was 1,9 mmoles ℓ^{-1}. Serum free fatty acid levels rose with increasing race distance. Running ability did not affect the post-race blood glucose, free fatty acid or insulin concentrations in the 88-km race but in the 42- and 56-km

races, the fastest runner had higher blood glucose and insulin and lower free fatty acid levels compatible with increased rates of carbohydrate utilization.

In the second study it was found that despite higher Respiratory Quotients during the race, indicating increased carbohydrate utilization, the better runners completed the races with higher muscle glycogen levels suggesting that they were less carbohydrate depleted.

The final study showed that the ingestion of the carbohydrate solutions during a standard 42-km marathon had no significant effect on the blood glucose, free fatty acid or insulin levels or on the rate of muscle glycogen utilization. In addition, blood glucose levels increased steeply for the first 10-km of the race but then fell slightly and were lowest 10-km from the finish. Blood glucose levels measured after the runners had stopped running at the end of the race were elevated, as was also found in the first study.

These studies allow the following conclusions :

(1) Blood glucose levels rise steeply at the cessation of marathon races so that blood glucose levels measured after the runners have finished running at the end of the race may be 1 - 2 mmoles ℓ^{-1} higher than the actual

values present during the race. Thus, blood samples taken at the end of a race after the athletes have stopped running, may underestimate the true incidence of hypoglycaemia during prolonged exercise.

(2) Carbohydrate ingestion by carbohydrate-loaded runners during a 42-km standard marathon race does not influence blood glucose levels or alter the rate of muscle glycogen depletion. Thus it would appear that adequate carbohydrate-loading before the standard marathon race is more important in the prevention of hypoglycaemia than is carbohydrate ingestion during competition.

(3) Hypoglycaemia could not explain the fatigue experienced by all runners in the 42-km marathon. It seems likely that the near total depletion of muscle glycogen stores at the end of the race, was the more likely cause.

(4) All athletes, regardless of their level of fitness or the speed at which they ran the 42-km marathon race, were equally carbohydrate-depleted at the finish. The faster runners did however have higher pre-race muscle glycogen levels and tended to have more rapid glycogen resynthesis after the race.

(5) Theoretical considerations do however suggest that

hypoglycaemia becomes increasingly more likely to be a limiting factor in exercise lasting more than 4 hours. Only carbohydrate-containing solutions that deliver more than 40g carbohydrate hr^{-1} to the intestine are likely to prevent hypoglycaemia during such exercise.

(6) The response of serum insulin and free fatty acid levels to exercise was not influenced by the type of carbohydrate ingested.

LIST OF TABLES

LIST OF FIGURES

Page No.

ABBREVIATIONS

$\dot{V}O_2$ max	=	maximum oxygen consumption
km	=	kilometers
mmoles ℓ^{-1}	=	millimoles per litre
g	=	gram/s
hr	=	hour/s
g kg^{-1} hr^{-1}	=	gram/s per kilogram per hour
mmoles kg^{-1}	=	millimoles per kilogram
mins	=	minutes
g hr^{-1}	=	gram/s per hour

CHAPTER 1

OUTLINE AND RATIONALE OF STUDY

The major fuels utilized during prolonged endurance exercise at 60 - 85% of maximum oxygen consumption ($\dot{V}O_2$ max) are carbohydrates (Bergström and Hultman, 1967; Christensen and Hansen, 1939a; Essén, 1977) and fats (Pernow et al, 1971; Rodahl et al, 1964). There is good evidence that exhaustion during this form of exercise may be due to carbohydrate depletion (Ahlborg, 1967a; Bergström and Hultman, 1967; Christensen and Hansen, 1939b; Dill et al, 1933; Hermansen et al, 1967; Levine et al, 1924; Saltin et al, 1967). Most workers feel that it is depletion of muscle carbohydrate stores which explains this exhaustion (Bergström et al, 1967; Gordon et al, 1925), but the possibility exists that liver glycogen depletion, leading to hypoglycaemia, could complicate this type of fatigue. The evidence for this includes the following :

First, Dill et al (1933) found that the trained terrier, Joe, was exhausted and hypoglycaemic after six and a half hours of treadmill running (blood glucose value approximately 3,7 mmoles ℓ^{-1}). However, when supplied with 30g of glucose every 60 minutes during exercise, Joe ran for 13 hours and was not exhausted. During this time his blood glucose rose steadily reaching hyperglycaemic levels at the end of exercise (blood glucose value approximately 8,2 mmoles ℓ^{-1}.)

Second, Levine et al (1924) observed that athletes, exhausted

after running the 1923 Boston marathon, showed symptoms that these authors considered to be identical to progressive insulin shock. Six of the eleven runners had blood glucose levels of 3,6 mmoles ℓ^{-1} and below, and there was a correlation between their blood glucose levels and their state of exhaustion. The athlete with the lowest blood glucose level was brought unconscious to the finish of the race by the police.

Third, studies by Wahren (1977) and Wahren et al (1971; 1973) have shown that during prolonged exercise lasting more than 180 minutes, liver glucose release falls below muscle glucose uptake. Thus, blood glucose levels must fall (Felig and Wahren, 1975; Wahren et al, 1971; Ahlborg and Felig, 1982).

Finally, some empirical observations have been made. In the 1979 90-km Comrades marathon, the athlete who was leading the field in record time was incapacitated 14-kms from the finish by a condition, the symptoms for which were indistinguishable from hypoglycaemia. On drinking a litre of Coca Cola^{R}, containing between 18 and 36g of sucrose, he made a dramatic recovery and went on to finish the race in second place, narrowly missing first place. This recovery resembles that of exhausted, hypoglycaemic subjects recorded by Boje (1936) and Christensen and Hansen (1936b), who were again able to

continue exercising after glucose ingestion.

The following year (1980) the athlete who had won the 1966 Comrades marathon, collapsed just short of the finish line. His blood glucose level, recorded with a dextrostix (Ames Company) showed a value below 2,5 mmoles ℓ^{-1}. He recovered rapidly when given an intravenous glucose infusion.

In order, therefore, to determine whether hypoglycaemia might occur more frequently than is recognised, we initally chose to measure a number of blood constituents, including blood glucose levels, in marathon runners competing in the premier South African long distance races. Thereafter, we studied blood glucose levels during and immediately after a 42-km standard marathon race and the effects of different carbohydrate drinks on those levels and on the rates of muscle glycogen utilization.

Parts of this thesis have already been published:

(1) McArthur, P.S.; Noakes, T.D.; Gevers, W.; Millar, R. Studies of the metabolic basis of fatigue during marathon and ultra-marathon races. S.A. Journal for Research in Sport, Physical Education and Recreation. 6(1):49 - 57, (1983)

(2) Noakes, T.D.; McArthur, P.S.; Koeslag, J.H. Hypoglycaemia during exercise (letter). New Eng. J. Med. 308:279 - 280, (1983)

In addition, the author assisted in a parallel study, the results of which have also been published.

(3) Noakes, T.D.; Kotzenberg, G.; McArthur, P.S.; Dykman, J. Elevated creatine kinase MB and creatine kinase BB - Isoenzyme fractions after ultra-marathon running. Eur. J. Appl. Physiol. 52:75 - 79, (1983)

CHAPTER 2

LITERATURE REVIEW

INTRODUCTION

There is good evidence that an important factor limiting performance during endurance exercise, is the depletion of the carbohydrate stores in either the active skeletal muscles (Ahlborg et al, 1976b; Bergström et al, 1967; Hermansen et al, 1967; Hultman, 1967; Costill et al, 1981; Sherman et al, 1983) or in the liver, or both (Ahlborg et al, 1974). Whilst the importance of hypoglycaemia as a cause of fatigue during prolonged exercise like marathon running, was recognised by the early workers (Best et al, 1930; Levine et al, 1924; Dill et al, 1933; Gordon et al, 1925; Schenk, 1925; Schenk and Craemer, 1930; Boje, 1936), more recently the emphasis has been on muscle glycogen depletion as the more important cause of this fatigue (Bergström et al, 1967; Hermansen et al, 1967).

This literature review looks particularly at the effect of exercise on muscle glycogen levels and on the blood levels of glucose, free fatty acids and other metabolic/hormonal constituents. Special attention has been paid to those studies showing that either muscle, or liver glycogen depletion is responsible for exhaustion during prolonged exercise.

2.1 MUSCLE GLYCOGEN AND EXERCISE

(i) Muscle Glycogen Utilization During Exercise

The muscle glycogen content of the exercising muscles decreases during prolonged exercise and its rate of utilization is related to work intensity expressed as a percentage of maximum oxygen consumption - $\dot{V}O_2$ max (Ahlborg et al, 1967a; Bergstöm and Hultman, 1967; Fröberg and Mossfeldt, 1971; Hermansen et al, 1967; Terblanche and Oelofsen, 1981). Muscle glycogen utilization is greatest during the first few minutes of exercise and thereafter the rate of utilization falls (Hermansen et al, 1967). At 28% $\dot{V}O_2$ max, Hermansen et al (1967) found that the rate of muscle glycogen utilization was 3g kg^{-1} wet muscle hr^{-1}, whereas at 78% $\dot{V}O_2$ max the rate was found to be 15,6g kg^{-1} hr^{-1}. Essén's (1977) results were similar, showing that at 75 - 85% $\dot{V}O_2$ max muscle glycogen utilization was approximately 9g kg^{-1} hr^{-1}.

Other factors that influence the rate of glycogen utilization include the duration of exercise (Saltin and Karlsson, 1971; Essén, 1977), the mode of exercise - for example, more glycogen is used in uphill running than during running on the level at the same % $\dot{V}O_2$ max (Essén, 1977), and the muscle group involved in the

exercise. Treadmill running on the flat results in greater depletion in the gastrocnemius muscles whereas uphill running results in three times greater depletion in the vastus lateralis muscles (Essén, 1977).

(ii) The Importance of Body Carbohydrate Stores for Endurance Performance

A. Carbohydrate is required to maintain a high work intensity

Hermansen et al (1967) and Saltin and Hermansen (1967) found that carbohydrate-depleted muscles were unable to maintain a high work output. Pernow and Saltin (1971) showed that when muscle glycogen stores are depleted by exhausting exercise which was followed by a one day carbohydrate-free diet, prolonged exercise could only be performed at a lower work intensity (approximately 25% less) and work time was reduced. When the supply of exogenous free fatty acids was reduced by the administration of nicotinic acid (which blocks the release of free fatty acids from adipose tissue), the capacity for prolonged exercise was reduced by a further 50%. Thus, with lowered glycogen stores, endurance exercise can only be performed if work intensity is reduced to less than 60 - 70% $\dot{V}O_2$ max and there is an adequate supply of free fatty acids.

B. Exhaustion occurs simultaneously with carbohydrate depletion

Bergström et al (1967) found that glycogen depletion in the exercising muscles, and not hypoglycaemia, is the major explanation for fatigue experienced during prolonged exercise. In their subjects exercising at 75% $\dot{V}O_2$ max to exhaustion on a bicycle ergometer after ingestion of three different diets (carbohydrate-rich, mixed and high protein), symptoms of hypoglycaemia were absent whereas muscle glycogen stores were 80 - 90% depleted. Post-race blood glucose values ranged between 3,5 ± 0,1 mmoles ℓ^{-1} in the group on a high carbohydrate diet and 2,8 ± 0,6 mmoles ℓ^{-1} in the group on a high protein diet.

Hermansen et al (1967) found that subjects working to complete exhaustion on a bicycle ergometer at work loads averaging 77% $\dot{V}O_2$ max, showed 92 - 96% depletion of muscle glycogen stores, but blood glucose levels remained in the normal range. Thus, the severe degree of muscle glycogen depletion was the likely explanation for fatigue. They postulated that the extent of the initial glycogen store in the exercising muscles may be the decisive factor determining the individual's capacity for prolonged, strenuous exercise.

C. Increased pre-exercise muscle glycogen stores result in increased performance

Many researchers have shown that the pre-exercise glycogen stores in exercising muscles appears to be the decisive factor determining the ability to perform prolonged exercise at or below 75% $\dot{V}O_2$ max (Ahlborg et al, 1967a; Astrand, 1967; Bergström et al, 1967; Costill and Winrow, 1970; Pernow and Saltin, 1971; Saltin and Hermansen, 1967).

For example, Bergström et al (1967) found a linear correlation between performance time and the initial, pre-exercise glycogen content in the exercised muscle. Muscle glycogen content after a fat and protein diet was 30 mmoles kg^{-1} and after a carbohydrate-rich diet, 235 mmoles kg^{-1}. Exercise time after the two diets (working on a bicycle ergometer to exhaustion at 75% $\dot{V}O_2$ max) was 59- and 189- minutes respectively. Karlsson and Saltin (1971) also studied the influence of a high initial muscle glycogen content on performance during prolonged exercise, a 30-km running race, and found that the subjects with the highest initial glycogen content performed best (Karlsson and Saltin, 1971; Saltin and Hermansen, 1967).

(iii) Manipulation of Muscle Glycogen Stores by Diet and Exercise

A. Glycogen Loading

Muscle glycogen stores can be altered according to the carbohydrate content of the diet and the duration and intensity of the preceeding exercise (Astrand, 1967; Bergström et al, 1967; Costill, 1974; Pruett, 1970), and can be markedly increased by a technique known as "carbohydrate loading" or "glycogen supercompensation".

In untrained subjects, the most profound effect is obtained by first emptying the glycogen content of the muscles by heavy prolonged exercise and then maintaining low muscle glycogen levels by eating a low carbohydrate diet for up to three days while continuing moderate training. This is then followed by three days of a high carbohydrate diet while exercise intensity is decreased (Ahlborg et al, 1967b; Astrand, 1967; Bergström et al, 1967; McArdle et al, 1981).

That this glycogen supercompensation after exercise-induced muscle glycogen depletion is confined to the exercised muscles has been shown by one legged exercise studies (Hultman, 1967b). Rapid glycogen resynthesis to supra-normal levels occurred only in the exercised

leg when one legged exercise was followed by a high carbohydrate diet (Hultman, 1967b). The glycogen concentration in the non-exercised leg did not alter significantly.

B. Effects of different carbohydrate diets and the carbohydrate depletion phase

MacDougall et al (1977) found that glycogen repletion following exercise-induced depletion took only 24 hours when a carbohydrate enriched diet was consumed. Glycogen levels reached 53% of pre-exercise levels 5-hours post exercise, 67% by 12-hours post exercise and 102% by 24-hours. In contrast, Piehl (1974) found that it took 46-hours to refill glycogen stores when a 60% carbohydrate diet was eaten, glycogen levels reached 68% within 10-hours after exercise.

Ahlborg et al (1967b) found that muscle glycogen content continued to increase even seven days post-exercise, if the carbohydrate loading phase followed a depletion phase. Thus, after a mixed diet, followed by exercise to exhaustion and then 7 days of a high carbohydrate diet, muscle glycogen levels peaked at 123 mmoles kg^{-1} wet muscle by the third day and then rose no further. When the first bout of exhaustive exercise occurred after a mixed diet and was followed by a one day

carbohydrate-free diet and another exhaustive bout of exercise, muscle glycogen levels again peaked at about 150 mmoles kg^{-1} wet muscle after the third day with no further increase over the next four days. However, when the initial bout of exercise to exhaustion was followed by a three-day carbohydrate-free diet, with a subsequent exercise bout, muscle glycogen levels continued to rise for 7 days reaching peak levels of 186 mmoles kg^{-1} wet muscle. Thus, maximum muscle glycogen levels were obtained when the loading phase follows an exhausting bout of exercise which is followed by three days of carbohydrate-free diet.

Recently, Costill et al (1981) have shown that the type of carbohydrate ingested influences the extent of carbohydrate loading. They found that complex carbohydrates eaten during the carbohydrate loading phase resulted in higher muscle glycogen levels than did a diet containing simple carbohydrates.

C. Effects of "fitness"

Costill and Miller's review (1980) has suggested that trained subjects do not need to "carbohydrate-load" since the daily training-induced depletion of body carbohydrate stores stimulates the potential for muscle glycogen resynthesis. Such athletes are then

able to store carbohydrates maximally on a high carbohydrate diet alone, without the need for the depletion phase. In a subsequent study by this group (Sherman et al, 1981), six well trained runners were used as subjects and three different diet regimens were compared. Diet "A" constituted three days of a low carbohydrate diet followed by three days of high carbohydrate diet; diet "B" constituted three days of a medium carbohydrate diet followed by three days of a high carbohydrate diet; and diet "C" constituted six days of a mixed carbohydrate-protein diet. During each dietary period, the subjects underwent the same 5-day depletion-taper exercise sequence in which they ran on a treadmill at 75% $\dot{V}O_2$ max with runs being 90-, 40-, 40-, 20-, and 20-mins respectively with rest on the sixth day. On the seventh day they ran a 20,9-km time trial. Muscle biopsies taken on the 4th day showed that diets "B" and "C" produced significantly higher muscle glycogen levels than did diet "A". But pre-exercise biopsies on the 7th day showed significantly higher muscle glycogen levels when diets "A" and "B" were followed. However, the most significant finding was that performance was the same in all three cases indicating that carbohydrate loading was of no benefit to performance for trained runners during a 20,9-km run. In addition, it was found that despite dissimilar muscle glycogen levels at the start of the race,

post-race muscle glycogen levels were similar in all groups. Thus, muscle glycogen was burned at a faster rate during exercise when pre-exercise muscle glycogen levels were elevated by carbohydrate loading.

D. Method by which elevated pre-exercise glycogen levels can enhance performance

Theoretically at least, elevated pre-exercise muscle glycogen levels should aid performance by allowing exercise of the same intensity to be sustained for a longer time before muscle and or liver glycogen levels are depleted causing exhaustion (see earlier). However, after an increase in the muscle glycogen stores, the proportion of carbohydrate used during exercise increases, and the supply and utilization of circulating free fatty acids decreases (Rennie and Johnson, 1974; Sherman et al, 1981). Thus, elevated pre-exercise muscle glycogen stores will not necessarily ensure that such stores last longer during subsequent exercise.

The findings of Sherman et al (1981) suggest that glycogen loading may be of most benefit to less well-trained athletes who may not yet have developed the capacity for optimum carbohydrate storage when on a high carbohydrate diet, without the added stimulus of carbohydrate depletion. Well trained runners may be

less likely to benefit from this procedure, particularly at shorter distance races.

It should, however, be noted that these studies have not considered the effects of the carbohydrate-depletion, carbohydrate-loading diet on liver glycogen storage. If liver glycogen stores are increased by this procedure, then this could be beneficial even to well trained athletes.

2.2 LIVER GLYCOGEN AND EXERCISE

(i) Liver Glycogen Levels During Exercise, Fasting and Carbohydrate Loading

In a normal subject with a 1,5 - 1,8kg liver, liver glycogen stores on a normal mixed diet amount to about 88 - 100g (Hultman, 1978). The size of this store is strongly influenced by diet. After a carbohydrate-rich diet, the liver glycogen stores can be as large as 180g while after a carbohydrate-poor diet, these stores can be as low as 12g. Liver glycogen stores are equally low after 24-hours starvation as they are when subjects are on a carbohydrate-poor diet. Liver glycogen stores can be virtually totally depleted within 48-hours of carbohydrate starvation (Hultman, 1978).

With regard to the rate of liver glycogen resynthesis, Hultman (1978) has found that on a high carbohydrate diet, liver glycogen stores are completely resynthesised within 24-hours. It is of interest that the rate of liver glycogen synthesis at rest was the same whether glucose was taken orally or infused. However, when fructose was infused the synthesis rate of liver glycogen was increased by 300%.

Liver glycogen is utilized during exercise, the rate of utilization being dependent on work time and work intensity. When pre-exercise liver glycogen levels are high, prolonged exercise (such as marathon running) can continue for up to three hours before liver glycogen levels run out. In this situation hepatic glucose release is derived mainly from glycogenolysis (Hultman, 1967; 1978).

(ii) Blood Glucose Levels During Exercise

Hypoglycaemia (the symptoms of which range from nervous irritability and an inability to concentrate, to collapse and even unconsciousness) will occur during exercise if the rate of delivery of glucose to the blood by hepatic glycogenolysis and gluconeogensis is less than the rate of glucose uptake by the active muscles (Ahlborg et al, 1974; Ahlborg and Felig, 1982).

During prolonged exercise the liver may become increasingly unable to match the increased rate of glucose uptake by active muscles (Ahlborg et al, 1974; Costill, 1974; Felig and Wahren, 1975; Ahlborg and Felig, 1982). This was also found during exercise in the dog (Issekutz et al, 1970).

Early researchers of blood glucose levels in marathon runners found definite evidence of hypoglycaemia in exhausted athletes after prolonged exercise. Levine et al (1924) measured low blood glucose levels (2,5 - 3,3 mmoles ℓ^{-1}) in exhausted runners after the 1923 Boston Marathon. In the following year's race, runners who consumed a high carbohydrate diet the day before the race and who ingested glucose during the race finished in good physical condition and their performances were improved (Gordon et al, 1925).

Similarly, Best et al (1930) measured blood glucose levels of 2,9 , 3,1 and 3,4 mmoles ℓ^{-1} in three finishers of a marathon race, all of whom were in poor physical condition and who displayed weakness and incoordination of movement. Others who have reported hypoglycaemia in marathon runners include Schenk (1925) and Schenk and Craemer (1930). Astrand (1967) reported critically low blood glucose levels at the point of

exhaustion in cross country skiers. After these skiers had ingested 200g of glucose, their hypoglycaemia was corrected and they were again able to continue exercising. The ability of hypoglycaemic, exhausted subjects to again be able to continue exercising after glucose ingestion has been reported also by Boje (1936) and Christensen and Hansen (1936b).

More recent studies have however, shown that blood glucose levels after marathon running may be normal (Beckner and Windsor, 1954; Keul et al, 1981; Magazanik et al, 1974; Muir et al, 1970; Scheele et al, 1979; Schmidt et al, 1970; Sutton et al, 1969) or even raised (Jooste et al, 1981; Kavanagh et al, 1974; Magazanik et al, 1974; Maron et al, 1975; Maron et al, 1978; McKechnie et al, 1982; Pugh et al, 1967; Viru and Körge, 1971).

Similarly, in a number of laboratory experiments on subjects exercising on the treadmill or bicycle ergometer, blood glucose levels have been found to decrease (Ahlborg et al, 1974; Christensen and Hansen, 1939a; Keul et al, 1974; Luyckx et al, 1978), remain constant (Hermansen et al, 1967) or rise (Rennie et al, 1974). During short duration (40 minutes) exercise on a bicycle ergometer at different work loads (400, 800 and 1200-kg

meters min^{-1}), the rise in blood glucose levels was related to work load so that the greater the load the greater the hyperglycaemia (Wahren et al, 1971).

It is therefore clear that the response of blood glucose levels to exercise is highly variable and appears to depend on the work intensity, the duration of exercise and also on whether or not glucose was ingested during exercise (see later).

The rate of hepatic glucose release during exercise is affected by the liver glycogen levels, which as already shown (Hultman, 1978), are strongly influenced by the pre-exercise diet. When liver glycogen stores are high, about 90% of all hepatic glucose release is derived from glycogenolysis. However, when liver glycogen levels are low, gluconeogenesis from lactate, glycerol and alanine partly replaces liver glycogenolysis as the source of hepatic glucose release. The maximal rate of liver gluconeogenesis is low however, so that as liver glycogen levels fall progressively, a point will be reached where hepatic gluconeogenesis is unable to make good the shortfall in glucose coming from hepatic glycogenolysis. At that point, hepatic glucose release must fall. If muscle glucose uptake remains constant, blood glucose levels will fall.

(iii) Hepatic Glucose Production and Muscle Glucose Uptake During Exercise

The rate of hepatic glucose production rises with increases in exercise time and is also related to work load (Wahren et al, 1971; Rowell et al, 1965). At work loads of 400- and 800-kg meters min^{-1} hepatic glucose production increased two-fold and three-fold respectively, with a five-fold increase demonstrated at 1200-kg work load (Wahren et al, 1971). Felig and Wahren (1977) and Ahlborg et al (1974) have shown that glucose uptake by muscles rises during exercise. Glucose utilization also increases as exercise continues beyond 40-minutes, peaking at 90 - 180-minutes of exercise and thereafter declining.

Ahlborg and Felig (1982) have shown that liver glucose production peaks after 90-minutes of exercise at 58% $\dot{V}O_2$ max (in bicycle ergometer exercise lasting 3 - 3,5 hours) and equals approximately 36g hr^{-1}. It then falls so that after 180-minutes of exercise, liver glucose output was only 13,5g hr^{-1} compared to a resting value production rate of 10g hr^{-1}. These workers also showed that muscle glucose uptake was greatest after 90-minutes of the same exercise measuring 43g hr^{-1}. Although muscle glucose uptake fell thereafter, at 180-minutes it was 30g hr^{-1}, thereby exceeding the rate of

hepatic glucose release by up to 17g hr^{-1}. As would be predicted from these data, in these experiments blood glucose levels began falling after 90-minutes exercise, and fell precipitously after 120-minutes exercise, reaching levels of 2,56 mmoles ℓ^{-1} after 210-minutes of exercise. for last no, → pg 30

(iv) The Effect of Glucose Ingestion During Exercise

Glucose ingestion during exercise has been reported to increase performance capacity, possibly by preventing or correcting hypoglycaemia (Bagby et al, 1978; Best and Partridge, 1930; Boje, 1936; Christensen and Hansen, 1939; Costill, 1974; Coyle et al, 1983; Dill et al, 1933).

Dill et al (1933) showed that when the external temperature was low, the performance of the dog, Joe, was virtually limitless when 30g of glucose and adequate water were supplied each hour. Christensen and Hansen (1939b) found that exhausted, hypoglycaemic subjects were able to resume exercise after glucose ingestion. They maintain that the increase in endurance following glucose ingestion is not due to increased glucose metabolism by muscles, but due to the correction of the hypoglycaemia and its symptoms. Ivy et al (1983) reported increased performance capacity when a glucose

polymer was ingested during exercise at 45% $\dot{V}O_2$ max.

Glucose ingestion during prolonged exercise results in an increased respiratory quotient indicating an increased carbohydrate utilization (Ahlborg and Felig, 1976; Benade et al, 1973), a decrease in plasma lipolysis (Ahlborg and Felig, 1976; Luyckx et al, 1978), decreased hepatic uptake of gluconeogenic substrates, increased hepatic glucose concentration and increased glucose uptake by the muscles in the exercising leg (Ahlborg and Felig, 1976). Similar effects were found when glucose was ingested 50-minutes before exercise commenced (Ahlborg and Felig, 1977). It is of interest that the rate of muscle glycogen utilization during exercise does not appear to be significantly reduced by glucose administration during exercise (Bergström and Hultman, 1967). Fructose ingestion may however act differently. Muscle glycogen depletion was significantly less during exercise when a fructose solution rather than a glucose solution or just water was ingested 45-minutes prior to a 30-minute treadmill run at 75% $\dot{V}O_2$ max (Levine et al, 1983).

In summary then, blood glucose levels at the end of exercise depend on the type of exercise, its duration, the work load and whether or not glucose is ingested

during exercise. Hypoglycaemia is most likely to occur when pre-exercise liver glycogen levels are low due either to fasting or eating a carbohydrate-poor diet, or when the exercise is of a moderate intensity and lasts more than two hours after which time, the rate of liver glucose production may fall behind that of muscle glucose uptake.

2.3 THE METABOLIC RESPONSE TO PROLONGED EXERCISE, MARATHON RUNNING IN PARTICULAR

(i) Serum Free Fatty Acid, Glycerol and Hormone Levels

During prolonged exercise there is a four-to-six-fold increase in plasma free fatty acid levels (Jooste et al, 1981; Keul et al, 1974; Keul et al, 1981; Luyckx, 1978; McKechnie et al, 1982; Rennie and Johnson, 1974) and a nine-to-fourteen-fold increase in plasma glycerol concentration (Jooste et al, 1981; Keul et al, 1974; Keul et al, 1981; McKechnie et al, 1982). Serum triglyceride levels fall (Keul et al, 1981), and serum growth hormone (McKechnie et al, 1982) and serum cortisol levels generally increase during exercise (Few and Thompson, 1980; Keul et al, 1981; Newmark et al, 1976) although one study (Viru and Körge, 1971) showed a decrease in serum cortisol levels. Serum insulin levels fall during prolonged exercise (Ahlborg and Felig, 1982).

(ii) The Respiratory Quotient (RQ) During Exercise

The Respiratory Quotient during exercise appears to depend on the fat and carbohydrate content of the athlete's pre-exercise diet, being high when a high-carbohydrate diet is consumed and low when a high-fat diet is consumed, and on the work intensity. The Respiratory Quotient is high as long as work intensity remains high, but decreases when carbohydrate stores become depleted (Christensen and Hansen, 1939a; Hedman, 1957; Hermansen et al, 1967). As described earlier, glucose ingested during exercise increases the Respiratory Quotient.

(iii) Fat Metabolism During Exercise

Prolonged dynamic exercise time stresses the oxidative system (Ahlborg et al, 1974) and as exercise becomes progressively prolonged, there is a shift in metabolism from carbohydrate oxidation to free fatty acid oxidation, with an accompanying decrease in work intensity.

Evidence for this is the decrease in Respiratory Quotient with increasing work time (Ahlborg et al, 1974; Costill, 1970; Hedman, 1957). Costill (1970) found that the Respiratory Quotient decreased from 0,88 at 10-minutes of exercise to 0,80 at 120-minutes of exercise, representing a metabolic shift from 61% carbohydrate and 39% fat oxidation to 33% carbohydrate and

67% fat oxidation. He has also shown that since blood glucose values were elevated for the duration of this exercise, this metabolic shift from mainly carbohydrate to mainly fat metabolism was unrelated to changes in blood glucose levels.

The post-race elevation in serum free fatty acids and glycerol (Jooste et al, 1981; Keul et al, 1974; Keul et al, 1981; McKechnie et al, 1982) is a result of increased lipid metabolism and further reflects a shift in metabolism with decreased carbohydrate utilization and increased fat oxidation.

The relationship between carbohydrate and lipid metabolism during and after prolonged exercise depends on the intensity, the type and the duration of muscular exercise, the nutrition of the athlete and the training state of the athlete (Scheele et al, 1979). Different dietary modifications have been shown to alter metabolism during exercise as a result of a change in muscle glycogen content which appears to influence free fatty acid availability (Astrand, 1967; Havel et al, 1963; Maughan et al, 1978; Rennie and Johnson, 1974b; Rodahl et al, 1964; Williams et al, 1976). Ingestion of carbohydrate during exercise decreases free fatty acid mobilisation and thus decreases free fatty acid availability as an oxidative substrate (Havel et al, 1963;

Rodahl et al, 1964). A high carbohydrate diet before exercise resulted in a decreased supply and utilization of free fatty acids during exercise (Maughan et al, 1978; Rennie and Johnson, 1974b; Williams et al, 1976) and the Respiratory Quotient during exercise was increased. In contrast, after a low carbohydrate diet, there was increased fat oxidation during exercise and this exerts a "glycogen sparing" effect on the low carbohydrate stores resulting from such a diet (Maughan et al, 1978). Costill et al (1977) have shown that the pre-exercise elevation of plasma free fatty acids caused a 40% decrease in the rate of muscle glycogen depletion during subsequent exercise. This would mean that in a near carbohydrate depletion state when free fatty acid levels are high, free fatty acid availability would exert a "glycogen sparing" effect.

Thus it would seem that fat metabolism during exercise becomes progressively more important as the body becomes progressively more carbohydrate depleted. Furthermore, high post-race serum free fatty acid levels would indicate the presence of near-total carbohydrate depletion. One study (Maron et al, 1975) reported the highest serum free fatty acid levels in the fastest marathon runners suggesting that they were the most carbohydrate depleted.

EFFECT OF TRAINING

Training brings about certain changes in the athlete's metabolic response to exercise. An increase in $\dot{V}O_2$ max (Bransford and Howley, 1979; Terblanche and Oelofsen, 1981), a decrease in resting plasma free fatty acid levels and a decreased rise of free fatty acid during exercise (Bransford and Howley, 1979; Johnson et al, 1969), a lower Respiratory Quotient during exercise (Bransford and Howley, 1979; Christensen and Hansen, 1939a; Hedman, 1957) with a slower rate of muscle glycogen utilization (Terblanche and Oelofsen, 1981) - indicate a greater reliance on fat metabolism during sub-maximal exercise after training.

SUMMARY

The most important points shown by this literature survey are the following :

(1) The muscle glycogen content of the exercising muscles decreases during prolonged exercise and the rate of utilization is related to work intensity and the level of pre-exercise glycogen stores.

(2) Endogenous carbohydrate stores are important in endurance

performance since :

(i) Carbohydrate is required in order to maintain a high work intensity,

(ii) exhaustion during prolonged exercise occurs simultaneously with carbohydrate depletion, and

(iii) by increasing pre-exercise muscle glycogen stores, performance may be improved.

(3) Muscle glycogen stores can be manipulated by a diet and exercise programme and are highest in trained subjects eating a high-carbohydrate diet. Less well-trained subjects may reach similar levels of muscle glycogen only if they follow a specific "carbohydrate-depletion, carbohydrate-loading" protocol.

(4) Blood glucose levels have been shown to increase, decrease or remain constant during exercise. Blood glucose levels at the end of the exercise depend on the duration of exercise, its work intensity and whether or not glucose is ingested during exercise.

(5) The extent to which hypoglycaemia could contribute to the fatigue experienced during prolonged exercise is presently unknown. Recent work suggests that hypoglycaemia is likely to occur when exercise at about 60% $\dot{V}O_2$ max is continued for longer than three hours

after which time, the rate of hepatic glucose production falls behind that of muscle glucose uptake.

CHAPTER 3

MATERIALS AND METHODS

In order to determine whether hypoglycaemia is a significant problem during marathon and ultra-marathon racing and the factors that influence the hypoglycaemia, three different studies were undertaken.

STUDY 1 : BLOOD BIOCHEMICAL CONSTITUENTS AT THE END OF MARATHON AND ULTRA-MARATHON RACES

In the first study, 25ml venous blood samples were drawn from a total of 256 runners at the completion of three South African long-distance races; the 42-km Peninsula Marathon, the 56-km Two Oceans Marathon and the 88-km Comrades Marathon (up run), all run during 1981.

From the samples taken at the 56-km and 88-km marathons, 10ml of blood from each sample was treated in the following manner: a 0,5ml aliquot of a preservative solution made up of Bacitracin^{R} (Bacidrin + Ginebatin (SERVA-Heidelberg) 1,45g $100ml^{-1}$ distilled water), plus Ethylenedianine Tetraacetic-acid (EDTA-3,7g $100ml^{-1}$), plus Trasylol^{R} (100 000 Kallidino-genase inhibitor units of Aprotinin in 0,9% NaCl + 0,9% Benzyl Alcohol (Sigma Chemical Laboratories) was added, and the sample was centrifuged once for 5-minutes, after it had been cooled on ice for about 30-minutes. The plasma was pipetted off and stored frozen for the assay of glucagon

and cortisol.

The remainder of the blood in each 25ml sample (15ml) and all samples from the 42-km race were centrifuged twice, once for 5-minutes and again 30-minutes later, a second time for 10-minutes. The plasma was pipetted off and stored frozen for later determination of glucose, free fatty acid, glycerol, insulin and alanine levels. Owing to cost considerations, serum alanine and glycerol concentrations were measured only on the fastest and the slowest runners in the 56-km and 88-km races.

On the day before the 42-km marathon, a separate group of 8 runners consented to have fasting blood samples drawn at the same time of day that they expected to finish the following day's race. Immediately after the race, blood samples were drawn from these athletes at 3-minute intervals for 15-minutes, and prepared and stored (as described above) for determination of glucose and free fatty acid concentrations in the immediate post-race period. The purpose of this section of the study was to establish the time course of the immediate post-race changes in these blood constituents. Immediate post-race urinary samples were collected and analysed for cortisol, sodium and potassium levels.

STUDY 2 : MEASUREMENT OF RESPIRATORY QUOTIENT DURING, AND MUSCLE GLYCOGEN LEVELS AFTER, MARATHON AND ULTRA-MARATHON RACES

In the second study, samples of expired respiratory gases were collected from runners (while they continued to run) competing in the same 42- and 56-km races, but in 1982. A 90-second sample of expired air was measured with one of three Koffrayani (Max Planck) Respirometers, which collected a 0,6% sample into a 2 litre sampling bag. The respirometers had been calibrated against a 5 litre syringe before the race and were carried on the back of a research assistant who ran next to the runner for the duration of the gas sampling. A second assistant ran alongside to control the sampling time and record the volume readings and temperature of the expired air before and after sampling. As soon as the sampling at each point was completed, the bags were transported directly to the Respiratory Clinic, Groote Schuur Hospital, for immediate analysis of their CO_2 and O_2 content. These data were used to calculate oxygen uptake and Respiratory Quotient by conventional equations (see Appendix 1).

To allow for changes in the gas composition of the sampling bags that occurred in the time period between gas sampling and gas analysis at the Respiratory Clinic - a delay which

due to travelling time was up to one hour on occasion - the following experiment was undertaken in the laboratory. Eight of the sampling bags were filled with samples of medical gas and the percentage CO_2 and O_2 of the air in the bags was sampled immediately and at 30-, 60-, 90-, 120- and 180-minutes. The mean values of the eight bags at each analysis time were calculated and used to plot a graph (Figure 1). The $\%O_2$ in the bags remained constant over the 180-minutes. The $\%CO_2$ in the bags fell at a constant rate over the time period at a rate of 0,24% every 60-minutes. This value was then used as a correction factor for the calculation of the $\%CO_2$ in the expired air at the time of sampling.

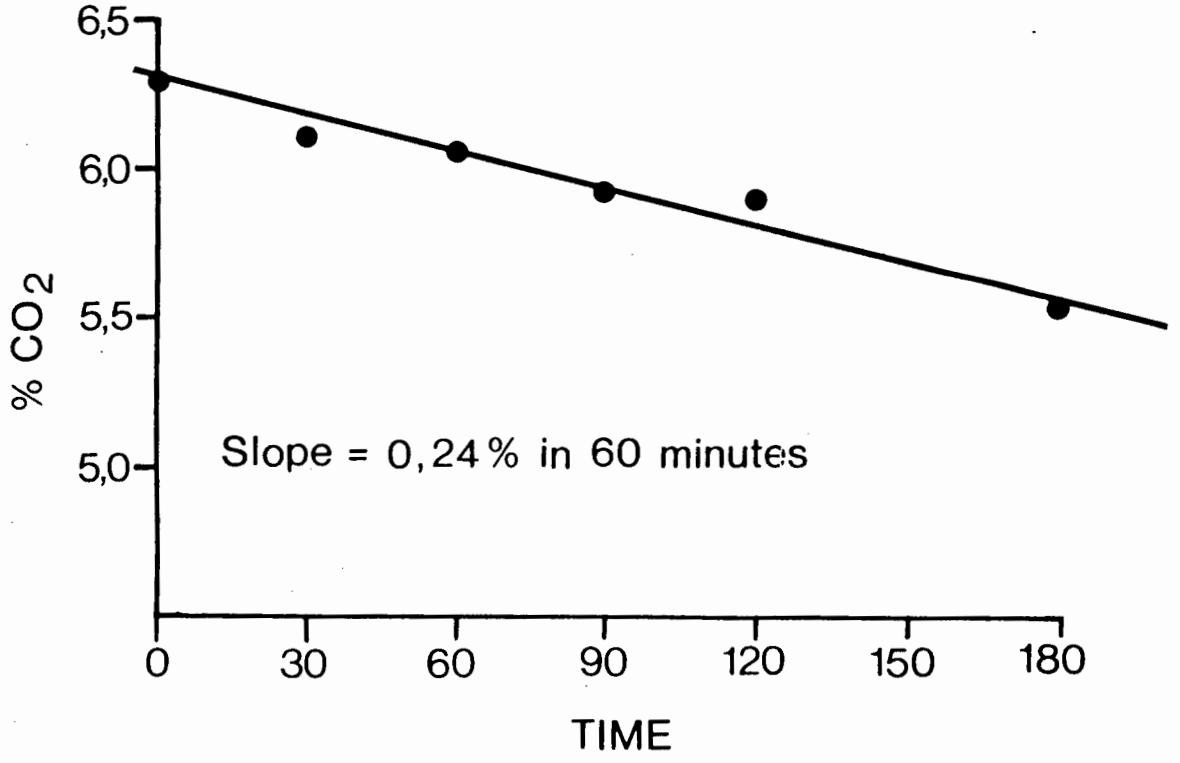


FIGURE 1 : CO_2 leaked at a constant rate from the sample bags - a rate of 0,24% in 60-minutes.

Eight runners were studied at three points (8-, 21- and 32-km) in the 42-km race and 11 runners, also at three points (10-, 38- and 51-km) in the 56-km race. Attempts to collect gas samples during the 1982 Comrades Marathon (91-km down run) proved largely unsuccessful due to poor concentration from the exhausted runners, especially near the end of the race, and the difficulty in getting adequate O_2 and CO_2 analyses performed in another centre.

At the completion of these races, muscle biopsies were performed with a 5,0mm Stille-Werner needle (Stille-Werner, Stockholm) on five runners after the 42- and 91-km races, and on 6 runners after the 56-km race. Muscle tissue was taken from the quadriceps muscle and the biopsy tissue was immediately stored in liquid nitrogen pending analysis for its glycogen content by conventional laboratory methods (App. 2).

It was planned to take samples of blood while the runners continued to run, at the same points as gas sampling was done. However, our method proved unsuccessful and we were unable to obtain worthwhile results. In order to collect that information, the third study was designed.

STUDY 3 : EFFECT OF INGESTION OF THREE DIFFERENT CARBOHYDRATE-CONTAINING DRINKS ON BLOOD BIOCHEMICAL PARAMETERS AND MUSCLE GLYCOGEN UTILIZATION DURING A 42-KM STANDARD MARATHON

The third study was undertaken in 1983 during the Stellenbosch Appletiser 42-km standard marathon.

Eighteen healthy, male university students living in a university residence, consented to participate. Since they were all in a residence their diets on the days prior to the race could be controlled. These runners were matched according to their projected times for the race, into 3 groups so that each group had equal numbers of fast, average and slow runners. The implications of the study, but not its scientific rationale, were explained to the subjects and all signed consent forms to participate in this study. All were financially rewarded for their participation.

The marathon was run on a Saturday morning. The previous Sunday, all participants started on a carbohydrate depletion diet eating a low carbohydrate diet (100 - 150g carbohydrate per day) and continued exercising daily for three days (Sunday, Monday and Tuesday). For the following three days (Wednesday to Friday) they consumed a high carbohydrate diet (300 - 320g carbohydrate per day). In addition, they ingested 300g of a glucose polymer (Breakthru Carbolode). On

the days following the marathon (Saturday to Wednesday) an average diet was consumed consisting of 400 - 550g carbohydrate per day. During the three days of carbohydrate loading, the subjects did not exercise. On the day before the marathon, all participants in the study reported to the exercise laboratory where they were weighed and their height and age recorded. Each underwent a muscle biopsy taken from the gastrocnemius muscle for the measurement of pre-race muscle glycogen content according to the technique of Good et al (1933). (See Appendix 2)

On the following day, shortly before the start of the race, venous blood samples were taken from a forearm vein from all participants.

During the race, the three matched groups were given different solutions to drink; Group 1 was given a 2% glucose in water solution, Group 2 an 8% fructose in water solution and Group 3 an 8% glucose polymer (Corn Syrup) in water solution. These drinks were colour coded and freely available to the participants of the study every three kilometers during the race. The subjects were unaware of the meaning of the colour coding and thus were ignorant of what they were drinking. No-one besides the researchers knew the composition of the drinks, nor the rationale for choosing the different solutions. The runners' fluid

intake was monitored at four points in the race and used to calculate their carbohydrate intake during the race. At three points during the race (10-, 21- and 32-km) the runners were stopped briefly in order to obtain a 25ml venous blood sample. The runners sat down while the blood sample was drawn from a forearm vein as quickly as possible. Samples for blood glucose analysis were usually obtained within 10-seconds of the athlete sitting down. While the blood was being sampled, the runner's subjective physical state was assessed on a scale of 1 - 6 :

6 = fresh, effortless
5 = fresh, moderate effort
4 = mildly fatigued, hard effort
3 = fatigued, able to concentrate
2 = exhausted, able to concentrate, still pushing
1 = exhausted, unable to concentrate, indifferent to what is happening

At the finish, another 25ml blood sample was taken after the runner had completed the formalities of finishing. This usually took between 2 - 5 minutes. Thereafter, a second biopsy was taken from each subject from the gastrocnemius muscle on the leg opposite to that which was biopsied before the race.

Rectal temperatures were measured with a clinical thermometer within 5-minutes of stopping exercise.

The blood samples were prepared and stored as previously described and later analysed for glucose, free fatty acid and insulin levels. Muscle biopsies were immediately stored in liquid nitrogen pending later analysis of their glycogen content.

The subjective perception of pain in the muscles following the marathon (post-marathon stiffness) was assessed on a scale of 0 - 3 from the night after the marathon to three days later.

0 = no pain

1 = pain on palpation of muscle belly (calf) only

2 = as 1, but unable to flex knee fully, minimal discomfort when walking

3 = as 2, with marked discomfort when walking

Body weight was measured on a SECA 77 Electronic Scale (Seca Instruments, Germany) before dietary control was instituted, after the carbohydrate depletion phase, after the carbohydrate loading phase, immediately pre- and post-race, the evening after the race, the morning and afternoon the day after the race and the next three days.

ANALYTICAL METHODS

Serum glucose levels were determined using an automated Beckman Glucose Analyser (Beckman Instruments, Fullerton, California). Serum free fatty acid levels were initially determined using the titration method of Dole and Meinertz (1960 - Appendix 3). Samples taken during the Stellenbosch Marathon were analysed at the National Research Institute for Nutritional Diseases (Dr A.J.S. Benade) using chromatography (Victor et al, 1983). Serum immunoreactive insulin and plasma immunoreactive glucagon concentrations were measured with, respectively, the Sorin Biomedica Insulin Radioimmunoassay Kit (Ref Insik-1) and the Glucagon Radioimmunoassay technique using pancreatic specific anitbody 30k obtain from Roger H. Unger (University of Texas, South Western Medical School, Dallas, Texas, U.S.A.). Alanine levels were determined by enzymatic analysis described by Lowry (1972) (Appendix 4).

Respiratory gases collected were analysed for their carbon dioxide (CO_2) and oxygen (O_2) content using Analysers at the Groote Schuur Respiratory Clinic (Appendix 5). Muscle biopsy tissue was analysed to determine its glycogen content using the method described by Good et al (1933) (Appendix 2).

METABOLIC CALCULATIONS

To calculate the oxygen consumption, carbon dioxide production and Respiratory Quotient during exercise, metabolic computations were carried out, as set out in, Appendix 1.

STATISTICAL METHODS

Statistical analysis was performed on a Hewlett Packard Calculator using conventional equations for mean and standard deviations, unpaired (studies 1 and 2) and paired (study 3) t-tests.

CHAPTER 4

RESULTS

STUDY 1 : BLOOD BIOCHEMICAL PARAMETERS AT THE END OF MARATHON AND ULTRA-MARATHON RACES

Blood Glucose, Free Fatty Acid, Insulin and Glucagon Levels

The blood glucose and serum free fatty acid levels of the eight subjects studied the day before, and for fifteen minutes immediately after the 42-km race in 1981 are shown in Figure 2. Both blood glucose and free fatty acid levels were significantly elevated after the race, and although the blood concentrations fell slightly during the first fifteen minutes after the race, the fall was not statistically significant.

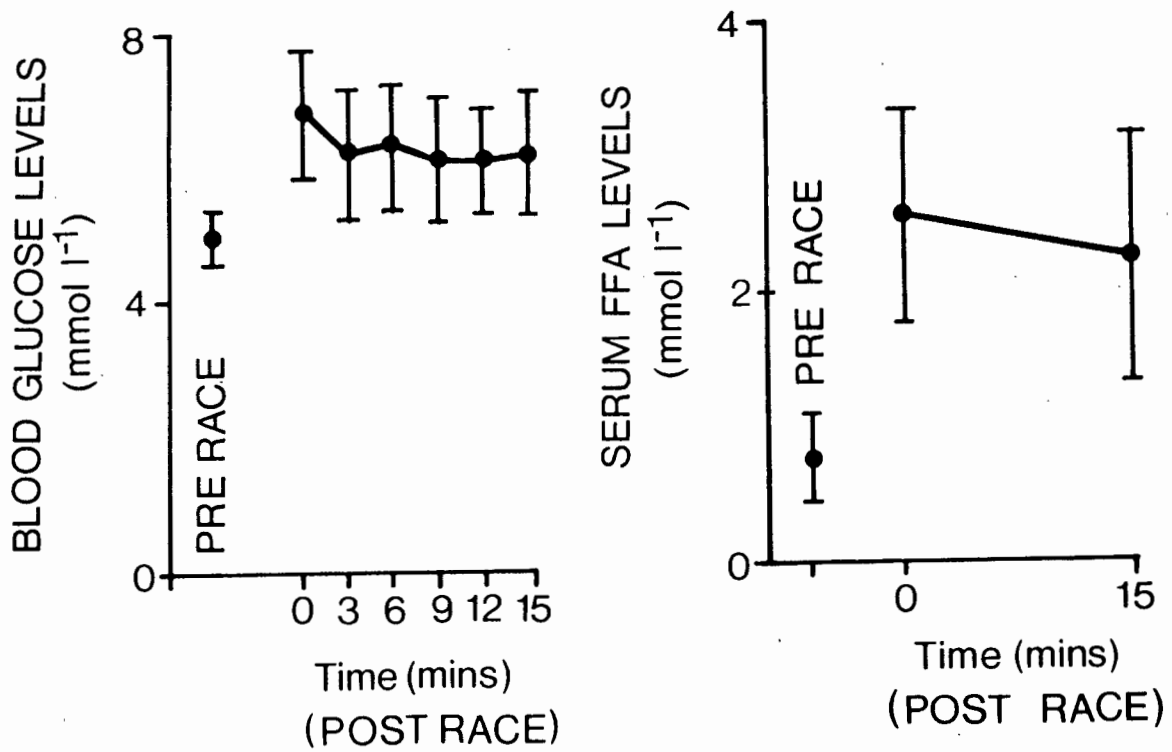


FIGURE 2 : Blood glucose and free fatty acid levels are elevated in the immediate post-race sample but do not change significantly during the subsequent 15-minutes.

Immediate post-race insulin levels (22,7 ± 15,4 μU ml^{-1}: mean ± standard deviation (SD) for 8 runners) were not significantly different from pre-race fasting levels (17,1 ± 2,6 μU ml^{-1}), and although insulin levels were lower fifteen minutes after the race than immediately after, the difference was again not significant. Glucagon levels were significantly elevated after the race (109,1 ± 20,1 pg ml^{-1} vs 72,3 ± 31,1 pg ml^{-1}; post-race vs pre-race; $p < 0,02$).

When comparing results from the three marathon races, it was found that mean blood glucose and insulin levels (Figure 3) fell progressively with each successively longer race, whereas serum free fatty acid, glycerol and glucagon levels rose with increasing race distance (Figure 3). Although blood glucose levels fell progressively with increasing race distance, these levels were all well above the mean resting level of 5,0 ± 0,4 mmole ℓ^{-1} measured in the fasting subjects before the 42-km race. Even after the 88-km Comrades race the mean blood glucose value was 5,6 ± 0,01 mmoles ℓ^{-1} (105 samples). Glycerol and alanine levels measured in the fastest and slowest runners in the 56- and 88-km races showed no trends with increasing distance, although the values tended to be lower in the better runners (data not shown).

To determine whether athletic ability influenced the blood

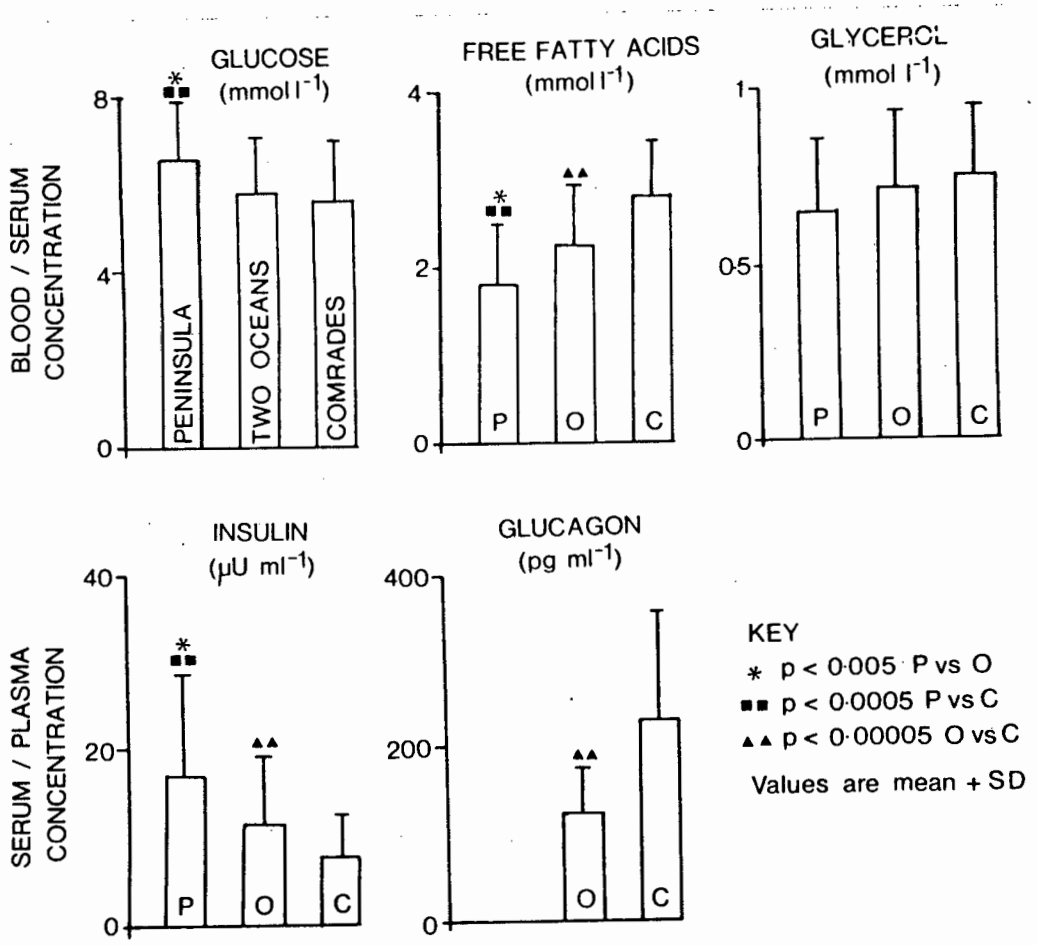


FIGURE 3 : Post-race blood glucose and serum insulin levels fell with increasing racing distance, whereas free fatty acid, glycerol and glucagon levels rose. Mean blood glucose levels remained above resting values at the end of all races.

fuel/hormonal response to marathon and ultra-marathon running, we compared the measured parameters in different groups of runners, grouped according to their finishing times in the respective races (Figures 4 - 7). The groups

were selected so that each included approximately the same number of runners (range between eight and fourteen). The groups included athletes finishing the different races in the following time spans : 42-km race within mean times ranging from 2-hrs 24-mins 23-sec (range; 2-hr 13-min 21-sec - 2-hr 29-min 11-sec) to 3-hrs 37-min 18-sec (range; 3-hr 24-min 14-sec - 3-hr 53-min 18-sec); the 56-km race within mean times ranging from 3-hrs 27-min 59-sec (range; 3-hr 05-min 37-sec - 3-hr 37-min 56-sec) to 5-hrs 16-min 05-sec (range; 5-hr 01-min 00-sec - 5-hr 34-min 31-sec); and the 88-km race within mean times ranging from 5-hrs 59-min 42-sec (range 5-hr 37-min 28-sec - 6-hr 18-min 08-sec) to 9-hrs 37-min 48-sec (range; 8-hr 52-min 40-sec - 10-hr 15-min 25-sec). The winner of each race was included in the study, as were six, four and eight of the first ten finishers in the 42-, 56- and 88-km races respectively.

Figure 4 shows that there were no significant differences in mean blood glucose levels between any of the groups in any of the races, although values in the 42- and 56-km races tended to be highest in the best runners. The lowest blood glucose level measured was a value of 1,9 mmoles ℓ^{-1} in the athlete who finished the 88-km race in the 691st position in a time of 7-hrs 58-min 47-sec. The only low value measured in a top finisher was one of 3,4 mmoles ℓ^{-1} in the athlete who finished eighth in the 88-km race in a time of 5-hrs 56-min 02-sec.

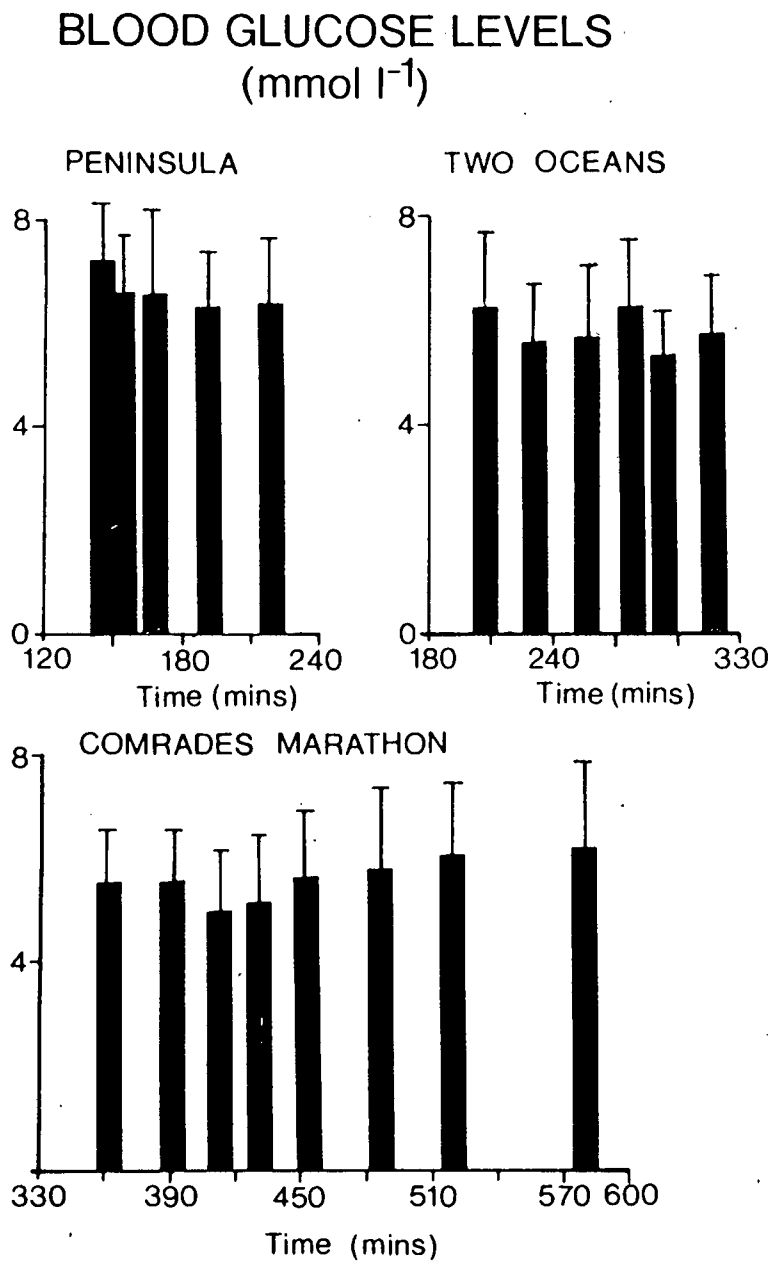


FIGURE 4 : Blood glucose levels were not different amongst the different groups of runners in any of the races.

Figure 5 shows that after the 42-km race the blood free fatty acid concentrations were significantly lower in the faster than in the slower runners. After the 56-km race, the mean blood free fatty acid level of the fastest group was significantly lower than that of the slowest group but after the 88-km race, no inter-group differences were found.

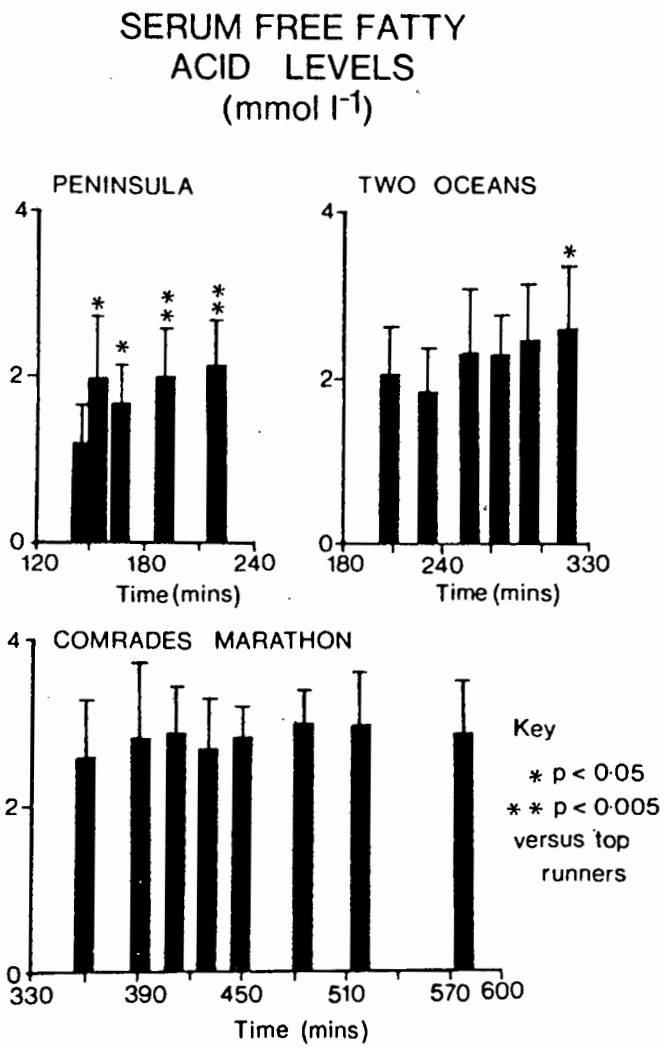


FIGURE 5 : Serum free fatty acid levels were lowest in the fastest runners after the 42- and 56-km races.

Insulin and glucagon levels were not different in any of the groups after any of the races (Figures 6 and 7), although there was a trend for insulin levels to be higher in the faster runners in the 42- and 56-km races and in the slower runners in the 88-km race. Glucagon levels tended to be highest in the group who completed the 88-km race just under seven and a half hours.

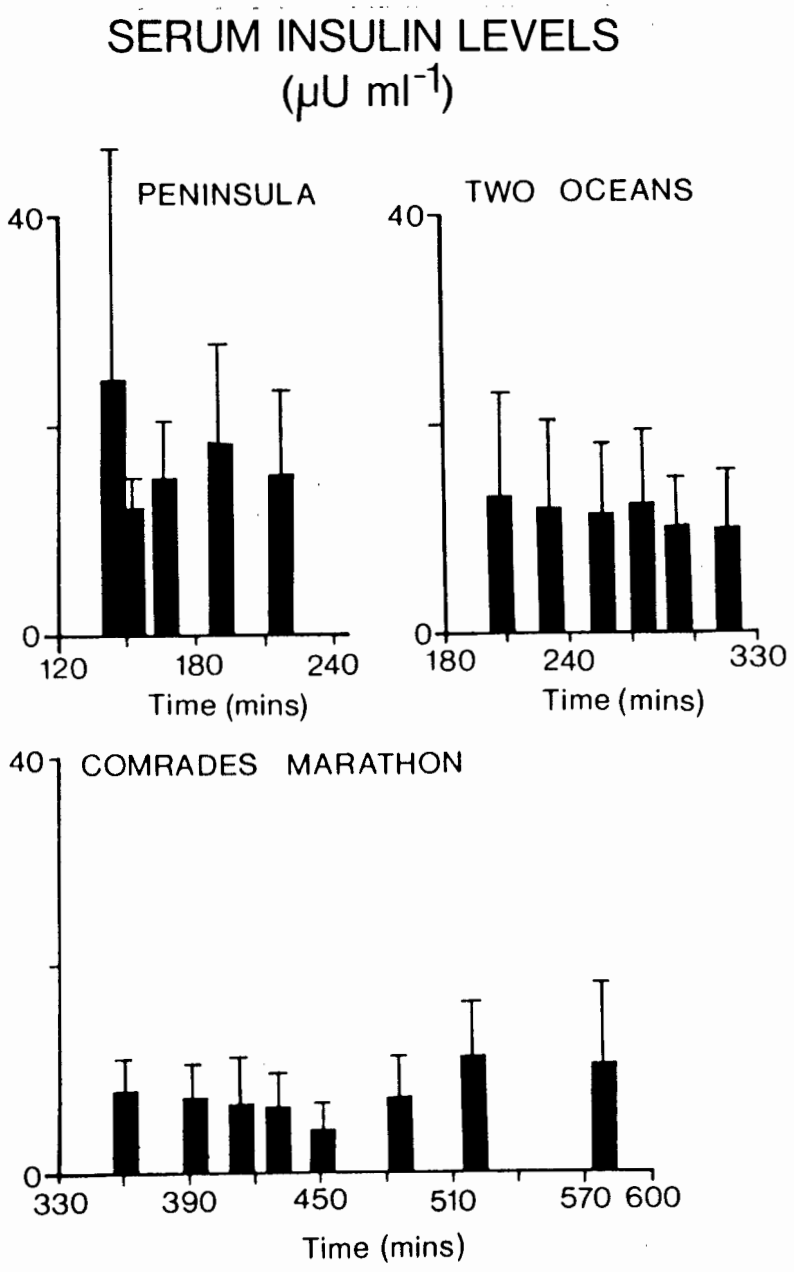


FIGURE 6 : Serum insulin levels were not different amongst the different sub-groups of runners in any of the races.

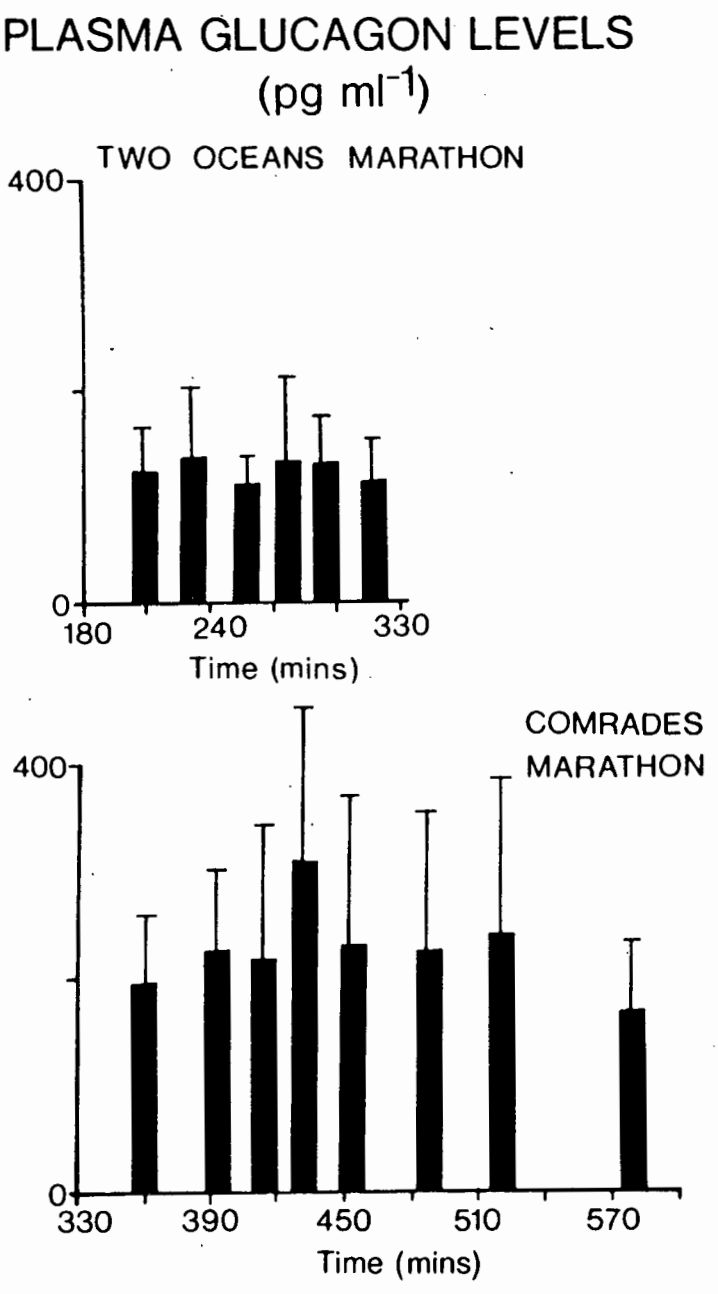


FIGURE 7 : Plasma glucagon levels were not different amongst the different sub-groups of runners in either the 56- or 88-km races.

There were no differences in the plasma or urinary cortisol levels in the different groups of athletes. Note that the different groups correspond with the groupings according to time over which marathon was run, referred to in the text.

TABLE 1 : Urinary and plasma cortisol levels in the different groups of runners after the 56- and 88-km ultra-marathon races.

88-km Comrades Marathon

GROUP	URINARY CORTISOL LEVELS (n mol ℓ^{-1})	PLASMA CORTISOL LEVELS (n mol ℓ^{-1})
1	980 ± 653 (mean ± SD)	1358 ± 215 (mean ± SD)
2	927 ± 496	1288 ± 498
3	788 ± 581	1173 ± 230
4	858 ± 1035	1163 ± 387
5	870 ± 715	1202 ± 351
6	989 ± 618	1008 ± 298
7	599 ± 550	1076 ± 345
8	536 ± 412	1085 ± 319

56-km Two Oceans Marathon

1	501 ± 265 (mean ± SD)	887 ± 258 (mean ± SD)
2	448 ± 203	808 ± 124
3	723 ± 1139	979 ± 395
4	424 ± 280	835 ± 270
5	459 ± 210	916 ± 310
6	591 ± 807	913 ± 305

STUDY 2 : MEASUREMENT OF RESPIRATORY QUOTIENT DURING, AND MUSCLE GLYCOGEN LEVELS AFTER, MARATHON AND ULTRA-MARATHON RACES

Figures 8 and 9 show the Respiratory Quotient (RQ) measured at different points during, and the muscle glycogen levels after, the different races in 1982. There was a progressive fall in RQ during the 56-km race with values approaching 0,70 near the end of the race, indicating that fat was the predominant energy source. RQ values during the standard marathon did not reach equally low values suggesting that the athletes were less carbohydrate-depleted at the end of the 42-km race than they were at the end of the 56-km race. Post-race glycogen levels were lower, the longer the race distance.

Table 2 lists individual values of RQ, Oxygen uptake ($\dot{V}O_2$) and post-race muscle glycogen levels in a group of runners, listed in the order that they finished the 1982 42-km race. The striking observation is that the fastest runners had much higher post-race muscle glycogen levels despite there being little difference in their respective RQ values during the race. The same pattern is apparent after the 56-km race of that year. After the 91-km Comrades marathon, all runners had low muscle glycogen levels.

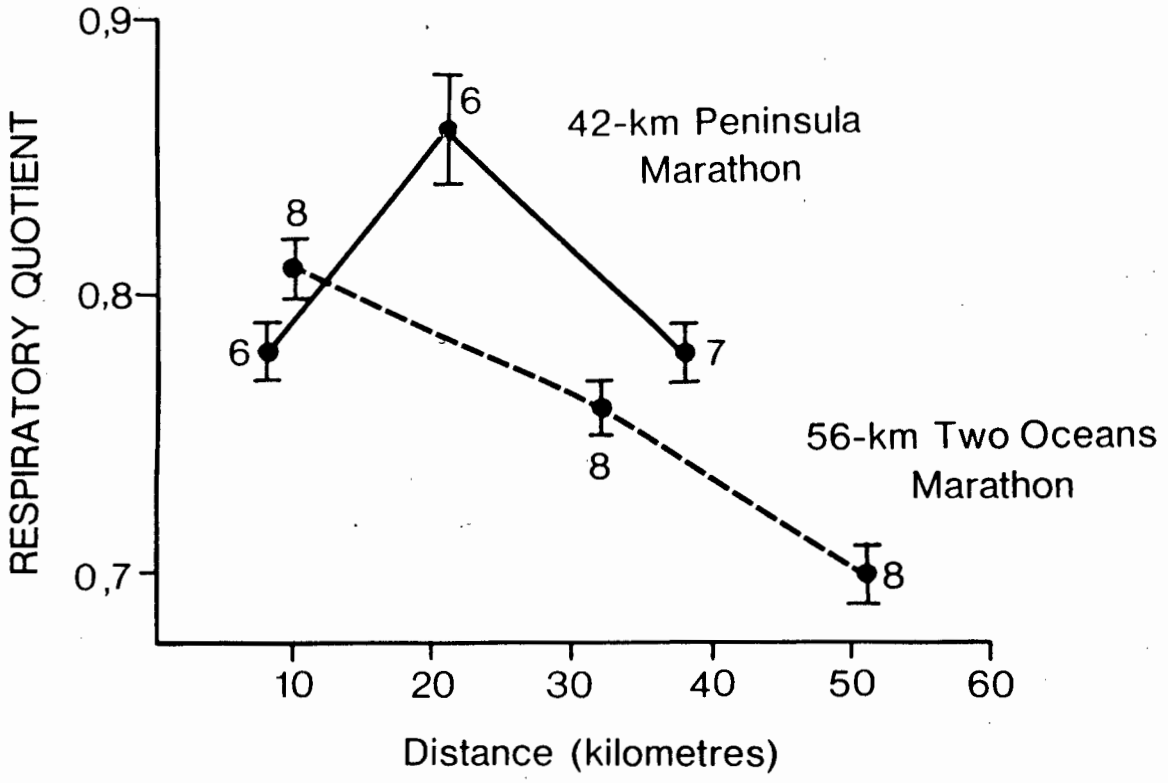


FIGURE 8 : RQ fell progressively during the 56-km race reaching levels close to 0,7 indicating predominantly fat oxidation. Values at the end of the 42-km race were not equally low, suggesting less severe carbohydrate-depletion. Values are mean and standard deviation (number of observations).

It is however, questionable whether the very high muscle glycogen levels in the fastest runners in the 42-km race (G.L. and B.M.) and in subject G.L. after the 56-km race can be accurate since it seems unlikely that such high values could be present after running a marathon.

However, if the subjects had heavily carbohydrate-loaded before the race and maintained a high carbohydrate intake during the race, then these values are feasible.

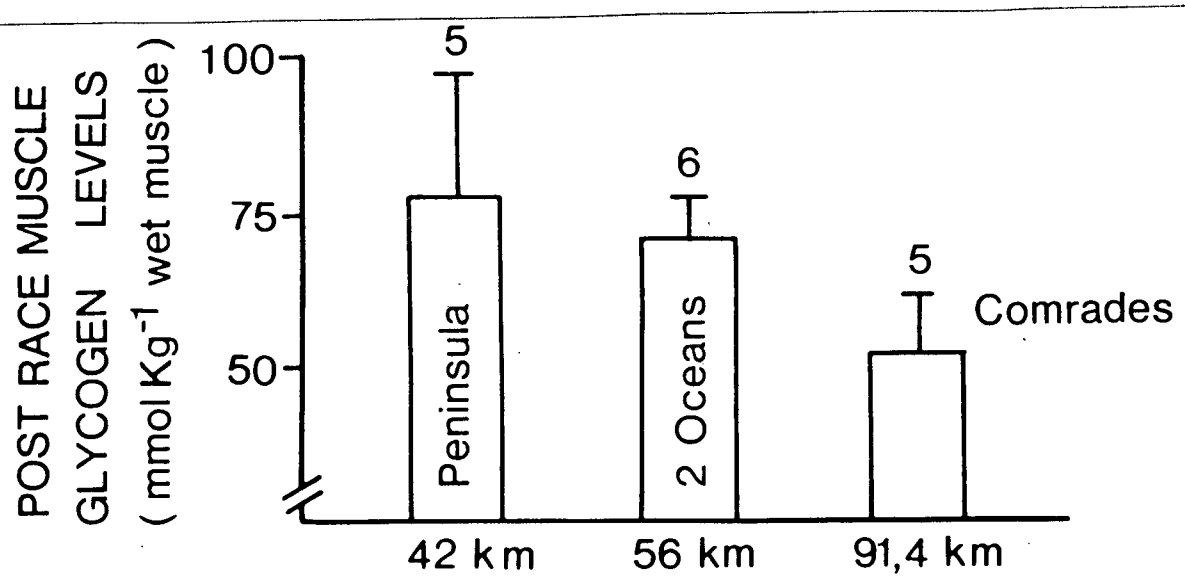


FIGURE 9 : Post-race muscle glycogen levels fell with increasing race distance. Values are expressed as mean and standard deviation (number of observations).

TABLE 2 : Respiratory Quotients and Muscle Glycogen Levels in Ultra-Marathon Runners

1982 42,2-km Peninsula Marathon

ATHLETE	RACE TIME (hr:min:sec)	RQ at 21-km	$\dot{V}O_2$ (litres min^{-1})	POST-RACE MUSCLE GLYCOGEN CONTENT (mmoles kg^{-1} wet muscle)
1 (G.L.)	2:30:24	0.86	4.17	229.3
2 (B.M.)	2:46:52	0.83	4.07	191.5
3 (R.G.)	2:48:31	0.85	3.47	167.6
4 (T.N.)	2:57:20	0.85	3.92	22.2
5 (G.I.)	3:02:29	0.82	2.64	43.6

1982 56-km Two Oceans Marathon

1 (G.L.)	3:28:52	0.86	4.02	219.8
2 (B.M.)	3:27:00	0.85	3.80	100.8
3 (R.G.)	3:56:20	0.83	4.02	48.8
4 (T.N.)	4:25:34	0.72	3.73	65.7
5 (D.L.)	4:35:30	0.70	2.32	116.8

1982 91,4-km Comrades Marathon

1 (T.N.)	7:17:30	-	-	64.2
2 (R.G.)	7:28:11	-	-	21.2
3 (G.I.)	7:37:20	-	-	92.3
4 (D.L.)	8:44:11	-	-	54.1

STUDY 3 : EFFECT OF INGESTION OF THREE DIFFERENT CARBOHYDRATE CONTAINING DRINKS ON BLOOD BIOCHEMICAL PARAMETERS AND MUSCLE GLYCOGEN UTILIZATION DURING A 42-km STANDARD MARATHON

The two previous studies appear to indicate :

(1) That hypoglycaemia is uncommon during marathon and ultra-marathon races, and

(2) that faster runners may be less carbohydrate-depleted at the end of marathon and ultra-marathon races.

However, in neither of these studies had blood glucose levels been measured during the race nor had post-race muscle glycogen levels been measured in a sufficiently large cross-section of runners to determine whether there were differences in the degree of muscle glycogen depletion between the best and worst runners. In addition, the effect of carbohydrate ingestion on blood glucose levels and muscle glycogen utilization during a 42-km standard marathon had not been studied.

We therefore undertook the third study in which blood glucose levels were measured in the three groups of athletes drinking three different carbohydrate-containing drinks. Blood samples were drawn at three points during, and again, immediately

after the race, when muscle biopsies were also taken. We were therefore able to study the effects of the different carbohydrate solutions on blood glucose levels and muscle glycogen utilization during the race.

(1) Carbohydrate and Fluid Ingestion

Carbohydrate consumption during the race was calculated from the fluid intake during the race and the carbohydrate content of the different drinks in each group (Table 3). The group drinking the 2% glucose solution ingested significantly less carbohydrate during the race than did the other two groups. Total fluid intake was not different between the groups.

TABLE 3 : Fluid and carbohydrate ingestion of the three different groups during the 42-km marathon.

CARBOHYDRATE INGESTION GROUP	AMOUNT OF FLUID INGESTED (ml)	AMOUNT OF CARBOHYDRATE INGESTED (g)
GLUCOSE (2%)	2015,0 ±332,5	40,3† ±6,7
GLUCOSE POLYMER (8%)	1653,3 ±422,1	132,2 ±33,8
FRUCTOSE (8%)	1850,0 ±474,9	148,0 ±37,9

† Significantly different from the other two groups

Values are expressed as mean ± SD for 6 runners in each group.

(2) Blood Glucose Levels

Figure 10 shows that there were no significant differences in blood glucose levels in any of the three groups at any point in the race. There was a tendency for blood glucose levels to be increased by 1 - 2 mmoles ℓ^{-1} at the 10-km mark and then to decrease at the 21- and 32-km marks to values equal to the pre-race level. The immediate post-race value was increased but was lower than the 10-km readings. This tendency was the same in all groups and was therefore not influenced by the type of fluid ingested.

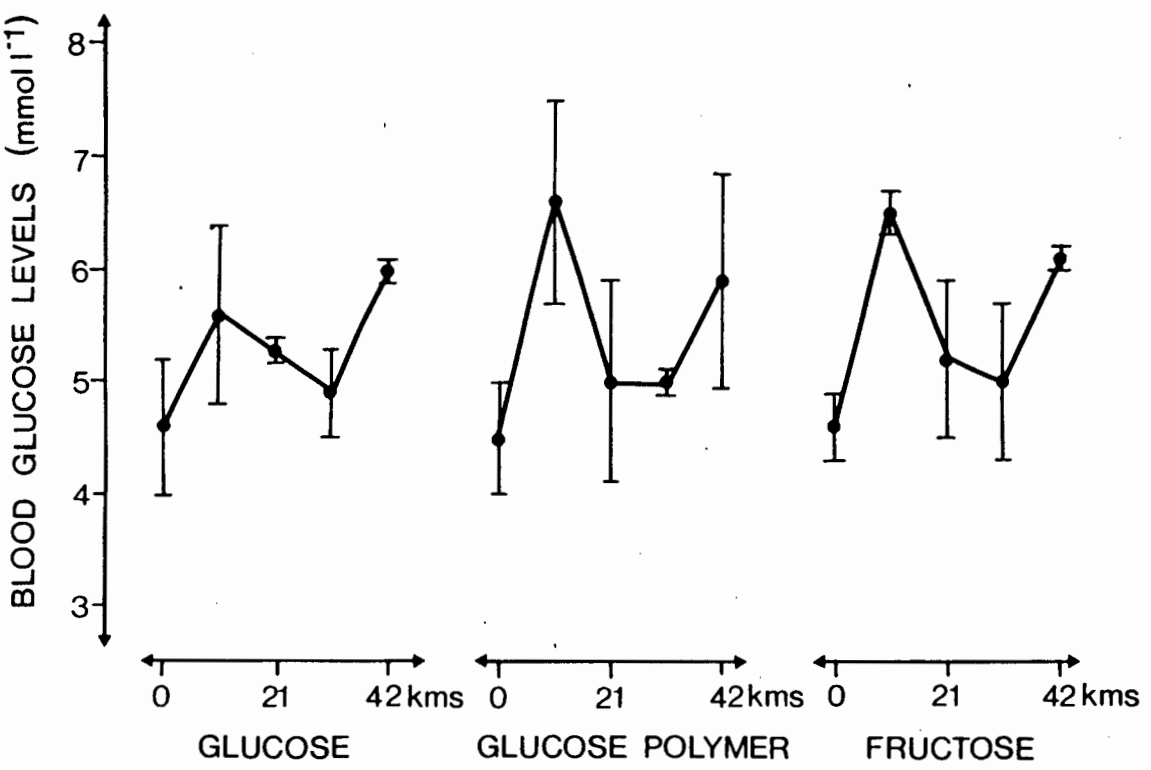


FIGURE 10 : After an initial rise, blood glucose levels fell during exercise and rose steeply at the end of exercise - probably due to stopping exercise.

There were no differences between the groups in blood glucose levels.

Of special interest was the finding that the blood glucose levels measured after the race were much higher than those measured at the 32-km mark. This is almost certainly an effect of stopping running, as the athletes did not ingest any carbohydrate between the time they stopped running and blood sampling. Furthermore, it is unlikely that the trend for blood glucose levels to be falling from the 10-km mark should suddenly be reversed by 42-km when hepatic glucose production is likely to be decreasing and muscle glycogen uptake to be increasing (Ahlborg et al, 1974; Ahlborg and Felig, 1982). This rise presumably represents relative glucose over-production by the liver. The same mechanism would explain the marked rise in blood glucose levels at the 10-km mark. Of the 90 blood glucose values measured in this experiment, only 4 were below 4 mmoles ℓ^{-1} and none were below 2,5 mmoles ℓ^{-1}. The lowest value (2,6 mmoles ℓ^{-1}) was measured in an athlete drinking the glucose polymer solution after he had run 32-km.

(3) Serum Free Fatty Acid Levels

Figure 11 shows serum free fatty acid levels during the race in the three different groups. No significant differences were noted between any of the groups at any point.

The tendency was for the free fatty acid levels in the blood to rise steadily up to the 32-km mark with a sharp rise at the 42-km mark, reaching values about four times greater than pre-race values.

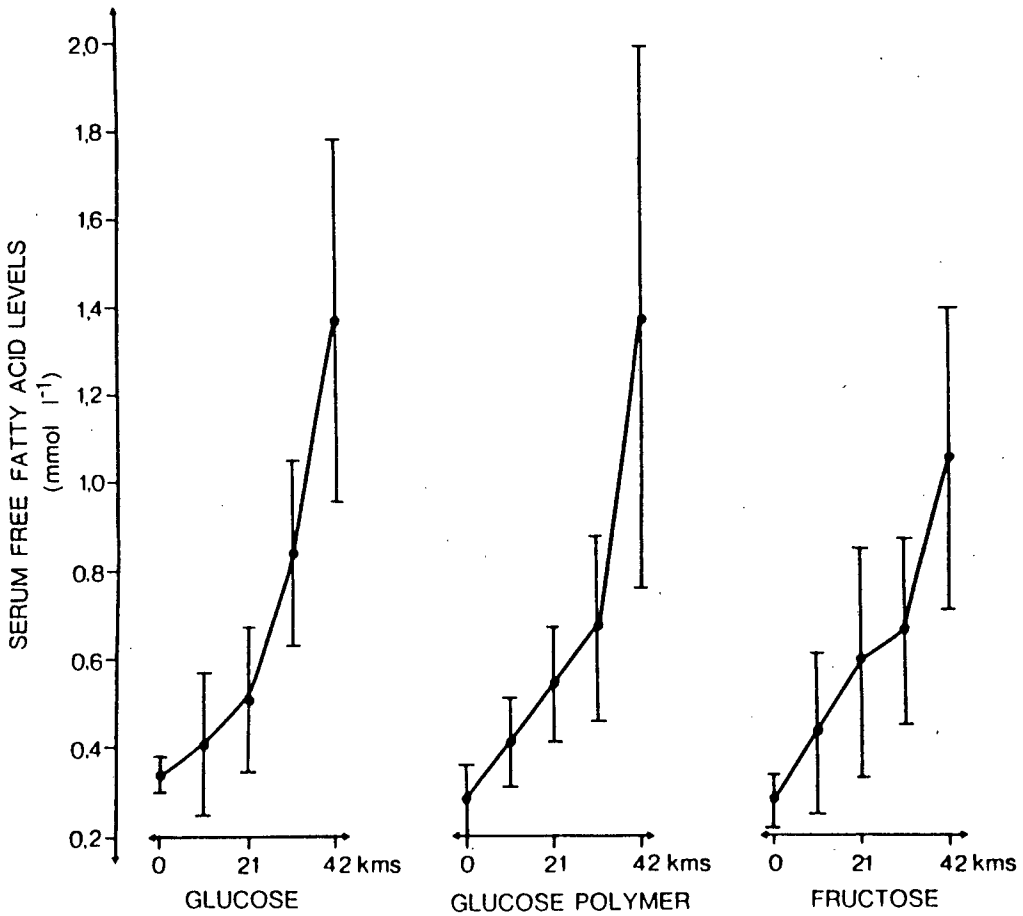


FIGURE 11 : Serum free fatty acid levels rose progressively and equally in all groups. The marked rise in all groups at 42-km may be due to the effects of stopping running. (See also, Figure 10)

(4) Serum Insulin Levels

Serum insulin levels in the three groups during the race

are shown in Figure 12. There were no significant differences between the three groups although the trend in the glucose and glucose-polymer groups was different to that of the fructose group. In the former, the trend was for the insulin levels to fall during the race, whereas in the fructose group, serum insulin levels were very low before the race, suggesting that the members of this group had not eaten breakfast, and were rising at 32-km.

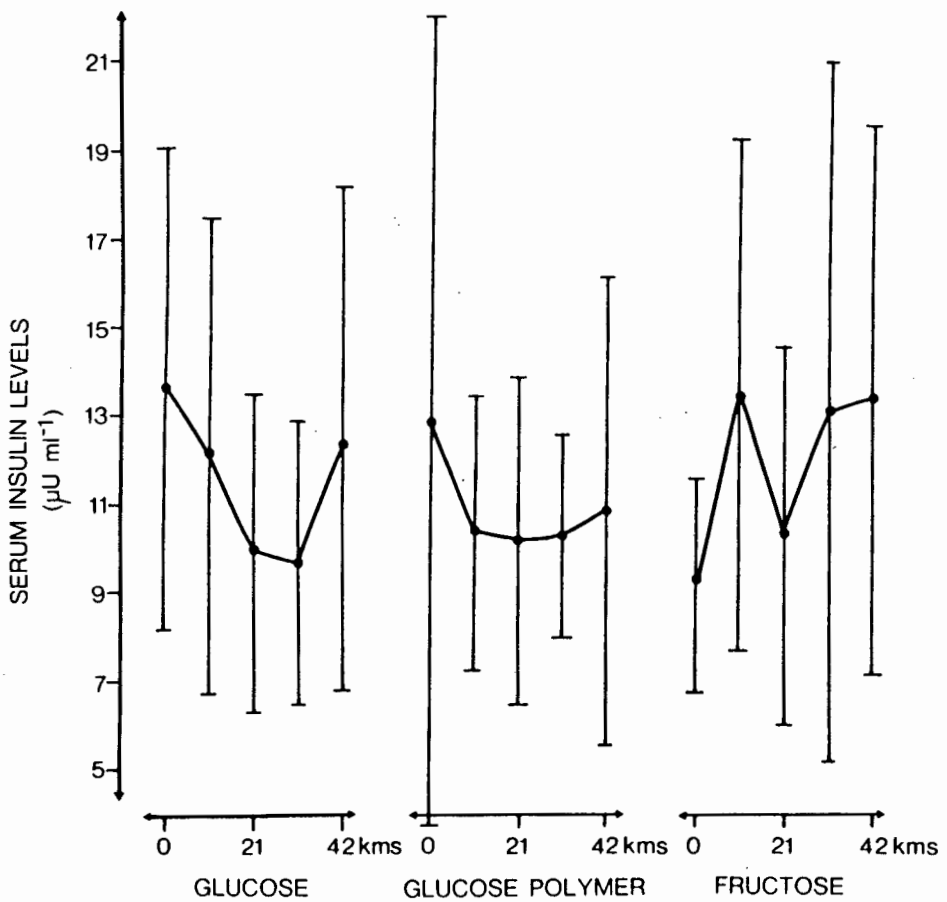


FIGURE 12 : Serum insulin levels fell during the race but, like blood glucose (Figure 10) and serum fatty acid levels (Figure 11), rose at the 42-km mark. There were no differences between groups in serum insulin levels.

(5) Muscle Glycogen Levels

There was no significant difference in the pre-race muscle glycogen levels between the three groups (mean values : 238,7 ± 34,8 mmoles kg^{-1} in the glucose group, 210,2 ± 64,8 mmoles kg^{-1} in the glucose polymer group and 242,3 ± 105,3 mmoles kg^{-1} in the fructose group). All three groups were equally depleted after the race reaching mean levels of 57,1 ± 30,2 mmoles kg^{-1}; 53,3 ± 32,2 mmoles kg^{-1} and 42,9 ± 42,3 mmoles kg^{-1} in the respective groups (Figure 13). Two days after the race the muscle glycogen content in the three groups had risen to mean levels of 214,0 ± 38,8 mmoles kg^{-1}, 159,4 ± 54,0 mmoles kg^{-1} and 144,6 ± 60,7 mmoles kg^{-1} respectively (Figure 13). These values were not significantly different.

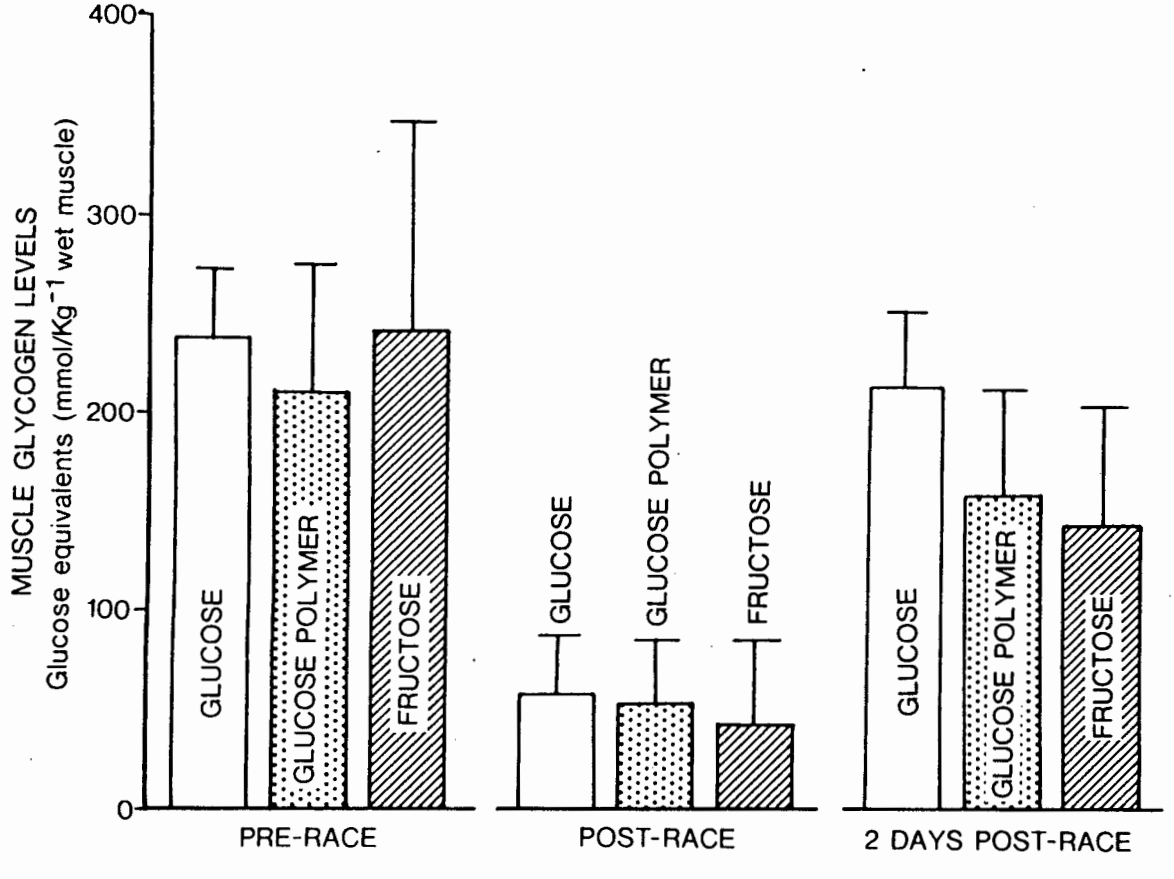


FIGURE 13 : Muscle glycogen levels were markedly reduced at the end of the race. The type of carbohydrate ingested during the race had no effect on muscle glycogen utilization during the race.

There was no difference in the degree of muscle glycogen depletion between runners finishing the race at different times (Table 4). Nor was there any difference between the groups in the rates of muscle glycogen combustion during the race (Table 5).

TABLE 4 : Muscle glycogen levels (mmoles kg^{-1} wet muscle) in runners finishing the marathon in different times.

	PRE-RACE	POST-RACE	2 DAYS POST-RACE
FASTEST GROUP (2:55 - 3:10)*	269,2 ±36,4	42,5 ±26,4	207,1 ±46,5
MEDIUM SPEED GROUP (3:17 - 3:45)*	233,8 ±100,1	61,4 ±42,4	160,7 ±76,1
SLOWEST GROUP (3:45 - 4:16)*	188,2 ±43,7	49,3 ±34,0	150,3 ±34,2

* Range and finishing times expressed in hr:min.

Values are expressed as mean ± SD for 6 runners in each group.

TABLE 5 : Rates of muscle glycogen combustion (mmoles kg^{-1} wet muscle min^{-1}) in the three different groups of runners.

	GLUCOSE GROUP	GLUCOSE POLYMER GROUP	FRUCTOSE GROUP
MUSCLE GLYCOGEN COMBUSTION	0,92 ±0,38	0,77 ±0,24	0,95 ±0,46

Values are expressed as mean ± SD for 6 runners in each group.

(6) Body Weight Changes

Figure 14 shows the changes in body weight during the 12 days of the experiment. Body weight fell by about 2,5-kg during the carbohydrate-depletion phase of the diet and rose a further 1,5-kg above the initial weight during the carbohydrate-loading phase. During the race, weight fell by about 2-kg reaching levels only slightly lower than they had been before the experiment began. After the race, body weight continued to rise for two days, reaching levels higher than the pre-race value before falling for the last two days of the experiment.

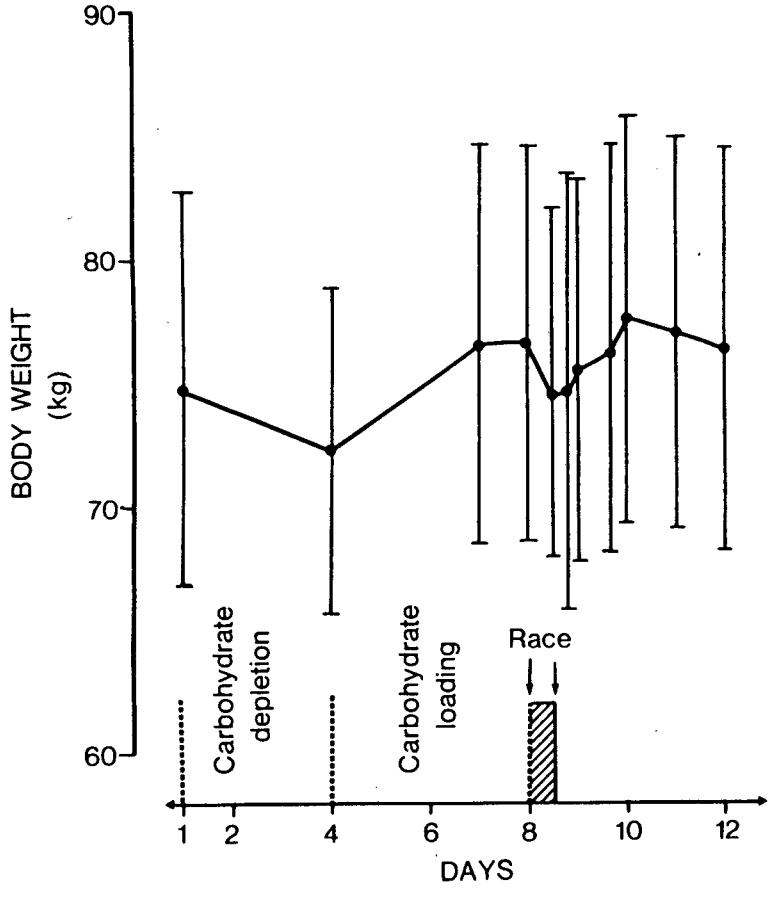


FIGURE 14 : Body weight fell during both the carbohydrate-depletion phase of the diet and the race itself, and rose during carbohydrate-loading and again for the first two days after the race.

(7) Post-Race Muscle Soreness

The subjective assessment of post-race muscle soreness in the quadriceps and calf muscles is shown in Figure 15. The muscle soreness gradually decreased over the four days and had subsided by the end of the fourth day.

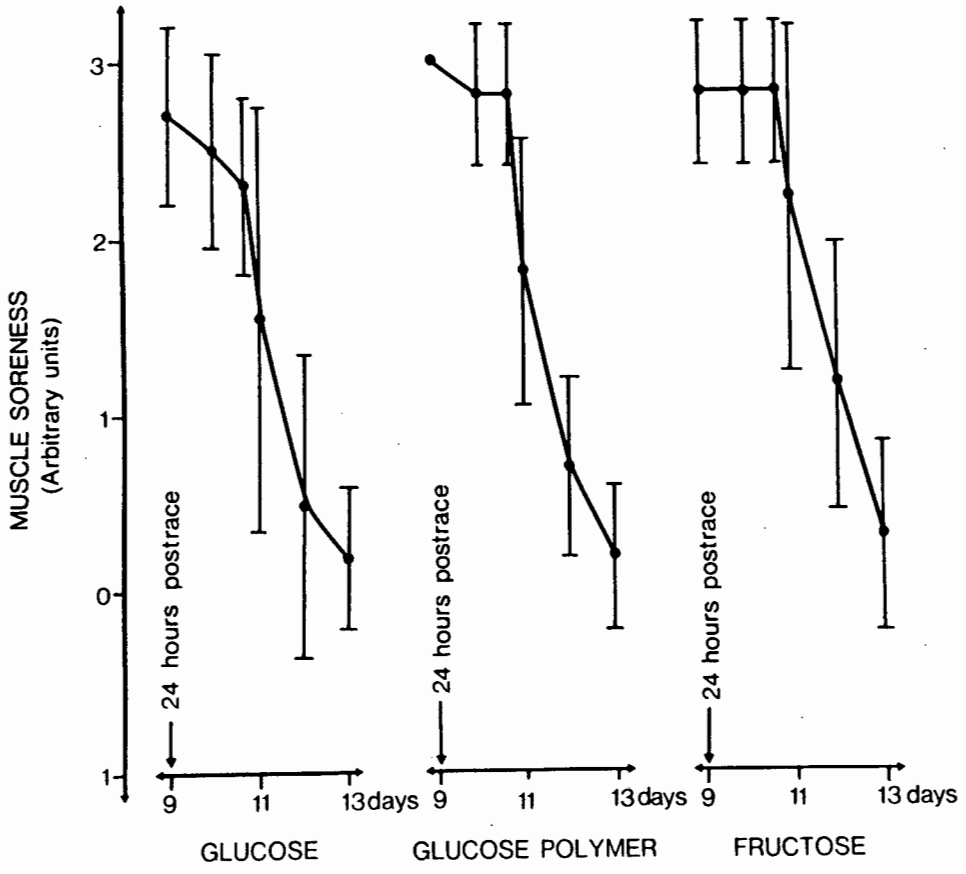


FIGURE 15 : Muscle soreness was maximal 24-hours after the race (Day 9) and had almost cleared 4 days later (Day 13).

(8) Subjective Assessment of Physical State

Figure 16 shows that the type of carbohydrate in the ingested fluid did not influence the subjective feelings the runners had of their physical state, since there was no significant difference between the groups at any point in the race. Subjective ratings fell linearly with time.

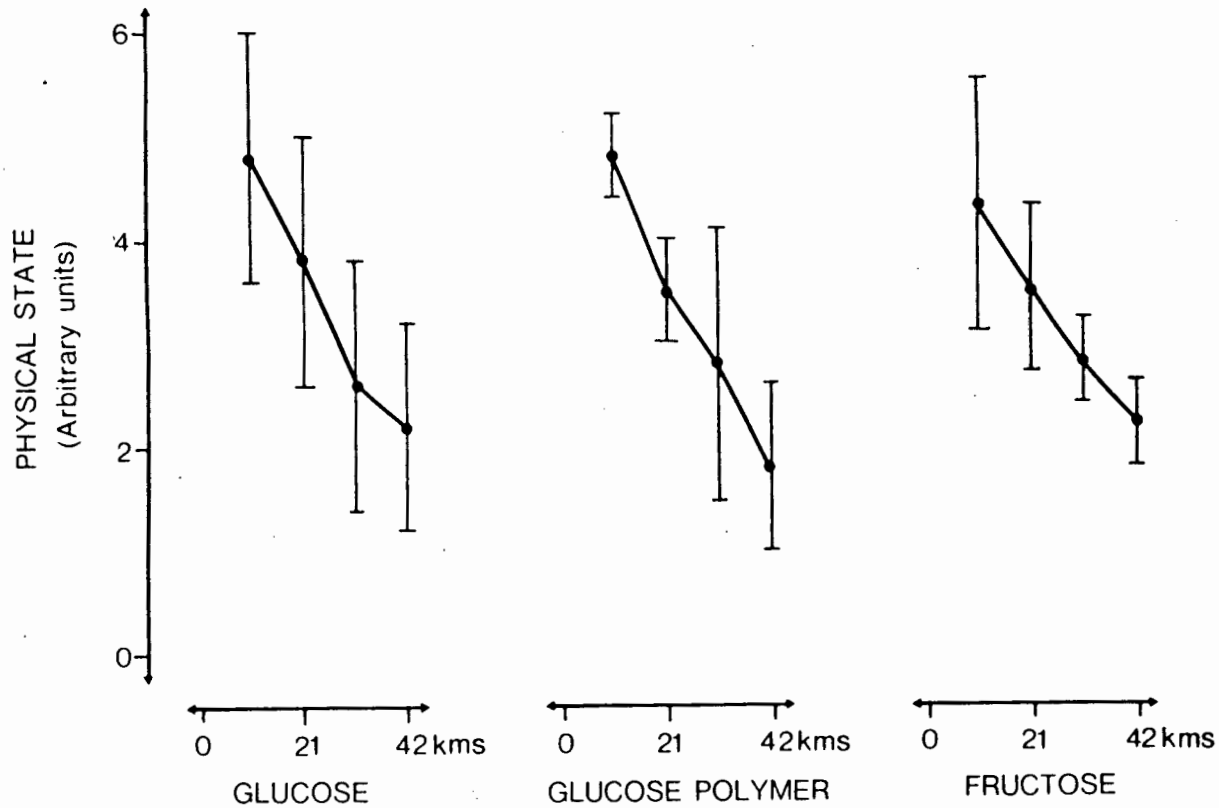


FIGURE 16 : The type of carbohydrate ingested during the race did not influence the subjective assessment of physical state. Note also that the fall in subjectively assessed physical state occurred despite the maintenance of normal blood glucose levels (Figure 10).

(9) Post-Race Rectal Temperatures

Post-race rectal temperatures were no different between the groups and were 38,7 ± 0,4°C; 38,5 ± 0,6°C and 38,8 ± 0,6°C in the glucose, glucose polymer and fructose groups respectively.

CHAPTER 5

DISCUSSION

5.1 BLOOD GLUCOSE LEVELS DURING MARATHON AND ULTRA-MARATHON RUNNING

The principle finding of these studies is that blood glucose levels in the majority of trained runners who have free access to carbohydrate-containing drinks, are elevated after competitive marathon and ultra-marathon races (Figures 2, 3, 4 and 10) confirming findings in some recent studies (McKechnie et al, 1982; Noakes and Carter, 1976). This would suggest that hypoglycaemia is uncommon during these races and is therefore likely to be an uncommon cause of fatigue.

Indeed, the characteristic response to all these races was hyperglycaemia at the end of the race. However, the third study in which blood samples were drawn three times during the marathon race as well as immediately after the race, indicates that the blood glucose levels rise precipitously immediately on cessation of exercise (Figure 10). This is probably because the elevated rate of hepatic glucose production continues into the immediate post-exercise period when muscle glucose uptake suddenly falls. Thus it is likely that the true incidence of hypoglycaemia during marathon and ultra-marathon races can only be determined if blood glucose levels are measured during the race when rates of hepatic glucose production may fall below rates of muscle

glucose uptake.

We found that blood glucose levels fell from 10- to 32-km in the race, despite an adequate carbohydrate intake in two of three study groups (Figure 10). Thus it is likely that blood glucose levels were still falling at the end of the race and may well have reached hypoglycaemic levels during exercise, if the race had been longer.

Other points that would have caused us to underestimate the true incidence of hypoglycaemia in these runners should also be considered.

First, in the initial study, blood glucose levels were measured only in volunteer runners who actually finished each race. It is possible that severely hypoglycaemic athletes may either not have finished the race, or have been too ill or too weak to participate in the study.

Second, after the 88-km Comrades Marathon, all runners with blood glucose levels at or below 4 mmoles ℓ^{-1} complained of hypoglycaemic symptoms. In resting, untrained subjects' blood glucose levels below 2,5 mmoles ℓ^{-1} are considered to cause hypoglycaemic symptoms. Thus, it is possible that trained persons are, like insulin dependant diabetics at rest (De Lorenzo et al, 1980), habituated to elevated

blood glucose levels during exercise, making them more sensitive to small changes in blood glucose levels than are untrained persons. Indeed, other workers (Astrand, 1967; Hedman, 1957 and Pruett, 1970) have noted hypoglycaemic symptoms in endurance trained athletes whose blood glucose levels were not markedly depressed. In a more recent study, Coyle et al (1983) found that the symptoms of hypoglycaemia in one subject exercising at 70 - 79% $\dot{V}O_2$ max on a bicycle ergometer, to the point of fatigue, were disproportionately severe compared with the mild decline in his blood glucose levels, further suggesting that the trained athlete is hypersensitive to a relatively mild decrease in blood glucose levels.

Thirdly, and alternatively, it is possible that blood glucose levels in our athletes who complained of hypoglycaemic symptoms and who had post-race blood glucose levels of about 4 mmoles ℓ^{-1}, may have had much lower blood glucose levels when they were running.

The results of the 15-minute trial done at the end of the 1981 42-km standard marathon (Figure 2) led us to conclude that blood samples drawn within 15-minutes of completion of the race, would be representative of blood glucose levels at the finish of the race. However, the third study showed that on cessation of exercise, blood glucose levels rise very rapidly and that values measured during the latter

stage of the race were lower by as much as 2,5 mmoles ℓ^{-1} than they were at the finish (Figure 10). It is probable that this rise in blood glucose levels immediately on the cessation of exercise is due to the sudden decrease in muscle uptake of blood glucose without an equivalent fall in the rate of glucose release by the liver. Thus the blood glucose levels of 4,0 mmoles ℓ^{-1} measured in symptomatic runners would have been nearer 1,5 - 2,5 mmoles ℓ^{-1} during the race. Such low levels would adequately have explained their hypoglycaemic symptoms during the race.

Finally, it is possible that the standard marathon distance (42-km) used in the third study was too short for carbohydrate loaded athletes to develop hypoglycaemia and therefore the effect of the three different carbohydrate-containing drinks to become apparent.

Recent studies (Ahlborg et al, 1982) show that the rate of glucose uptake by muscle during prolonged exercise at 60% $\dot{V}O_2$ max is about 36g hr^{-1}. Of this 36g, about 10g hr^{-1} is likely to come from gluconeogenesis whereas the other 26g from hepatic glycogenolysis. Thus, during prolonged exercise like marathon running, the liver is likely to become depleted at a rate of at least 26g hr^{-1}, and probably faster because standard marathon races are run at closer to 85% $\dot{V}O_2$ max (Davies and Thompson, 1979).

If we assume that the carbohydrate-loaded liver contains about 150gm of glycogen (Hultman, 1978), and that the rate of liver glycogen depletion at 85% VO_2 max is 40g hr^{-1}, it can be calculated that in the absence of an exogenous glucose supply, liver glycogen stores will become depleted after (150 ÷ 40 hours) or three and three quarter hours of running. In our athletes the ingestion of about 40g hr^{-1} of carbohydrate by the fructose and glucose polymer groups would, if completely absorbed, have prevented their ever becoming hypoglycaemic as it would have exactly replaced that glucose coming in from hepatic glycogenolysis, thereby preventing liver glycogen depletion.

On the other hand the carbohydrate intake of the glucose group was only about 10g hr^{-1}, thus their liver glycogen stores would have been depleting at a rate of about 30g hr^{-1} and they would likely have become hypoglycaemic in races lasting longer than 5 hours.

* These arguements suggest that in carbohydrate-loaded runners hypoglycaemia is likely to occur only when they have ingested no exogenous carbohydrate during exercise and have exercised for more than three and three quarter hours.

The finding of a low incidence of hypoglycaemia in these runners compared with the early studies of Levine et al

(1924) can probably be explained on the basis of three factors : first, that the athletes were "carbohydrate-loaded"; second, that they were well trained; and third, that they ingested carbohydrate during exercise. In such runners, hypoglycaemia is likely to occur only when they run for more than three and three quater hours.

(i) Carbohydrate Loading

This practice which has become common only since the early 1970's was unknown to the early marathon runners. However, Levine and co-workers (1924) did report that when runners ate a high carbohydrate diet the day before the race, and ingested glucose during the run, those that had previously become hypoglycaemic during the Boston Marathon, finished the next year's race "stronger" and had higher post-race blood glucose levels (Gordon et al, 1925).

(ii) Carbohydrate Ingestion During Exercise

In contrast to the former practice (with the exception of Levine et al's (1924) runners), modern marathoners regularly ingest carbohydrate during marathon and longer races. All the subjects in our three studies had free access to liberal amounts of carbohydrate drinks at least every two to four kilometers during the races.

Results obtained in other studies have indicated that carbohydrate ingestion during prolonged exercise at 70 - 80% $\dot{V}O_2$ max may postpone the onset of fatigue and improve performance in trained athletes (Ahlborg and Felig, 1976; Bagby et al, 1978; Bergström and Hultman, 1967; Boje, 1939; Christensen and Hansen, 1939b; Costill, 1974; Coyle et al, 1983; Gordon et al, 1925). Furthermore, cross-country skiers have always ingested considerable carbohydrate during long distance ski races (Astrand, 1967). In addition, the athlete who finished first in the 1981, 1982, 1983 and 1984 Comrades Marathon and the 1981, 1982 and 1983 London to Brighton Race, discovered independently that a high carbohydrate intake during these races has significantly aided his performance by preventing symptoms which he recognises to be due to hypoglycaemia.

The mechanism by which ingested carbohydrates aid performance during prolonged exercise would seem to be through correcting the hypoglycaemia, since our third study showed that adequate carbohydrate ingestion during competition did not influence the rate of muscle glycogen depletion so that muscle glycogen levels were markedly depleted at the end of the standard 42-km marathon despite carbohydrate ingestion during exercise.

We found however, that although the glucose group consumed, on average, three and a half times less carbohydrate during the race than did the other two groups (Table 3), their blood glucose levels were not different from the other groups. However, as discussed earlier, it is likely that the inability of the glucose solution to prevent hypoglycaemia would only have become apparent had the race lasted longer than 5-hours.

It is of interest that the type of carbohydrate ingested during exercise had no effect on free fatty acid and insulin (Figure 12) levels. This was expected for the fructose and glucose polymer solutions, the ingestion of which do not induce a marked insulin response. The failure of glucose ingestion to stimulate an insulin response during exercise is compatible with findings of other workers (Galbo, 1983).

(iii) It has been suggested that running ability may influence post-race blood biochemical parameters as previous workers have found that post-race blood glucose levels are highest in the fastest runners (Maron et al, 1975; Scheele et al, 1979).

In our first study we found that the fastest runners had significantly lower blood free fatty acid levels

after the 42- and 56-km races (Figure 5) with a tendency for higher glucose (Figure 4) and insulin levels (Figure 6). No such difference was evident in the 88-km race nor in the third study.

5.2 EFFECTS OF RUNNING ABILITY ON THE EXTENT OF BODY CARBOHYDRATE DEPLETION DURING MARATHON RUNNING

The state of the body's carbohydrate stores strongly influences the blood fuel/hormonal response during exercise (Koeslag et al, 1980, 1982; Rennie and Johnson, 1974b; Williams et al, 1976). Thus the lower free fatty acid and higher glucose and insulin concentrations in the fastest runners is compatible with their being less carbohydrate depleted than the slower runners, at the end of the race. This is contrary to the generally held belief that the superiority of the elite marathon runners is due to their greater capacity for fat metabolism, when they are carbohydrate depleted (Gollnick, 1977).

In the second study we found that the Respiratory Quotient values during, and the muscle glycogen values after, marathon races (Table 2) were higher in the better runners, thus providing further evidence for the hypothesis that the better runners were less carbohydrate depleted.

However, the final study showed that muscle glycogen depletion was not different between runners of differing abilities (Table 4) although the faster runners tended to have higher pre-race and two day post-race muscle glycogen levels suggesting that fitness may increase the capacity to both store and resynthesize muscle glycogen.

5.3 MUSCLE GLYCOGEN AND EXHAUSTION

Our third study showed that the ingestion of different carbohydrate drinks had no effect on the extent of muscle glycogen depletion during the marathon race. Of special interest was the finding that the group ingesting fructose was as equally carbohydrate depleted as were the other groups. This finding is contrary to that of Levine et al (1983) who reported that in comparison to glucose or water ingestion, fructose ingestion spared muscle glycogen during a 30-minute treadmill run at 75% $\dot{V}O_2$ max. This finding was interpreted to indicate that fructose provided a source of slowly releasable glucose from the liver which stimulated glucose uptake by muscles and therefore spared muscle glycogen (Sherman et al, 1984).

The discrepancy between our respective results would be that our subjects ran for much longer but at a lesser work

intensity than did those of Levine et al (1983). It would seem that Levine et al's (1983) results are not applicable to marathon runners.

CHAPTER 6

OVERALL SUMMARY AND CONCLUSIONS

The principal findings from these studies are the following :

(1) Hypoglycaemia occurs uncommonly in marathon and ultra-marathon runners who have carbohydrate loaded.

(2) Hypoglycaemic symptoms were reported by runners whose blood glucose levels were less than 4 mmoles ℓ^{-1} after the 88-km race suggesting perhaps that trained runners are habituated to high blood glucose levels during exercise and become sensitive to even quite small reductions in these levels.

(3) Alternatively, as blood glucose levels rise steeply upon cessation of exercise and may be 1 - 2 mmoles ℓ^{-1} higher than values measured during exercise, the blood glucose levels of these symptomatic runners during exercise may well have been in the range of 1,5 - 2,5 mmoles ℓ^{-1}, values which would certainly explain their hypoglycaemic symptoms.

(4) Since blood glucose levels rise steeply on cessation of exercise, it follows that studies to determine the true incidence of hypoglycaemia in marathon and ultra-marathon races would need to measure blood glucose levels near the end of such races but before the runners had stopped running.

(5) Glycogen loading before, and ingesting water only during a 42-km marathon race would appear to be sufficient to prevent hypoglycaemia since it can be calculated that liver glycogen stores probably last for up to four hours during such exercise. Thus carbohydrate-loading before a 42-km race would appear to be more important in the prevention of hypoglycaemia than carbohydrate ingestion during competition.

(6) The finding in the final study that post-race muscle glycogen levels were very low whereas blood glucose levels were normal or elevated, indicates that muscle glycogen depletion would seem to be the more likely explanation of the fatigue experienced in such races.

However, whereas hypoglycaemia is unlikely to be a cause of fatigue in the 42-km standard marathon, theoretical considerations indicate that this is more likely to become a limiting factor in exercise lasting longer than 4-hours.

When liver glycogen stores are depleted, hypoglycaemia will only be prevented if 40g of carbohydrate reaches the hepatic circulation every hour. This study showed that a 2% glucose solution is unable to provide such a high carbohydrate load, whereas runners drinking the

8% fructose and glucose polymer solutions did indeed ingest 40 or more grams of glucose per hour during the race.

(7) The type of carbohydrate ingested did not influence the response of serum insulin or free fatty acid levels to exercise.

(8) Future studies should determine the effects of the different carbohydrate solutions on blood glucose levels during longer races when hypoglycaemia is more likely to occur.

Research should also be conducted to determine :

(i) The exact rate of liver glycogen depletion during exercise at between 70 and 85% $\dot{V}O_2$ max, and

(ii) the optimum carbohydrate solution that will provide an equivalent amount of carbohydrate to the hepatic circulation every hour during prolonged exercise.

APPENDICES

APPENDIX 1

METABOLIC COMPUTATIONS FOR THE DETERMINATION OF RESPIRATORY QUOTIENT

$\dot{V}e$ = expired air volume per minute (read off by research assistant from spirometer)

$\dot{V}O_2$ = volume of oxygen consumed per minute

$\dot{V}CO_2$ = volume of carbon dioxide output per minute

RQ = Respiratory Quotient

$\%O_2$ = percentage O_2 in sample bag

$\%CO_2$ = percentage CO_2 in sample bag x correction factor (see Figure 1)

1. Standardisation of $\dot{V}e$ to STPD

Standard temperature = 273°A or 0°C

Standard pressure = 760 mmHg

Dry conditions = no water vapour

$$\dot{V}e_{STPD} = \dot{V}e \left(\frac{273}{273 + T°C}\right)\left(\frac{P_B - P_{H_2O}}{760}\right)$$

P_B = Barometric pressure of experimental conditions (i.e. of the day)

P_{H_2O} = Vapour pressure in moist air at the temperature of the experimental conditions (i.e. at the time of sampling)

2. Calculation of O_2 Consumption ($\dot{V}O_2$)

$$\dot{V}O_2 = \dot{V}e_{STPD}\left[(\%N_{2E} \times 0,265) - \%O_{2E} \right]$$

$$\%N_{2E} = 100 - (\%O_{2E} + \%CO_{2E})$$

3. Calculation of CO_2 uptake ($\dot{V}CO_2$)

$$\dot{V}CO_2 = \dot{V}e_{STPD} (\%CO_{2E} - \%CO_{2I})$$

$$= \dot{V}e_{STPD} (\%CO_{2E} - 0,03\%)$$

4. Calculation of Respiratory Quotient (RQ)

$$RQ = \frac{\dot{V}CO_2}{\dot{V}O_2}$$

APPENDIX 2

METHOD FOR THE MEASUREMENT OF MUSCLE GLYCOGEN

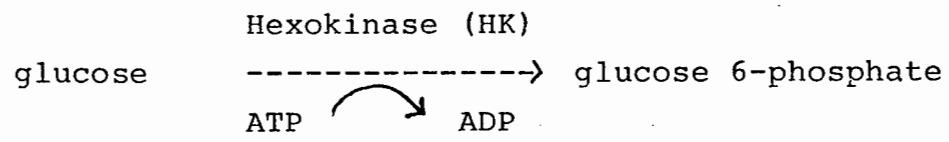
(Adapted from Passonneau and Lauderdale (1974)

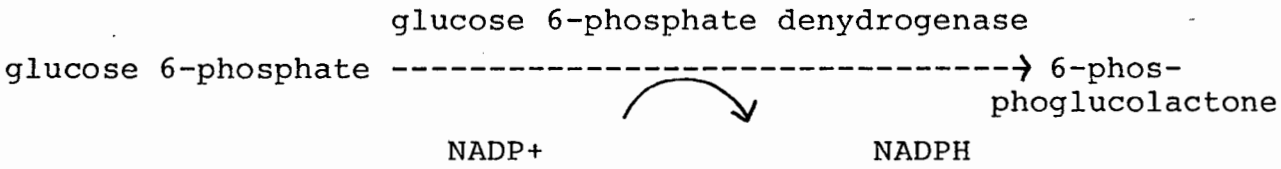
1. Sample Preparation

The tissue which is stored in an eppendorff tube in a freezer at -80°C, is placed into a metal mortar which has been immersed in an ice bucket containing liquid nitrogen (N_2), and powdered with the use of a metal pestle. The sample is placed into a pre-weighed plastic culture tube containing 1 ml of KOH (40%). The tube is then re-weighed to calculate the mass of the tissue.

2. Principle

Glycogen is the macromolecular storage form of carbohydrates in the human body. It consists of alpha 1-4 and alpha 1-6 linkages of glucose residues. Acid hydrolysis of this macromolecule yields free glucose residues which can be assayed through the following series of reactions :





The appearance of NADPH yields an observable method to quantify the glucose residues.

3. Reagents

40% KOH

95% Ethanol

2N HCL

Tris buffer (pH 7,5) 0,2M

2N NADH

ATP 20mM

Mg Cl_2 $6H_2O$ 1M

1% NADP (TPN)

Boehringer ready mixed HK G 6 PDH suspension (Cat. No. 127825)

Assay Procedure

The tubes are placed into an oven at 100°C for 30 minutes. Four ml of percent ethanol is added to each tube and the content of the tubes is gently mixed on a vortex mixer. The tubes are replaced into the boiling water for about 30 seconds

until bubbles emerge (ethanol precipitates the glycogen).

The tubes are left in a refrigerator overnight after which they are spun at 40°C for 10 minutes at 5 000 rpm. The supernatant is removed using a venturi pump system and 5 ml of 95 percent ethanol is added to each tube. The tubes are mixed on a vortex and respun for 10 minutes at 5 000 rpm at 40°C. The supernatant is once again removed (this process of washing, cleans the glycogen present and rids the pellet of remaining KOH and alcohol which could interfere with the assay.

One ml of 2 N HCl is added in order to hydrolyse the glycogen to glucose and the solution is placed into the water bath at 100°C for 3 hours.

0,2 M of Tris buffer is added to bring the pH of the solution up to 7,5. 200ml of the resultant solution is placed into a cuvette and 2,8ml of the reagent mix is added. The absorption is read at 340 nm on a Beckman Spectrophotometer (Model 35).

Reagent Mixture	Per Cuvette
Tris buffer	1 ml
ATP 20 mM	0,1 ml
Mg Cl_2 $6H_2O$ 1M	0,1 ml
NADP 1% (TPN)	0,1 ml
Distilled water	1,5 ml
	2,8 ml

200ml of reagent mix is added to 200ml of µℓ glucose standard and 20 µℓ of distilled water to be used as blank and their absorptions at 340 nm recorded.

10 µℓ of HK/GPDH suspension is added to each cuvette and after standing for 30 minutes, the absorption at 340 nm is again read.

Calculation

The glycogen content (in mmole glucosyl $\mu.kg^{-1}$wet mass) is calculated using the following formula :

$$\frac{\Delta OD - \Delta OD_{blank}}{0,414} \times \frac{3 \text{ (vol. in cuvette)}}{\text{wet mass (in mg)}}$$

The recovery was calculated by :

$$\frac{\text{Standard reading} \times 100}{0,414}$$

The recovery during the experiments was greater than 90 percent.

APPENDIX 3

METHOD FOR THE MEASUREMENT OF FREE FATTY ACIDS (FFA)

Principle

Lipids exist in the serum in the most part as neutral fats, phospholipids, cholesterol esters and free cholesterol. The neutral fats, or triglycerides, may be relieved of their fatty acid component by the action of lipases. The free fatty acids concentration is, however, normally very low.

Thus, removing the free fatty acids from their aqueous environment to a solvent in which they may be colorimetrically quantified requires a rather sensitive procedure.

In order to best extract the free fatty acids, methanol is mixed in low concentration with the highly non-polar solvents, chloroform and heptane. This solvent can then be used as an environment where free fatty acids can form copper salts when added to a copper-Tea solution.

The copper salts of the free fatty acids can then form a coloured product upon reacting with the Na salt of diethyl-dithiocarbamic acid (DDC).

$FFA + CU^{++} = FFA - CU : FFA - CU + DDC =$ coloured product

Reagents

(1) Nile blue indicator (stock solution)

100mg Nile blue in 100ml CO_2-free water (bubble N_2 through distilled water). Filter and dilute to 500ml (i.e. 5X), wash with iso-octane until washings are colourless.

(2) Working Standard (of Nile blue)

Dilute stock Std. 5X with Absolute alcohol FRESHLY BEFORE USE. Check pH = 8,0 (add 0,2ml of 0,1N NaOH if necessary).

(3) Dole's mixture

	Parts	vol/1	vol/21
ISOPROPYL ALCOHOL	40 x 5 = 200	800	1600ml
Heptane	10 x 5 = 20	200	400
1N H_2SO_4	1 x 5 = 5	20	40

(4) 0,02N NaOH

Use MERCK Titrisol.

(5) Std. Palmitic Acid (mw 256.43)

0.1536g/100ml heptane (6000µeq/l)

1µeq/l working std. dilute 6 x with heptane.

500µl std. + 3000µl hept.

Sample Preparation

(1) Spin down blood as soon as possible @ 2000rpm for 10 minutes @ 4°C. (Lipase broken down triglycerides to free fatty acids.)

(2) Remove serum and store deep frozen until needed.

(3) Defrost and keep on ice.

Method

Pipette into tubes with ground stoppers (glass).

	Blank	Std.	Test
DDW ml	0,6ml	-	-
working std.	-	0,6ml	-
serum	-	-	0,6ml
Doles reagent	3,0ml	3,0ml	3,0ml
SHAKE FOR 3 MINUTES			
Heptane	1,8ml	1,2ml	1,8ml
DDW	1,2ml	1,8ml	1,2ml
SHAKE FOR 4 MINUTES			

Allow phases to separate (±5mins or overnight in 'fridge or centrifuged).

Titrate 1ml of SNF (i.e. top of phase of B1, Std. and test

with 1ml Nile blue indicator against 0,02N NaOH (or 0,01N) while bubbling N_2 through tube. (Try to keep titration rate constant throughout.)

Calculation

1st Way

51ml Doles contains 10ml heptane

3ml " " " " $\frac{3}{51}$ x 10 = 0,59ml heptane. (i.e. 0,6ml)

Added to tube 1,8ml heptane

Total heptane = 2,4ml

Vol. NaOH x strength NaOH = vol. x strength FFA

y x 0,02 = $\frac{0,6}{2,4}$ x strength FFA (20eq/l = 0,02µeq/l)

Therefore, strength of FFA = $\frac{y \times 0,02 \times 2,4}{0,6}$

= $8 \times 10^4 \times y$ µeq/l

1 unit on micrometer syringe = 0,0002ml

Therefore, FFA = $8 \times 10^4 \times 0,0002 \times y$

= 16 x y µeq/l

where "y" is number of units on syringe.

2nd Way

Stock std. = 6000 µeq/l (i.e. 6µeq/l)

Working = 6 x dil.

= 1µeq/l

= 1000 µeq/l $\frac{(I - B_1)}{(Std - B_1)} \times 1000$ = µeq/l

APPENDIX 4

METHOD FOR THE MEASURMENT OF SERUM ALANINE

Principle

1-alanine + ∝ketoglutarate + L-glutamate pyruvate; pyruvate + NADH → L-lactate + NAD

The decrease in the absorbance (@ 340 nm) is used to calculate the amount of alanine reacted.

Reagents

1. 0,055M Tris-HCL Ph 8,1 mw 121,14
   (1,67 g/250 ml)

2. 0,05 mm ∝ Kg mw 168,1
   (2,1 mg/25 ml)

3. 20 mM NADH
   (14,19 mg/ml)

4. LDH - dilute (25 mg/5 ml) strong LDH
   50x to give 1 µg/ml
   i.e. 10 µl in 0,5ml

5. 8µ/ml GIPT (glutamic pyruvic trasaminase) (SIGMA No. G-8255 200µ/4,7 mg)

   1 mg/ml (Sigma) = 53 u - 1 mg solid - 1 ml in bottle

   4,6 mg solid - 4,6 ml ⟶ 46 tests

   Calculate final volume needed.

   Use 100 µl 1100 µl ± 5U

6. Alanine Standard. (DL - alanine mw 89,09)

   0,5 MM 44,6 mg/10 ml

   Working std. dilute 100x

   1mm alanine = 1 µmole/ml

   0,05 MM 0,05 µmole/ml

   = 50 µmole/ml

   Stock alanine (new 89,09)

   0,5 mm = 44,55 µg/ml

   = 445,5 µg/10 ml

   = 0,4455 mg/10ml

   Make stock 100x stronger therefore 44,55 mg/10 ml.

   Make working std. from stock dilute 1/100 - then use 100 µl of working std. ⟶ 1000 µl = 0,05 mm = 50 nmole/ml

100 nmole at 1 ml 1cm = ΔA340 = 0,620

50 nmole at 1 ml 1cm = Δ " = 0,310

50 nmole at 1,1 ml 1cm = " = 0,279

Methods

1. Deproteinise and neutralise as for glycerol.

2. Add into tubes :

TRIS - HCL buffer	900 µl
∝KG	100 µl
NADH	10 µl
LDM	10 µl
Sample/std.	100 µl
Blank ($d.H_2O$)	100 µl

3. Mix and allow to stand for a few minutes.

4. Add 100 µl GIPT to each tube. Mix - allow to stand for 45 mins at room temperature. (Switch on spectrophotometer)

5. Then read @ A340 against water blank (i.e. zero - on your blank)

Calculation

$$\frac{\Delta E(abs)}{6,22} \times \frac{1120}{100} \times \frac{1500}{500} \times \frac{1500}{1} = \frac{F}{1000}$$

(to mℓ)

= F x 8,1 µmol/ml

(std. blank)

APPENDIX 5

METHOD FOR THE MEASURMENT OF $\%O_2$ AND $\%CO_2$ CONTAINED IN SAMPLE BAGS

In order to determine the Respiratory Quotient, the oxygen and carbon-dioxide content of the expiratory gas samples collected had to be determined. This was done using the Electronics Inc. S3A and Morgan 901 MK II gas analysers for oxygen and carbon-dioxide respectively. These were calibrated using room air and three other known gas mixtures of differing oxygen and carbon-dioxide concentrations. The recorder used was a Beckman RR-2 recorder.

APPENDIX 6A

BLOOD GLUCOSE LEVELS (mmoles ℓ^{-1}) IN INDIVIDUALS OF STUDY 3

Glucose Group

Athletes	Start	10-km	21-km	32-km	Finish
J.B.	3,5	6,2	6,9	5,0	4,7
D.P.	4,9	6,2	6,2	5,3	6,3
J.M.	4,7	5,5	5,0	5,0	8,0
T.H.	4,9	6,2	5,2	4,5	6,2
D.B.	5,3	5,5	4,8	5,4	6,1
D.S.	4,4	4,1	3,7	4,3	4,5
Mean ± SD	4,6 ± 0,6	5,6 ± 0,8	5,3 ± 0,1	4,9 ± 0,4	6,0 ± 0,1

Glucose Polymer Group

N.D.	5,0	7,4	6,4	5,0	5,8
P.P.	3,8	5,0	4,0	2,6	5,7
D.C.	4,6	7,5	5,8	6,3	7,0
P.Ph.	4,9	5,8	4,3	4,9	4,5
A.L.	4,4	6,8	4,5	6,3	5,6
A.R.	4,1	6,9	4,9	5,1	7,0
Mean ± SD	4,5 ± 0,5	6,6 ± 0,9	5,0 ± 0,9	5,0 ± 0,1	5,9 ± 0,95

Fructose Group

I.D.	4,9	7,1	5,9	6,0	5,4
J.S.	4,5	6,2	5,0	5,5	6,6
G.W.	4,1	4,7	6,3	5,2	6,2
Q.V.E.	4,3	4,4	4,4	4,1	4,4
G.H.D.	5,0	5,6	5,1	4,4	6,0
E.L.	4,5	10,9	4,6	4,9	7,8
Mean ± SD	4,6 ± 0,3	6,5 ± 0,2	5,2 ± 0,7	5,0 ± 0,7	6,1 ± 0,1

APPENDIX 6B

MUSCLE GLYCOGEN LEVELS (mM Kg^{-1} MUSCLE) IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Pre-Race	Post-Race	2-Days Post-Race
J.B.	247,2	20,0	270,5
D.P.	265,8	55,1	217,6
J.M.	283,8	37,1	240,5
T.H.	237,6	57,6	199,9
D.B.	201,2	63,0	157,6
D.S.	196,3	109,5	198,1
Mean ± SD	238,65 ±34,77	57,05 ±30,20	214,03 ±38,83

Glucose Polymer Group

N.D.	236,8	88,8	145,1
P.P.	164,7	29,2	73,7
D.C.	310,6	96,3	214,3
P.Ph.	243,3	37,2	221,2
A.L.	168,3	50,2	146,6
A.R.	137,7	18,1	155,4
Mean ± SD	210,23 ±64,75	53,30 ±32,24	159,38 ±53,97

Fructose Group

I.D.	246,5	18,1	165,6
J.S.	335,1	35,8	203,2
G.W.	83,4	20,2	52,9
Q.V.E.	363,1	128,0	201,9
G.H.D.	263,6	32,2	91,5
E.L.	161,8	22,8	152,5
Mean ± SD	242,25 ±105,27	42,85 ±42,29	144,60 ±60,74
O/L Mean O/L SD	230,38 ±71,17	51,07 ±33,74	172,67 ±57,70

APPENDIX 6C

PLASMA FREE FATTY ACID LEVELS (mmoles ℓ^{-1})

IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Start	10-km	21-km	32-km	42-km
J.B.	390,5	360,4	317,2	577,0	1149,4
D.P.	298,4	696,7	680,4	817,7	1444,0
J.M.	285,7	370,0	359,3	662,3	675,6
T.H.	361,8	433,1	723,6	1083,2	1758,7
D.B.	357,9	286,0	501,8	852,8	1446,1
D.S.	328,1	259,5	507,0	1075,1	1745,5
Mean ± SD	337,1 ±40,29	404,3 ±158,71	514,9 ±164,00	844,7 ±207,70	1369,9 ±408,46

Glucose Polymer Group

N.D.	253,9	337,7	469,8	533,0	962,5
P.P.	280,0	476,0	436,5	724,3	2238,6
D.C.	290,0	355,1	429,7	391,9	765,0
P.Ph.	304,6	570,0	633,0	696,0	1213,1
A.L.	150,1	307,1	505,0	678,3	992,0
A.R.	406,5	444,0	761,4	1022,4	2042,2
Mean ± SD	280,9 ±82,77	415,0 ±99,79	539,2 ±131,68	674,3 ±211,60	1368,9 ±617,40

Fructose Group

I.D.	300,0	562,5	528,2	442,5	490,6
J.S.	225,7	280,7	484,6	691,0	897,4
G.W.	288,0	717,9	1120,4	1020,6	1504,1
Q.V.E.	204,4	255,2	475,1	674,0	1086,1
G.H.D.	292,7	422,0	444,6	462,3	1063,8
E.L.	366,2	331,7	493,5	655,2	1235,1
Mean ± SD	279,5 ±57,82	428,3 ±180,55	591,1 ±260,73	657,6 ±208,6	1046,2 ±340,03

APPENDIX 6D

PLASMA INSULIN LEVELS ($\mu U\ m\ell^{-1}$) IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Start	10-km	21-km	32-km	42-km
J.B.	8,3	8,0	8,0	8,0	8,0
D.P.	22,9	22,6	16,8	16,0	22,2
J.M.	8,0	10,1	8,0	8,0	11,5
T.H.	14,0	12,8	11,0	10,1	8,3
D.B.	13,6	11,0	8,0	8,0	16,8
D.S.	14,8	8,0	8,0	8,0	8,0
Mean ± SD	13,60 ±5,43	12,08 ±5,47	9,97 ±3,56	9,68 ±3,21	12,47 ±5,86

Glucose Polymer Group

N.D.	8,0	11,0	8,0	8,7	8,0
P.P.	8,0	8,0	8,0	8,0	8,0
D.C.	8,0	8,0	9,2	8,0	8,0
P.Ph.	8,0	8,3	10,1	12,3	8,0
A.L.	30,5	16,1	8,3	12,8	21,8
A.R.	15,3	11,4	17,6	11,9	11,5
Mean ± SD	12,97 ±9,07	10,47 ±3,15	10,20 ±3,72	10,28 ±2,28	10,88 ±5,33

Fructose Group

I.D.	8,0	16,0	18,8	8,3	8,0
J.S.	14,0	18,8	11,0	25,9	25,1
G.W.	8,0	8,0	8,0	8,0	13,2
Q.V.E.	8,0	8,0	8,0	8,0	8,3
G.H.D.	9,2	9,2	8,0	8,0	14,0
E.L.	8,0	21,1	8,0	20,3	11,9
Mean ± SD	9,20 ±2,40	13,52 ±5,85	10,30 ±4,33	13,08 ±7,96	13,42 ±6,24
O/L Mean O/L SD	11,92 ±6,21	12,02 ±4,84	10,16 ±3,65	11,02 ±5,05	12,26 ±5,63

APPENDIX 6E

BODY WEIGHT (kg) IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Day 1	Day 4	Day 7	Pre-Race	Post-Race
J.B.	61,2	59,7	61,6	61,5	60,4
D.P.	70,9	68,6	70,8	71,5	70,4
J.M.	70,8		73,0	73,2	70,6
T.H.		70,8	73,8	71,4	70,8
D.B.	77,5	77,9	79,6	79,3	77,6
D.S.	74,3	73,6	74,7	75,1	72,6

Glucose Polymer Group

N.D.	69,2	68,0	70,0	70,5	69,1
P.P.	69,6	67,0	71,1	71,7	69,3
D.C.	80,3		83,3	83,1	81,8
P.Ph.	82,1		84,2	82,6	79,6
A.L.	71,0	71,4	72,7	72,7	70,4
A.R.	92,5		94,5	95,5	92,0

Fructose Group

I.D.	68,4	67,9	71,0	70,6	68,6
J.S.	84,7	82,6	84,5	86,0	84,3
G.W.		80,7	81,1	80,6	79,2
Q.V.E.	73,7	75,7	78,8	78,8	75,6
G.H.D.	84,0	81,0	86,1	86,3	83,0
E.L.	66,9	66,4	68,0	68,3	66,9
Mean ± SD	74,82 ±8,09	72,24 ±6,64	76,60 ±8,00	76,59 ±8,12	74,57 ±7,67

APPENDIX 6E (Cont)

BODY WEIGHT (kg) IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Day 8	Day 9 (am)	Day 9 (pm)	Day 10	Day 11	Day 12
J.B.	59,1	61,6	61,3	61,8	61,7	61,2
D.P.	70,0	70,2	70,6	72,2	72,2	71,5
J.M.	71,9	73,4	73,8	75,5	74,2	72,4
T.H.	70,9	72,3	73,1	73,1	71,9	70,4
D.B.	76,6	76,8	78,5	80,9	80,6	80,2
D.S.	73,2	74,1	74,7	79,8	77,6	77,0

Glucose Polymer Group

N.D.	68,4	69,3	70,4	71,2	70,0	69,8
P.P.	69,8	69,6	70,6	70,8	72,7	70,8
D.C.	81,8	83,0	83,4	83,2	82,5	82,3
P.Ph.	80,7	82,4	84,1	84,8	84,3	83,9
A.L.	70,4	70,6	71,1	73,4	72,8	71,8
A.R.	93,4	92,6	94,6	96,8	96,2	95,4

Fructose Group

I.D.	68,3	70,4	69,8	71,2	70,6	70,5
J.S.	84,0	84,7	86,1	85,8	86,4	85,8
G.W.	79,3	80,8	81,8	82,2	82,2	81,6
Q.V.E.	77,4	77,1	77,1	79,8	77,6	77,0
G.H.D.	83,5	85,0	86,0	86,5	86,3	85,5
E.L.	66,3	66,6	67,8	68,7	68,4	68,2
Mean ± SD	74,72 ±8,10	75,58 ±7,84	76,38 ±8,25	77,65 ±8,29	77,12 ±8,26	76,46 ±8,26

APPENDIX 6F

MUSCLE SORENESS EVALUATION IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	Day 9	Day 10 (am)	Day 10 (pm)	Day 11	Day 12	Day 13
J.B.	2	3	2	1	0	0
D.P.	2	2	2	0	0	0
J.M.	3	2	2	0	0	0
T.H.	3	2	2	2	1	0
D.B.	3	3	3	3	2	1
D.S.	3	3	3	2	0	0
Mean ± SD	2,7 ±0,52	2,5 ±0,55	2,3 ±0,52	1,3 ±1,21	0,5 ±0,84	0,2 ±0,41

Glucose Polymer Group

N.D.	3	3	3	2	1	1
P.P.	3	3	3	2	1	0
D.C.	3	3	3	1	0	0
P.Ph.	3	2	2	1	0	0
A.L.	3	3	3	3	1	0
A.R.	3	3	3	2	1	0
Mean ± SD	3 ±0	2,8 ±0,41	2,8 ±0,41	1,8 ±0,75	0,7 ±0,52	0,2 ±0,41

Fructose Group

I.D.	3	3	3	3	2	0
J.S.	3	3	3	3	1	0
G.W.	3	3	3	2	1	1
Q.V.E.	3	3	3	1	0	0
G.H.D.	2	2	2	1	1	0
E.L.	3	3	3	3	2	1
Mean ± SD	2,8 ±0,41	2,8 ±0,41	2,8 ±0,41	2,2 ±0,98	1,2 ±0,75	0,3 ±0,52
O/L Mean O/L SD	2,83 ±0,38	2,72 ±0,46	2,67 ±0,49	1,78 ±1,00	0,78 ±0,73	0.22 ±0,43

APPENDIX 6G

SUBJECTIVE ASSESSMENT OF PHYSICAL STATE IN INDIVIDUALS IN STUDY 3

Glucose Group

Athletes	10-km	21-km	32-km	42-km
J.B.	6	6	5	4
D.P.	3	3	2	2
J.M.	6	4	2	1
T.H.	5	3	2	2
D.B.	5	4	2	2
D.S.	4	3	2,5	2
Mean ± SD	4,8 ±1,2	3,8 ±1,2	2,6 ±1,2	2,2 ±1,0

Glucose Polymer Group

N.D.	5	4	4	3
P.P.	4	3	2	1
D.C.	5	4	5	2
P.Ph.	5	3	2	1
A.L.	5	4	2	2
A.R.	5	3	2	2
Mean ± SD	4,8 ±0,4	3,5 ±0,5	2,8 ±1,3	1,8 ±0,8

Fructose Group

I.D.	6	4	3	2
J.S.	4	3	3	2
G.W.	5	4	3	2
Q.V.E.	3	4	3	3
G.H.D.	5	4	3	2
E.L.	3	2	2	2
Mean ± SD	4,3 ±1,2	3,5 ±0,8	2,8 ±0,4	2,2 ±0,4

APPENDIX 6H

INDIVIDUAL RESULTS OF STUDY 3

Glucose Group

Athletes	Fluid Intake	CHO Content	Rectal Temp.
J.B.	2100 ml	42 gm	38,3°C
D.P.	1540 ml	30,8 gm	38,4°C
J.M.	1950 ml	39 gm	38,7°C
T.H.	2200 ml	44 gm	39,1°C
D.B.	2500 ml	50 gm	39,3°C
D.S.	1800 ml	36 gm	38,5°C
Mean ± SD	2015,0 ±332,49	40,30* ±6,65	38,72 ±0,402

Glucose Polymer Group

N.D.	1820 ml	145,6 gm	37,7°C
P.P.	1240 ml	99,2 gm	38,5°C
D.C.	2000 ml	160 gm	38,2°C
P.Ph.	1020 ml	81,6 gm	39,2°C
A.L.	1800 ml	144 gm	38,1°C
A.R.	2040 ml	163,2 gm	39,0°C
Mean ± SD	1653,33 ±422,12	132,27 ±33,77	38,45 ±0,568

Fructose Group

I.D.	1350 ml	108 gm	38,7°C
J.S.	2520 ml	201,6 gm	37,8°C
G.W.	1250 ml	100 gm	38,3°C
Q.V.E.	2000 ml	160 gm	39,2°C
G.H.D.	1950 ml	156 gm	39,0°C
E.L.	2030 ml	162,4 gm	39,5°C
Mean ± SD	1850,0 ±474,9	148,0 ±37,93	38,75 ±0,622
O/L Mean O/L SD	1839,44 ±417,34	106,86 ±56,22	38,64 ±0,525

* Significantly different from other two.

APPENDIX 6I

MUSCLE GLYCOGEN LEVELS (IN ORDER OF ABILITY) (mM/kg MUSCLE) IN INDIVIDUALS IN STUDY 3

Fast

Athletes	Pre-Race	Post-Race	2 Days Post-Race
J.B.	247,2	20,2	270,5
D.P.	265,8	55,1	217,6
J.M.	283,8	37,1	240,5
N.D.	236,8	88,8	145,1
I.D.	246,5	18,1	165,6
J.S.	335,1	35,8	203,2
Mean ± SD	269,2 ±36,38	42,5 ±26,4	207,1 ±46,5

Medium

G.W.	83,4	20,2	52,9
P.P.	164,7	27,2	73,7
D.C.	310,6	96,3	214,3
P.Ph.	243,3	37,2	221,2
T.H.	237,6	57,6	199,19
Q.V.E.	363,1	128,0	210,9
Mean ± SD	233,8 ±100,1	61,4 ±42,4	160,7 ±76,1

Slow

G.H.D.	263,6	32,2	91,5
D.B.	201,2	63,0	157,6
A.L.	168,3	50,2	146,6
D.S.	196,3	109,5	198,1
A.R.	137,7	18,1	155,4
E.L.	161,8	22,8	152,5
Mean ± SD	188,2 ±43,7	49,3 ±34,0	150,3 ±34,2

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