FUEL KINETICS DURING INTENSE RUNNING AND CYCLING WHEN FED CARBOHYDRATE

Thesis submitted for the degree of Master of Science

by

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DEDICATION

To my wife, Belinda, for her encouragement, love and faith that helped me to complete this thesis.

And to my parents for making it all possible.

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DECLARATION

I, *Kevin Dale Derman*, do hereby declare that the experiments described in this thesis were conceived and executed by myself and, apart from the normal guidance from my supervisors, I have received no assistance.

Neither the substance nor any part of this thesis has been submitted in the past, or is being, or is to be submitted for a degree in the University or any other university.

This thesis is presented in fulfillment of the requirements for the degree of MSc.

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ABSTRACT

On two occasions six competitive, male triathletes performed in random order, two experimental trials consisting of either a timed ride to exhaustion on a cycle-ergometer or a run to exhaustion on a motor-driven treadmill at 80% of their respective peak cycling and peak running oxygen uptakes (VO_{2peak}). At the start of exercise, subjects drank 250 ml of a 15 g.100 ml^{-1} $w.v^{-1}$ glucose solution with U-^{14}C glucose added as tracer and, thereafter, 150 ml of the same solution every 15 min. Despite identical metabolic rates (VO_2 3.51 ±0.06 vs. 3.51 ±0.10 $l.min^{-1}$; values are mean ± SEM for the cycling and running trials, respectively), exercise times to exhaustion were significantly longer during cycling than running (96 ±14 vs. 63 ±11 min; $P<0.05$). The superior cycling than running endurance was not associated with any differences in either the rate of blood glucose oxidation (3.8 ±0.1 vs. 3.9 ±0.4 $mmol.min^{-1}$), nor the rate of ingested glucose oxidation (2.0 ± 0.1 vs. 1.7 ±0.2 $mmol.min^{-1}$) at the last common time point (40 min) before exhaustion, despite higher blood glucose concentrations at exhaustion during running than cycling (7.0 ±0.9 vs. 5.8 ±0.5 $mmol.l^{-1}$; $P<0.05$). However, the final rate of total CHO oxidation was significantly greater during cycling than running (24.0 ±0.8 vs. 21.7 ±1.4 mmol $C_6.min^{-1}$;$P<0.01$). At exhaustion, the estimated contribution to energy production from muscle glycogen had declined to similar extents in both cycling and running (68 ±3 vs. 65 ± 5%). These differences between the rates of total CHO oxidation and blood glucose oxidation suggested that the direct and/or indirect (via lactate) oxidation of muscle glycogen was greater in cycling than running.

PUBLICATIONS

The work described in this thesis has been published in the following journal articles:

Full Papers

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CHAPTER ONE

INTRODUCTION AND AIMS OF THESIS

1.0 Introduction, aims of thesis

The effect of carbohydrate (CHO) ingestion on energy metabolism and endurance capacity during prolonged (>90 min), low-to-moderate intensity (55-70% of maximal oxygen uptake [VO_{2max}]) exercise has largely been studied in cyclists protocols (Coggan & Coyle 1988, 1989; Coyle et al. 1983, 1986; Flynn et al. 1987; Ivy et al. 1983; Neufer et al 1987; Hawley et al. 1992a & b; Rauch et al. 1995). Only a few groups have examined fuel kinetics during running (Wilber & Moffat 1992; Madsen et al 1990; Tsintzas et al. 1993a,b). An impression from the cycling and running studies is that the exercise times to exhaustion during moderate-intensity exercise are generally longer during cycling than during running (i.e. compare Coyle et al. 1983 with Tsintzas et al. 1993b; 157±5 min during cycling vs. 125±8 min during cycling). However, the mechanism(s) for a greater endurance during cycling than during running remain unclear.

There is also surprisingly little information on the metabolic demands of exercise at intensities which correspond to race-pace in a variety of athletic events (i.e. >80% of VO_2max). Although prolonged (>90min), moderate-intensity (~70% VO_2max) exercise has received a great deal of scientific attention, many popular sports require participants to exercise at higher (>80% of VO_{2max}) intensities for < 90 min.

Accordingly the aim of the current investigation was to compare the metabolic and endurance responses of well-trained subjects during high-intensity (80% of VO_{2max}) exercise to exhaustion when running and cycling. To my knowledge, no previous study has examined such responses in athletes who are equally well-trained in running and cycling.

CHAPTER TWO

LITERATURE REVIEW

FUEL KINETICS DURING RUNNING AND CYCLING WITH CARBOHYDRATE INGESTION

2.1 Introduction and Historical overview

It has long been the practice of participants in endurance cycling and running events to consume carbohydrates (CHO) during exercise. As early as 1911, respiratory exchange ratio (RER) measurements showed that both fat and carbohydrate (CHO) oxidation provide energy in exercising humans (Zuntz, 1911). These findings were later confirmed by Krogh and Lindhard (1920). They showed that the relative contributions of CHO and fat to energy production was influenced by the pre-exercise diet. These early investigations were primarily concerned with identifying the fuels for muscular activity, relying exclusively upon measurements of RER or plasma glucose concentrations or both to estimate indirectly CHO metabolism during exercise. These experiments, however, were unable to differentiate between endogenous and exogenous sources of glucose in subjects fed CHO during exercise.

Interest in CHO ingestion during exercise arose when three out of 12 runners in the 1923 Boston Marathon were found to have low plasma glucose concentrations after the race (Levine et al., 1924). Levine et al. (1924) proposed that hypoglycaemia was the cause of the extreme fatigue seen in these runners. In order to test their hypothesis, this group subsequently studied the same runners when they consumed CHO during the following year's race (Gordon et al.,1925). In that race, none of the runners developed hypoglycaemia, and all their performances were better than in the previous year's race. Hence, this was probably the first study to indicate that CHO ingestion during exercise could postpone fatigue and enhance performance.

Later, Best and Partridge (1930) also observed hypoglycaemia in runners who completed the 1928 Amsterdam Olympic Marathon, and suggested that the consumption of CHO before and during the race would rectify this problem. Schenk and Craemer (1930) also found low plasma glucose concentrations in runners after a marathon race.

In 1928, the contribution of CHO to energy production was shown to increase at higher exercise intensities (Bock et al. 1928). This finding was later confirmed by Carpenter and Fox (1931) who fed subjects 50 grams (g) of glucose before they cycled at a variety of different work rates. Their conclusion was that, "when glucose is ingested (before exercise), part of the ingested glucose is used for the performance of muscular work". Dill et al. (1932), at the Harvard Fatigue Laboratory in Boston arrived at a similar conclusion. They observed that when their dogs, Joe and Sally, performed treadmill exercise without being fed CHO they became hypoglycaemic and fatigued after 4-6 hr. However, when fed CHO at regular intervals during running, the dogs' endurance increased by about 13 hours. Boje (1936) also showed that feeding CHO solutions to exercising human subjects, at exhaustion, could restore their capacity to continue exercising.

The classic studies of Christensen and Hansen (1939a, 1939b) confirmed the essential role of CHO in the performance of prolonged exercise. They found that endurance was markedly increased after a high CHO diet. The same authors also showed that, when subjects ingested a large quantity (200 g) of glucose at "almost complete exhaustion", it enabled them to perform an additional hour of exercise (Christensen and Hansen, 1939c).

These studies, and those of Boje (1936) showed that glucose ingestion at exhaustion did not significantly alter respiratory exchange ratio values, suggesting that the reversal of fatigue after CHO ingestion was not associated with increases in the rate of CHO oxidation. Instead, it was postulated that the most probable cause of fatigue was the effect of the hypoglycaemia on the central nervous system.

Despite these and other studies showing the importance of CHO ingestion and fluid replacement during exercise (Adolf & Fulton, 1924; Dill et al., 1932; Adolf & Dill, 1938), athletes were unaware of the benefits of ingesting CHO solutions during exercise. In the 1953 International Amateur Athletic Federation (IAAF) Handbook, it was stated that, "*No refreshment may be carried or taken by a competitor other than that provided by the organisers......*" Presumably the only refreshment provided during the race was water.

It was only in 1966 that there was a re-emergence of interest in CHO ingestion and it's effect on exercise performance. Interest in CHO and exercise followed the development of the percutaneous needle muscle biopsy technique which allowed for the determination of the fuel stores within the working muscle. In one-legged cycling studies, Bergstrom and Hultman (1966) showed that the glycogen content of the exercising muscles declined to very low levels at exhaustion, whereas the glycogen content of the non-exercising muscle remained normal. At around the same time, it was also shown that exhaustion was associated with low muscle glycogen contents (Hermansen et al. 1967) and that when exercise began with "supercompensated" muscle glycogen stores, both endurance capacity and the rate of CHO utilisation were increased (Ahlborg et al. 1967a,1967b). Thus, the cause of fatigue in endurance exercise was attributed to a depletion of muscle glycogen stores (Hermansen et al. 1967).

It was only after the late 1970's and early 1980's that ^{13}C and ^{14}C labelling techniques were used to calculate rates of ingested CHO oxidation during exercise (Lefebvre et al. 1975; Lefebvre et al. 1979; Pirnay et al. 1977a, 1977b, 1982; Jandrain et al. 1984, 1989; Krzentowski et al. 1984; Massicotte et al. 1986, 1989, 1990; Pallikarakis et al. 1986; Peronnet et al. 1990; Hawley et al. 1992a; Moodley et al. 1992). Such studies were, on the whole performed on cyclists, possibly because it is easier to take blood from a cyclist on a stationary ergometer that from a runner on a treadmill. Relatively few groups have examined the effects of CHO ingestion in runners and there are no studies in which the rates of ingested CHO oxidation were measured during running (Williams et al. 1990; Wilber and Moffatt, 1992; Tsintzas et al. 1993a,1993b).

2.2 Carbohydrate ingestion and exercise performance

This section of the thesis will review the recent studies on the effects of CHO ingestion on metabolism and exercise performance. Between 1972 and 1983, there were several studies which compared the effects of CHO ingestion and placebo ingestion on exercise times to fatigue.

In 1972, Green and Bagley fed subjects either a placebo or 230 g of a maltodextrin solution prior to and during a canoe race that lasted ~150 min. When the subjects ingested the CHO, they were able to maintain their pace during the final 30 min of the race, whereas when they ingested placebo, they were forced to reduce their pace by 10-15% over the same period.

Two years later, Brooke and Green (1974) examined the effect of an ingestion of glucose syrup on the ability to continue exercising after a previous ride at 70% of VO_2max until exhaustion. Time to fatigue in this study was defined as the duration of exercise until RER fell to 0.73. At this point (153 min) the subjects rested for 40 min and then consumed either CHO or placebo. When subjects were fed CHO rather than placebo, plasma glucose concentrations were maintained during the subsequent exercise bout and exercise times to exhaustion were significantly increased. These authors concluded that "neural factors were probably involved in the differences between treatments". However, Maughan (1991) suggested that the enhanced amount of work performed after the CHO feedings may have been due to an increased availability of plasma glucose for oxidation by the muscles.

Brooke et al. (1975) also reported that CHO ingestion could potentially enhance performance in prolonged moderate intensity exercise. They examined well trained cyclists while they simulated a 100 mile (160 km) race at 67% of VO_{2max}, and consumed either a placebo or CHO. With CHO feedings the rates of CHO oxidation were increased and "work cut off times" were enhanced by approximately 20 min. The authors proposed that "the intensity and duration of work is such that the muscle glycogen (stores) must reduce considerably, and the only way of maintaining such a high carbohydrate participation is by the use of blood glucose..... to support muscle metabolism". Recent studies have supported these observations.

Further evidence that CHO ingestion may improve endurance exercise performance was provided by Ivy et al. (1979). These workers had trained cyclists ingest either ~80 g of maltodextrin or a sweetened placebo while trying to maximise work output during 120 min of cycling. The ingestion of CHO increased the subjects power outputs in the last 30 min of the 120 min of exercise. Later, Ivy et al. (1983) completed a study on 10 experienced cyclists exercising at 74% VO_{2max}. Subjects ingesting CHO showed a significant increase in endurance. However, on closer examination of the results, it was found that only those subjects who developed hypoglycaemia in the placebo trial exhibited an increase in endurance following the ingestion of CHO.

In contrast to the above studies, Felig et al. (1982), reported no differences in the time to fatigue during cycling at 60-65% of VO_2max when subjects ingested either a 5 g.100 ml^{-1} or a 10 g.100 ml^{-1} glucose solution every 15 min throughout exhausting exercise. In agreement with other workers, they showed that prolonged exercise of moderate intensity precipitated hypoglycaemia in approximately 37% of subjects. However, despite correcting this hypoglycaemia, glucose ingestion failed to cause a consistent increase in exercise time to exhaustion. In addition, the time to fatigue in the subjects who ingested only water and who did not become hypoglycaemic was not different from those subjects who developed hypoglycaemia during exercise. Felig et al. (1982), therefore, concluded that “exercise can be continued in the presence of hypoglycaemia,” which “does not support a role for glucose ingestion in improving performance during prolonged exercise.” This work supported the opinion in the late 1970s that glucose ingestion contributed little to the total energy utilised during prolonged exercise (Costill and Miller, 1980).

Several authors in the late 1960's believed that it was possible to increase exercise performance by slowing the rate of muscle glycogenolysis by increasing the availability of blood glucose (Ahlborg et al. 1967; Bergström and Hultman 1967b; Hultman 1967). These authors concluded that net glycogen degradation during intermittent exercise could be reduced by up to 20% with CHO ingestion. This view was also supported by later workers (Ehrenstein et al. 1970; Bagby et al. 1978; Coyle et al. 1983)

However, some scientists in the 1980's, showed that CHO ingestion during exercise had no effect on rates of muscle glycogen utilisation (Fielding et al. 1985; Coyle et al. 1986; Flynn et al. 1987; Hargreaves & Briggs 1988; Noakes et al. 1988b; Mitchell et al. 1989), while others demonstrated a sparing of muscle glycogen when CHO's were ingested during exercise (Björkman et al. 1984; Hargreaves et al. 1984; Erickson et al. 1987; Simard et al. 1988; Brouns et al. 1989). The question of whether water or CHO replacement should be emphasised during prolonged exercise received a large amount of attention in the mid to late 1980s when commercial interests sponsored this type of research.

In 1983, Coyle and co-workers (1983) had 10 trained, overnight fasted, subjects cycle until fatigue at 74% of VO_2max while ingesting either a glucose polymer or a placebo solution. The exercise times to exhaustion were significantly greater with the CHO feedings than with the ingestion of placebo (157 ± 5 min vs. 135 ± 6 min). Coyle et al (1983) concluded that "carbohydrate administration during exercise may result in increased utilisation of blood glucose with a proportional slowing of muscle glycogen depletion". Interestingly, muscle glycogen content was not determined in that study, and subsequent studies have shown this assumption to be incorrect (Coyle et al., 1986; Hargreaves and Briggs, 1988).

At around the same time Bjorkman et al. (1984) investigated the effects of CHO ingestion on the rate of muscle glycogenolysis and the capacity to perform prolonged moderate intensity exercise. They tested eight men who cycled at 68% of VO_2max until exhaustion, while consuming either glucose, fructose, or water. When corrected for an order effect the study showed that time to exhaustion was significantly improved with glucose ingestion, however, muscle glycogen concentration decreased similarly during all three trials.

The question of whether CHO ingestion slowed the rate of muscle glycogenolysis during exercise was raised again by Coyle and co-workers in 1986 (Coyle et al., 1986). Utilising essentially the same protocol as in their earlier study (Coyle et al., 1983), they studied the decreases in muscle glycogen concentration during cycling, with and without CHO feedings. Mean exercise time to fatigue was 33% longer when the subjects were fed a CHO solution, than when they were fed a placebo solution. However, there were no differences in the depletion of muscle glycogen for the first three hours of exercise. Interestingly, the additional hour of exercise, made possible by ingesting CHO, occurred without a further decline in muscle glycogen content. Coyle et al. (1986), concluded that "when blood glucose concentration was maintained, highly trained endurance athletes were capable of oxidising CHO sources other than muscle glycogen at high (~2 $g.min^{-1}$) rates during the latter stages of prolonged strenuous exercise". However, measurements of the rates of plasma glucose oxidation were not determined in that study, and since then no other laboratory has been able to demonstrate such high rates of blood glucose oxidation under euglycaemic conditions.

Further investigations by these workers stressed the importance of CHO feedings during exercise and, in particular, the crucial role of blood glucose as a substrate late in exercise (Coggan and Coyle, 1987; Coggan and Coyle, 1988; Coggan and Coyle, 1989). Coggan and Coyle (1987) showed that fatigue could be reversed by the consumption of CHO in a 20 minute rest period before further exercise. Those athletes consuming CHO in the rest period were able to continue exercising for more than twice as long as those receiving placebo (26 ± 4 min vs. 10 ± 1 min). The onset of fatigue in these trials was associated with a decrease in plasma glucose concentration. In a third trial the athletes were intravenously infused with glucose throughout the subsequent exercise bout. The infusion rate was adjusted to maintain plasma glucose concentration at approximately 5 $mmol.l^{-1}$. These cyclists were able to continue exercising for 43 ± 5 min, i.e. four fold longer than in the placebo trial, and two fold longer than in the CHO ingestion trial. These data suggest that blood glucose concentration falls after ~2 hr of exercise in fasted subjects when no CHO is consumed and that CHO ingestion improves performance by preventing hypoglycaemia.

The findings of Coyle et al. (1983, 1986) and Coggan and Coyle (1987, 1988, 1989) have been supported by other groups utilising a variety of CHO solutions and exercise performance tests (Ivy et al., 1983; Hargreaves et al., 1984; Murray et al., 1987; Neufer et al., 1987; Davies et al. 1988a, 1988b;

Murray et al., 1989a; Murray et al., 1989b; Williams et al., 1990; Murray et al., 1991; Wilber and Moffatt, 1992; Millard-Stafford 1992; Murdoch et al., 1993; Wright et al., 1991; Below et al. 1995). Most of these groups have examined cyclists during exercise at intensities of between 65-75% VO_2max. Relatively few groups have examined the effects of CHO ingestion on running performances.

Williams et al. (1990), showed that subjects completing a 30 km treadmill run were able to run significantly faster in the final 5 km when ingesting CHO than when ingesting placebo. Improved performances with CHO ingestion during running, have also been found by Chryssanthopoulos and Williams, (1996) and Doyle and Elliot, (1996). Chryssanthopooulos and Williams (1996) showed that CHO ingestion increased the running times to exhaustion at exercise intensities of between 67 - 70% VO_2max by 22%. Wilber and Moffat (1992) found that the ingestion of a glucose polymer drink increased endurance performance at 80% VO_{2max} by 29%. Thus, CHO ingestion appears to improve endurance in both cycling and running exercise.

2.3 Rates of plasma glucose oxidation

From the literature, it appears that athletes who do not consume CHO during prolonged exercise, develop a low blood glucose concentration towards the end of the exercise period (Ahlborg et al., 1974; Ahlborg & Felig, 1976; Coggan and Coyle, 1987). A decreased blood glucose concentration may be one cause of fatigue in athletes (Levin et al. 1924; Gordon, 1925). Other possible causes of fatigue include muscle glycogen depletion, potassium accumulation in the muscle T-tubules, a reduction in the force generating capacity of the myofibrils due to acidosis, or a failure of Ca^{2+} homeostasis, or fatigue due to neural causes (Dill et al. 1932; Sjoggard et al. 1986; Vollestäd et al. 1988; Vollestäd et al. 1990).

2.3.1 Effect of exercise duration

Rates of blood glucose oxidation depend on the duration of exercise at any given intensity. In the early stages of moderate intensity (60-70 % VO_{2max}) exercise blood glucose utilisation represents approximately one fourth of total oxidative energy production and one third to one half of total CHO energy production (Wahren et al. 1971; Ahlborg et al, 1982; Coyle et al., 1986; Katz et al. 1986; Janssen & Kaijser, 1987; Broberg & Sahlin, 1989). However, as exercise continues and muscle glycogen stores become depleted, blood glucose oxidation plays a progressively more important role in the maintenance of energy supply (Ahlborg et al. 1982; Coyle et al., 1986; Coggan & Coyle, 1987; Broberg & Sahlin, 1989; Stein et al. 1989; Bosch et al., 1993a). Even in low intensity exercise (30% VO_{2max}), blood glucose oxidation increases to a plateau over the first 90 min (Ahlborg et al., 1974).

2.3.2 Effect of exercise intensity

At low exercise intensities of 30% VO_{2max}, however, fat remains the main source of energy, as evidenced by low RER values (Ahlborg et al.1974). Glycogen and blood glucose only become increasingly important for providing energy at higher exercise intensities(Wahren et al. 1971; Coggan and Coyle, 1987).

In order to determine the effect of exercise intensity on ingested CHO oxidation Pirnay et al. (1982), examined four different exercise intensities ranging from 22 to 64% of VO_2max. Their results showed that exogenous CHO oxidation only increased with exercise intensities up to 50% of VO_2max. From 50 to 64% of VO_2max the rates of exogenous CHO oxidisation reached a plateau at 0.59 $g.min^{-1}$. These and other studies suggest that rates of ingested glucose oxidation during exercise at intensities up to 70% of VO_{2max} may be limited to ~1.0 $g.min^{-1}$ (for review see Hawley, Dennis and Noakes 1992).

At higher (>70% VO2max) exercise intensities, the oxidation of ingested CHO are not known. In theory, high exercise intensities reduce ingested CHO oxidation by slowing splanchnic blood flow and intestinal absorption (Barclay and Turnberg 1987; Maughan et al. 1990; Williams et al. 1964).

The reported relative contributions of absorbed glucose to the total amount of CHO oxidised during exercise vary considerably. In some cases, the oxidation of ingested CHO has been reported to contribute only 5 - 10% of total CHO oxidation (Costill et al. 1973; Van Handel et al. 1980) but those studies are the exception. The majority of trials have shown that the contribution from ingested glucose to total CHO oxidation during exercise is between 18 to 40% (Pirnay et al. 1977a; Jandrain et al. 1984, 1989; Decombaz et al. 1985; Massicotte et al. 1986, 1989, 1990; Hawley et al. 1992b) and up to 68% in fasted subjects (Pallikarakis et al. 1986).

2.4 Comparison of Cycling versus Running

The above data were all obtained from studies of cyclists. To my knowledge there is no information on the rates of ingested CHO oxidation in runners. Only the rates of glycogen utilisation have been compared in running and cycling. Pascoe et al., (1990) showed that muscle glycogen disappearance was greater in cycling than in running for 60 min at 75% of VO_2max. Differences in the rate of muscle glycogen utilisation suggest that individual motor units may be working at a lower relative exercise intensity in running than in cycling. Running is thought to involve a greater muscle mass than cycling (Hermansen and Saltin 1969).

In keeping with the above assumption, it is expected that the athlete performing the exercise modality involving the greatest muscle mass, would produce the higher VO_{2max} value (Astrand, 1970). Indeed, when examining single sport athletes, cyclists produce VO_{2max} values of 9 to 11% less than the treadmill values (Astrand, 1970; McArdle et al., 1981). This percentage decreases with well trained cyclists. However, this percentage difference has been shown to decrease when testing triathletes whom are trained in both exercise modalities, with cycling VO2max values being only three percent less than the running values (O'Toole et al., 1987, Roalstad 1989). It has been suggested that the smaller difference is perhaps due to the concurrent training in cycling and running carried out by the triathlete compared to the more mode-specific training of a single sport athlete (Roalstad, 1989)

2.5 Gastric Emptying

Running and cycling exercise may also differ in term of gastric emptying, but again, the majority of studies have been conducted on cyclists or at rest. A particularly important factor influencing gastric emptying, is the volume of solution ingested.

Larger ingested volumes have been shown to empty more rapidly from the stomach than smaller ingested volumes (Marbaix 1898; Hunt and MacDonald, 1954; Costill and Saltin, 1974; Minami and McCallum, 1984; Noakes et al. 1991). Davenport (1969) showed that gastric peristalsis is increased by parasympathetic activity and decreased by sympathetic stimulation.

The osmotic pressure and caloric content of gastric contents has also been shown to influence the rates of gastric emptying (Foster et al., 1980; Costill 1990). Isotonic solutions leave the stomach more rapidly than hypotonic or hypertonic solutions, however with glucose solutions hypotonic solutions tend to empty far more rapidly than isotonic solutions (Carnot and Chassevant, 1905).

Gastric emptying may also be affected by exercise. It has long been noted that moderate to severe exercise has an adverse effect on gastric emptying and digestion (Beaumont, 1833; Graham, 1851; Campbell et al., 1928). This finding was supported by later authors who observed that exercise had no effect on gastric emptying until the exercise intensity exceeded 65-80% of VO_2max (Costill and Saltin, 1974; Feldman and Nixon, 1982). At high exercise intensities rates of gastric emptying may be slowed by the inhibitory effects of increased catecholamine and endogenous opiod levels on splanchnic blood flow and gastric motility (Murray, 1987; Neufer et al. 1989a). In contrast, mild to moderate exercise (walking and slow running) appears to increase the rates of gastric emptying above those at rest (Neufer et al., 1986, 1989b).

Comparisons of the effects of running and cycling on gastric emptying are equivocal. Mitchell et al. (1989) showed a consistently faster rate of gastric emptying of six and 10 g.100 ml^{-1} CHO solutions, during running at 70% VO_2max than cycling at the same intensity. They felt that the running motion may facilitate gastric emptying by shifting the stomach's contents towards the antrum, and thereby promoting the delivery of chyme to the duodenum (Neufer et al. 1986). In contrast, Rehrer et al. (1990) found that gastric emptying rates were equal for the first 40 min of running and cycling at 70% VO_2max, but were faster later during cycling than during running when an isotonic CHO solution was repeatedly ingested during exercise. However, there were no differences in the rates of gastric emptying for the entire 80 min period when a hypertonic drink was consumed. Houmard et al (1991), also found no differences in the rates of gastric emptying in subjects running and cycling at 75% of their respective VO_{2max}'s for 1hr.

2.6 Summary

For over 70 years exercise scientists have examined the effects of CHO ingestion on metabolism and performance during exercise (Levine et al. 1924; Gordon et al. 1925). However, the majority of these studies have focused on endurance cyclists. This was probably due to the ease of testing the cyclist on a stationary ergometer in the laboratory compared to the runner. Since this time it has largely been assumed that the information gained from these studies could be utilised and extended to runners. However, the fuel kinetics between running and cycling may not necessarily be identical (Madsen et al. 1990; Wilber and Moffat, 1992; Medelli et al. 1993). Further, making practical recommendations to runners based on the results of studies performed on cyclists could lead to adverse effects on running performance.

Most (Hargreaves et al, 1984; Fielding et al. 1985; Coyle et al. 1986; Coggan and Coyle 1987, 1989; Williams et al. 1990; Wilber and Moffat, 1992), but not all (Felig et al., 1982; Sasaki et al., 1987, Noakes et al. 1988b, Riley et al., 1988) studies have shown that CHO ingestion can improve exercise performance. Indeed, a recent study has shown a large 29% increase in the endurance performance of runners when ingesting CHO and exercising at speeds eliciting 80% of VO_2max (Wilber and Moffat, 1992).

It has been hypothesised that CHO ingestion may delay fatigue during endurance exercise by sparing muscle glycogen. Over the past 30 years there has been evidence both for and against muscle glycogen sparing when ingesting CHO while exercising (Ahlborg et al. 1967b; Bergström and Hultman 1967; Hultman 1967; Ehrenstein et al. 1970; Bagby et al. 1978;

Björkman et al. 1984; Hargreaves et al. 1984; Fielding et al. 1985; Coyle et al. 1986; Flynn et al. 1987; Erickson et al. 1987; Hargreaves & Briggs 1988; Simard et al. 1988; Brouns et al. 1989; Noakes et al. 1988; Mitchell et al. 1989). However, Coggan and Coyle (1991) concluded that CHO ingestion while cycling at moderate-intensity (70-75% of VO_2max) does not reduce the rate of muscle glycogen utilisation.

Tracer techniques have shown that ingested CHO oxidation increases with increasing exercise intensity and duration up to about 50% of VO_2max. Thereafter, CHO ingestion seems to plateau at around 1 $g.min^{-1}$ after approximately 90 min of moderate intensity (70% VO_2max) exercise (Hawley, Dennis and Noakes 1992a).

It is now generally accepted that CHO ingestion while running will elicit a greater gastric discomfort when compared to cycling at the same relative intensity (Rehrer et al. 1989, 1990). However, some studies have shown that the rates of gastric emptying between the two exercise modalities are similar (Mitchell et al. 1989; Rehrer et al. 1990; Houmard et al. 1991), while others have shown greater rates of emptying while cycling at the same relative intensity than running (Rehrer et al. 1990).

In either running or cycling there is relatively little data on the metabolic demands of exercise at high exercise intensities (> 75% of VO_2max). CHO ingestion has been shown to improve performance in both running and cycling. However, it remains unclear if the metabolic advantages are equal during cycling and running. Accordingly, this study will examine the fuel kinetics during cycling and running at 80% of VO_2max in an attempt to answer the question of whether the ingestion of CHO remains an important source of energy in exercise at high intensities.

CHAPTER THREE

METHODOLOGY

METHODS

3.1 Subjects

Six competitive male triathletes who where involved in regular endurance training and competition participated in this study. Only subjects who felt that they were equally trained in running and cycling prior to and during the testing period were selected for the investigation. This was also confirmed in discussion regarding their training schedules. The characteristics of the subjects are shown in Table 1 (see results section).

All procedures in this study were approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town. As tracer amounts of U-^{14}C-glucose (~ 0.7 mBq) were ingested and blood samples were taken, the risks was fully explained to all subjects before they provided their written consent. The total radiation dose received by each subject was < 0.04 mSv which was well below the accepted safe dose in this country of 5 mSv.yr^{-1} or 1.3 mSv.13 wk^{-1} (Bosch et al. 1993a).

3.2 Preliminary testing

3.2.1 Peak sustained power outputs (PPO)

Peak power outputs (PPO) in Table 1 were determined during a progressive, maximal exercise protocol on an electronically braked cycle ergometer (Lode, Bilthoven, Holland), as described in detail previously (Hawley and Noakes 1992). Briefly, after a 10 min "warm-up", each subject started cycling at a work rate of 3.33 Watts (W).kg^{-1} body mass for 150 sec, after which the work rate was increased by first 50 W and then 25 W.150 .sec^{-1} until the subject became exhausted.

PPO was defined as the last completed work rate in W plus the fraction of time spent in the final non-completed work rate multiplied by the 25 W work rate increase. This was calculated using the following equation (Kuipers et al. 1985; Hawley and Noakes 1992)

$$\mathbf{PPO = W_{final} + ((t / 150) \cdot 25\ W)}$$

where PPO is defined as the peak sustained power output (W); W_{final} was the last exercise intensity (W) the subject completed for the full 150 sec; t was the number of seconds for which the final, uncompleted exercise intensity was sustained; and 25 W was the final work rate increment . Exhaustion was defined as a drop in the pedalling frequency of > 10 revolutions.min^{-1} and was always associated with a respiratory exchange ratio (RER) of > 1.10.

3.2.2 Peak treadmill running velocity

Peak treadmill running velocity was determined during a maximal test on a horizontal, motor-driven treadmill (Powerjog EG30 Sports Engineering, Birmingham, UK), as previously described by Scrimgeour et al. (1986). The treadmill was calibrated before all maximal tests and the subsequently described trials. The initial treadmill speed for the maximal test was 13 km.hr^{-1} which was increased by 1 km.hr^{-1} every 60 sec until the subject felt he could no longer maintain the pace. Peak treadmill velocity was taken as the final work rate a subject could maintain for 60 sec.

3.2.3 Peak cycling and running oxygen consumption (VO_{2peak})

During both the progressive cycling and running exercise tests, the subjects wore a nose-clip and inspired air via a Hans Rudolph 2700 one-way valve (Vacumed, Ventura, CA, USA) connected to a dry gas meter. Expired air was passed though a 15 l baffled mixing chamber and a condensation coil to an Ametek S-3A/I O_2 analyser with a N-22M sensor and a CD-3A CO_2 analyser with a P-61B sensor (Thermox Instruments, Pittsburgh, USA). Before each test, the gas meters were calibrated with a Hans Rudolph 5530 3 l syringe and the analysers were calibrated with a 16% CO_2 : 4% O_2 : 80% N_2 gas mixture of known composition. The instrument outputs were processed by an on-line IBM computer which calculated inspired ventilation volume (V_i), VO_2 and carbon dioxide production (VCO_2) values over each minute using conventional equations (Jones 1982). Peak cycling and running VO_2 values in the last completed work rate, were used to determine the exercise intensities corresponding to 80% of each subject's peak cycling and running VO_2 values for the subsequent experimental trials.

3.3 Experimental trials

Each subject performed a random order of two experimental trials. The resulting outcome of this randomisation was that four subjects performed the running trials first and two the cycling first. In these trials the subjects either rode to exhaustion on the cycle-ergometer or ran to exhaustion on the treadmill at exercise intensities corresponding to 80% of their peak cycling and running VO_2max, respectively (Figure 1). Trials were separated by a period of seven days during which time the subjects continued with their normal training and consumed their normal diet. Training and dietary records were kept by each subject to aid compliance.

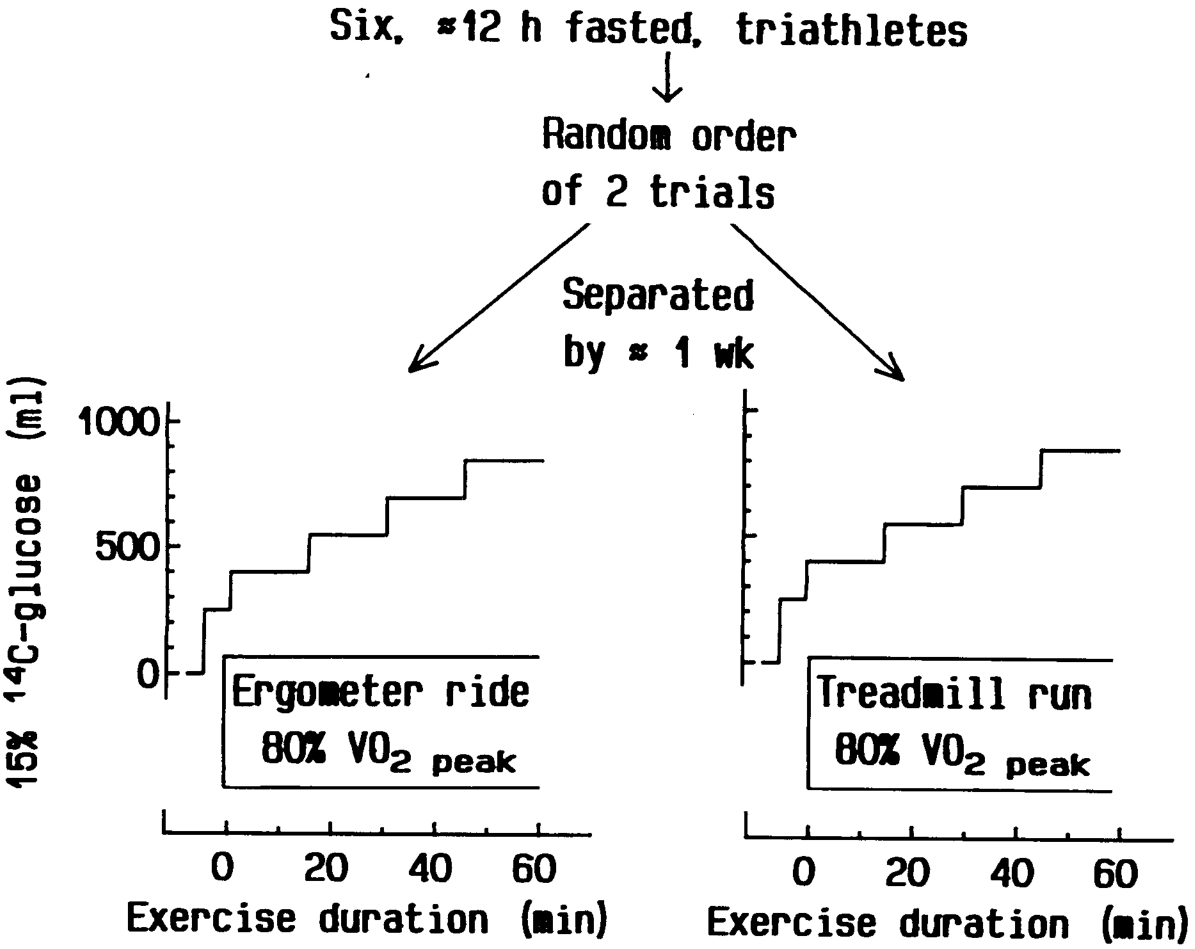

Figure 1. Schematic representation of experimental trials for both cycling and running groups, showing the ingestion protocol for the 15% (U-^{14}C) glucose solution.

On the day before each trial, subjects refrained from exercise and were instructed to ingest meals of a balanced nature. Prior to the second trial, subjects were instructed to ingest meals similar to those consumed the night before the first trial. This was verified by checking their training and dietary records. The next day, the subjects reported to the laboratory in a 12-14 hr overnight-fasted state. Then, with subjects in the supine position, a 21-gauge cannula was positioned in an antecubital vein and connected to a three-way stop-cock for the collection of blood samples (10 ml) at rest and during the subsequent described exercise bout. For the cycling trials, the stop-cock was attached directly to the cannula on the inner forearm and, for the running trials, the stop-cock was connected to the cannula via a length of sterile tubing and secured on the back of the wrist. After each blood sample, the tubing and/or cannula was flushed with 2-3 ml of sterile saline containing heparin (5 $IU.ml^{-1}$) and, for that reason, the first 2-3 ml of the next blood sample was discarded.

Following the withdrawal of a resting venous blood sample, the subjects ingested 250 ml of a ~ 0.9 $kBq.mmol^{-1}$ (~25 $\mu Ci.mmol^{-1}$), 15 g.100 ml^{-1} $w.v^{-1}$ U-^{14}C labelled -glucose solution (Amersham International, Buckinghamshire, UK) and began a 5 min "warm-up" at a speed eliciting ~50% of VO_{2peak}. Thereafter, the exercise intensity was increased and the subjects exercised to exhaustion at 80% of VO_{2peak} while ingesting 150 ml of the same U-^{14}C labelled - glucose solution every 15 min.

Prior to each drink, the subjects were asked to report their rating of perceived exertion (RPE) using the Borg 10 point scale (Borg, 1975). At the same time, they were asked to rank their stomach fullness on a scale of one (empty) to five (uncomfortably bloated).

3.4 Respiratory gas exchange measurements

After 10 min of exercise and at each subsequent 15 min until exhaustion, $V^{14}CO_2$ specific radioactivity (in $dpm.mmol^{-1}$) were measured by passing expired air from the VO_2 and VCO_2 analyser vents though a solution of 1 ml of 1N hyamine hydroxide in methanol, 1 ml of 96% ethanol and 1-2 drops of phenolphthalein. Expired air was bubbled though this solution for 2-3 min until the phenolphthalein indicator changed from pink to clear, at which point 1 mmol of CO_2 was trapped as previously described (Scherrer et al. 1978). Liquid scintillation cocktail (10 ml, Ready Gel, Beckman Instruments, Fullerton, CA, USA) was then added to the solution and $^{14}CO_2$ disintegrations.min^{-1}. (dpm. $mmol^{-1}$ were counted in an Insorb 460C Automatic Liquid Scintillation counter (United Technologies, Packard, Illinois, USA). All ^{14}C counts were corrected for differences in quench and background counts.

3.5 Rates of carbohydrate oxidation

After the ^{14}C in the expired CO_2 had been counted in a scintillation counter the rate of ingested CHO oxidation were calculated from the following equation:

$$\mathbf{Ing.\ CHO_{ox} = (^{14}CO_2 \bullet 6 / [(SA_{CHO} / CHO_d) \bullet MW_{CHO}]) \bullet VCO_2 \bullet 1.35}$$

where Ing. CHO_{OX} is the rate of ingested CHO oxidation in $g.min^{-1}$; $^{14}CO_2 \bullet 6$ is the $^{14}CO_2$ $dpm.mmol^{-1}$ count multiplied by six, as there are six carbon atoms per molecule of monosaccharide absorbed in the blood; SA_{CHO} is the radioactivity counts in the ingested solution in dpm.ml^{-1}; CHO_d is the CHO content of the ingested drink in g/l; MW_{CHO} is the molecular weight of the CHO; VCO_2 is the volume of expired CO_2 in l/min; and 1.35 is the number of grams of hexose oxidised to produce 1l of CO_2.

3.6 Plasma glucose and insulin concentrations

Venous blood samples (10 ml) collected at rest and during the measurements of gas exchange were divided into two aliquots and stored on ice until the end of the trial. One series of aliquots were placed in tubes containing lithium heparin, centrifuged at 3,000 rev.min^{-1} for 15 min at 4°C and the supernatant was stored at -20°C for radio-immunoassay of plasma insulin concentration (Pharmacia Diagnostics AB, Uppsala, Sweden). The remaining aliquots were placed into tubes containing potassium oxalate and sodium fluoride, processed as before, and the frozen supernatants were used for (a) determinations of plasma glucose concentrations by an automated glucose analyser (LM3 Glucose Analyser, Analox Instruments, London, UK) and (b) measurements of plasma glucose and lactate specific radioactivity's. The validity of the glucose assay was tested prior to each group of samples being processed.

3.7 Plasma glucose and lactate specific radioactivity's

Plasma samples (1 ml) for the separation of glucose and lactate were adjusted to ~ pH 4 with 60 μl of H_2PO_4 (2 mol.l^{-1}), placed in sealed tubes and deproteinised by heating for 10 min at 70°C in a shaking water bath. After the samples had cooled on ice for 10 min, condensation on the inside walls of the tubes was washed down with 1 ml of H_2O (raised to pH 8.0 by addition of traces of NaOH) and the samples were adjusted to pH 7.0 with ~ 40 μl of K_2CO_3 (3 mol.l^{-1}) and centrifuged at 5,000 rev.min^{-1} for 10 min. Following centrifugation, the supernatant was stored on ice and the pellet was re-suspended in 0.75 ml of H_2O and re-centrifuged a total of three times.

The supernatants from the washed pellets were added to the previous supernatant and passed though a 500 mg Sephadex anion exchange column (Bakerbond SAX, Cape Town, RSA) that had been conditioned with several void volumes of distilled water adjusted to pH ~8.0 with traces of NaOH. Glucose appeared in the void volume and was fully eluted into a scintillation vial with 3 ml of H_2O. Lactate was subsequently eluted into a second scintillation vial with 2 ml of $CaCl_2$ (1 mol.l^{-1}), adjusted to a pH of 2.0 with HCl. Passage of the solution though the exchange column was accelerated to ~1 ml.min^{-1} with a vacuum processor. After collection, the elutes were evaporated to near dryness at 70 °C for ~20 h before liquid scintillation cocktail was added for radioactive counting.

Each time plasma glucose and lactate were separated, a non-labelled plasma sample was spiked with a known quantity of U-^{14}C-glucose and run simultaneously to correct the measured dpm values for the percentage recovery. Corrections for the percent recovery were performed so that plasma glucose dpm.mmol^{-1} specific radioactivity could be calculated from the glucose concentrations measured in corresponding undiluted plasma samples. U-^{14}C glucose recoveries were all > 92%.

3.8 Glucose oxidation

As plasma lactate counts were not significantly different from the background counts, the rates of plasma glucose oxidation did not need to be corrected for ^{14}C lactate oxidation and were determined with the following formula:

$$\mathbf{GLU_{ox} = (SA\ CO_2 / SA\ glu) \bullet VCO_2}$$

In this equation, GLUox is the rate of plasma glucose oxidation in mmol.min^{-1}; SA CO_2 is the specific (radio) activity of expired $^{14}CO_2$ in dpm.mmol^{-1}; SA glu is the corresponding specific (radio) activity of the plasma glucose in dpm.mmol^{-1}; and VCO_2 is the volume of expired CO_2 in mmol.min^{-1}, calculated from the l.min^{-1} VCO_2 and the 22.4 ml.mmol^{-1} gas volume. Since the complete conversion of one molecule of U-^{14}C-glucose to six molecules of $^{14}CO_2$ decreases the dpm.mmol^{-1} specific radioactivity by a factor of six, the VCO_2 values did not need to be divided by six to allow for six CO_2 molecules arising from oxidation of one glucose molecule.

The same equation was used to determine the rates of ingested CHO oxidation. However, in this case, SA glu was the specific radioactivity of the drink in dpm.mmol^{-1} glucose equivalents.

This formula does not take into account the time taken to equilibrate $^{14}CO_2$ with the HCO_3^- pool. Although this time has been reported to vary between five (Costill et al. 1973) and 90 min (Coggan et al 1993), it can be predicted from the flux of CO_2 through the body HCO_3^- stores that equilibration is essentially complete in 20-30 min (Bosch et al. 1993b). More to the point, any systematic lag in the appearance of $^{14}CO_2$ in the breath would have been similar from trial to trial.

3.8 *Rates of total carbohydrate and fat oxidation*

Rates of total CHO oxidation and fat oxidation were calculated from the $l.min^{-1}$ VCO_2 and VO_2 values using the formulae of Consolazio et al. (1963), assuming a non-protein RER. Rates of CHO oxidation (in $g.min^{-1}$) were converted to mmol $C_6.min^{-1}$ units by dividing the values by the 180 $mg.mmol^{-1}$ mol. wt of glucose. Differences between the mmol $C_6.min^{-1}$ rates of total CHO oxidation and plasma glucose oxidation provided an estimate of the direct and indirect (via lactate) oxidation of muscle glycogen.

3.9 *Statistical analyses*

All results are expressed as means ± the standard error of the mean (SEM). Since some of the six subjects fatigued after ~45 min, only the measurements at the earlier time points and at exhaustion have been compared. Differences in times to exhaustion were analysed with a paired Students *t*-test. Statistical significance's of differences between trials over time were assessed by a two-way analysis of variance (ANOVA) for repeated measures and located by Scheffes post hoc tests. A value of $P < 0.05$ was regarded as significant.

CHAPTER FOUR

RESULTS

4.0 Results

Table 1 shows the cycling and running peak VO_2 values of the triathletes in this investigation.

Table 1. Subject Characteristics

Age (yr)	**Height (cm)**	**Mass (kg)**	**PPO (W)**	**Peak Treadmill velocity ($km.h^{-1}$)**	**VO_{2peak} (Cycling) ($l.min^{-1}$)**	**VO_{2peak} (Running) ($l.min^{-1}$)**
28.7	179	72.8	358	21.2	4.53	4.50
(2.0)	(0.01)	(1.1)	(9.0)	(0.2)	(0.09)	(0.09)

PPO, Peak sustained power output determined during the maximal cycle test;

Treadmill velocity, the peak treadmill velocity attained during the maximal running test (see text for details); VO_{2peak}, peak oxygen uptake.

All values are mean ± SEM of 6 subjects.

Because the triathletes' cycling and running VO_{2peak} values were identical, the rides to exhaustion at 80% of peak VO_2 were performed at comparable metabolic rates. Average absolute VO_2 values were 3.51 ± 0.06 $l.min^{-1}$ in the cycling trial and 3.51 ± 0.10 $l.min^{-1}$ in the running trial (Table 2).

Table 2. Steady-state gas exchange data during cycling and running at 80% of VO_{2peak} while ingesting carbohydrate

Exercise time (min)	10	25	40	Exhaustion
VO_2 (l.min^{-1})				
Cycling	3.48	3.44	3.53	3.58
	(0.06)	(0.08)	(0.08)	(0.06)
Running	3.38	3.48	3.60	3.58
	(0.08)	(0.08)	(0.07)	(0.10)
RER (units)				
Cycling	0.99	0.97	0.97	0.96
	(0.01)	(0.01)	(0.01)	(0.01)
Running	0.96*	0.96	0.95*	0.94*
	(0.01)*	(0.01)	(0.01)	(0.01)

VO_2, Oxygen consumption; RER, respiratory exchange ratio.
* Denotes a significance difference ($P<0.05$) between running and cycling.
All values are mean ±SEM of 6 subjects.

4.1 Running and cycling times to exhaustion

Despite the similar metabolic rates, however, there were large differences in the exercise times to exhaustion for the cycling and running trials (Figure 2). Whereas subjects were able to cycle for 96 ± 14 min at 80% of peak VO_2, they could run for only 63 ± 11 min at the same exercise intensity ($P< 0.05$).

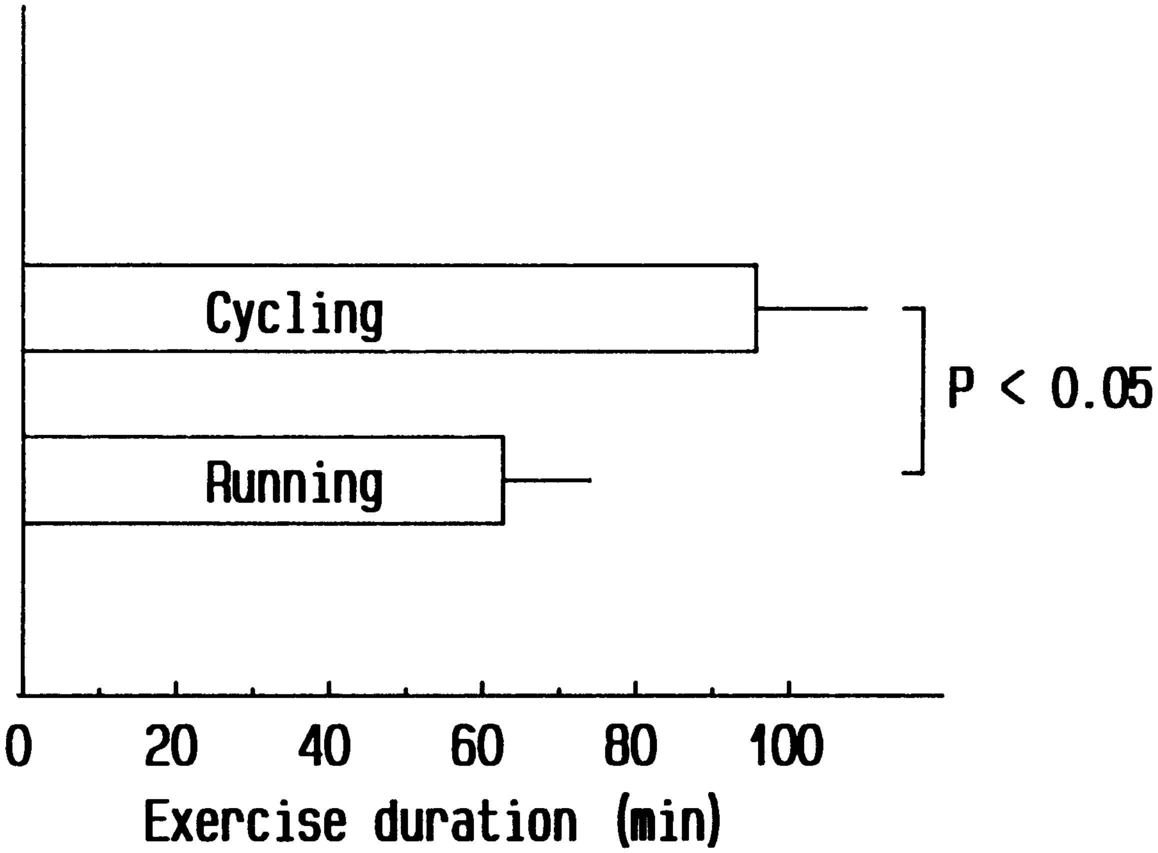

Figure 2 Cycling and running times to exhaustion at 80% of maximal oxygen uptake when ingesting carbohydrate

4.2 Ratings of perceived exertion and stomach fullness

The longer endurance times in cycling than in running were not associated with any real differences in RPE during exercise (Figure 3). Although the RPE values at 40 min tended to be lower when cycling than when running, the differences did not reach statistical significance.

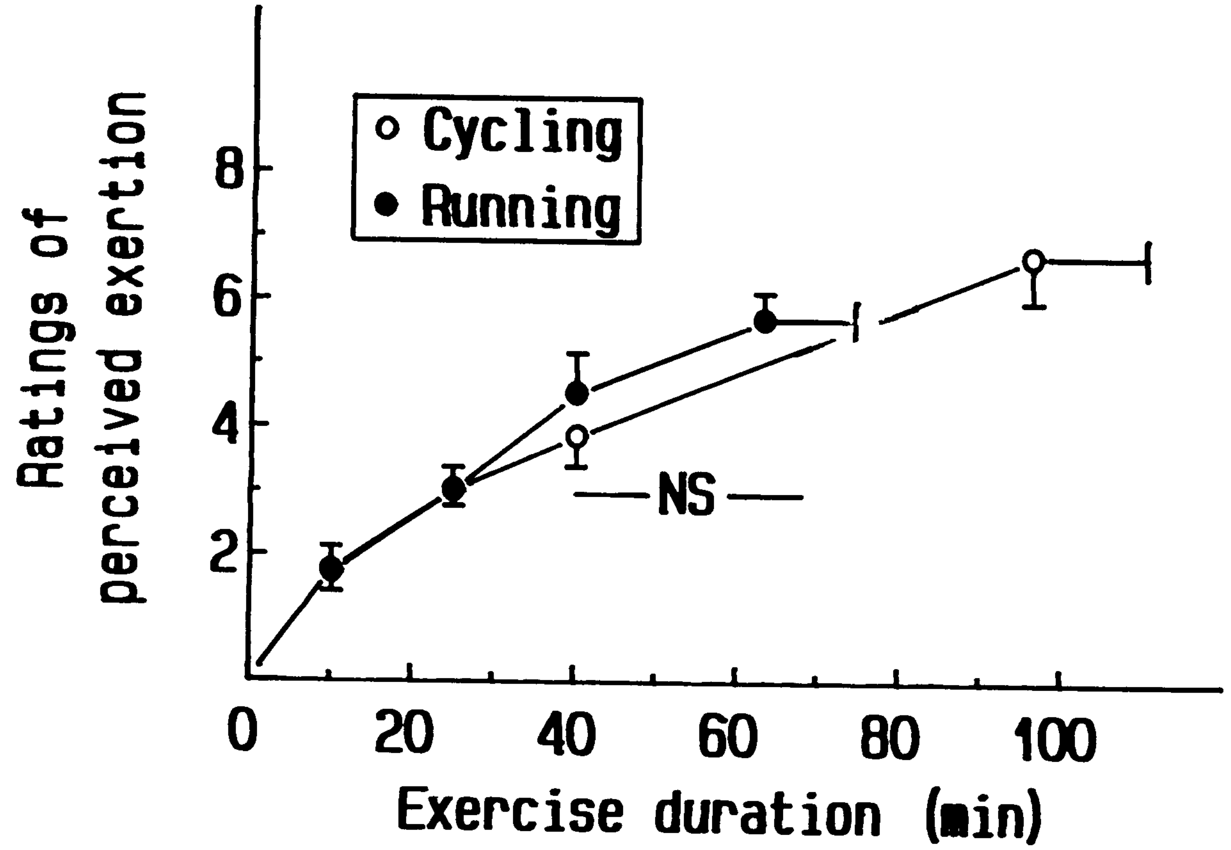

Figure 3 Ratings of perceived exertion during cycling and running at 80% of VO2max while ingesting carbohydrate

The consistently lower ratings of stomach fullness in the cycle rides than in the runs were also not significant (Figure 4). However, while none of the subjects complained of gastric distress during the cycle rides, five of the six subjects felt nauseous at the end of the run. The nausea was not severe enough to cause the termination of the exercise trial.

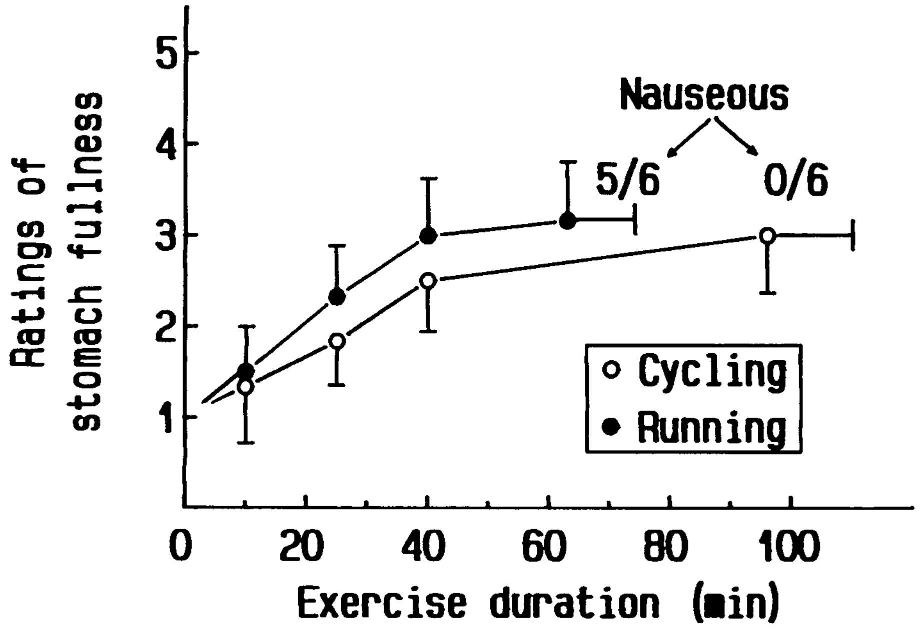

Figure 4 Ratings of stomach fullness on a five-point scale, during cycling and running at 80% of VO_{2peak} while ingesting carbohydrate

4.3 Plasma glucose and plasma insulin concentrations

Differences in the rises in plasma glucose concentration during the cycling and running trials are shown in Figure 5. In the cycling trial, plasma glucose concentration increased from 4.8 mmol.l^{-1} to a plateau of 5.8 mmol.l^{-1} after ~40 min. However, during the running trial, plasma glucose concentration continuously rose from 4.9 ± 0.1 to 7.0 ± 0.9 mmol.l^{-1} and was significantly greater than in the cycling trial at 40 min and at exhaustion.

Greater rises in plasma glucose concentration in the running trial than in the cycling trial, however, had no effect on circulating plasma insulin concentrations. During both trials, plasma insulin concentrations remained relatively constant over time and averaged 5.8 ± 0.5 mU.l^{-1} during the cycle rides and 5.7 ± 0.6 mU.l^{-1} during the runs.

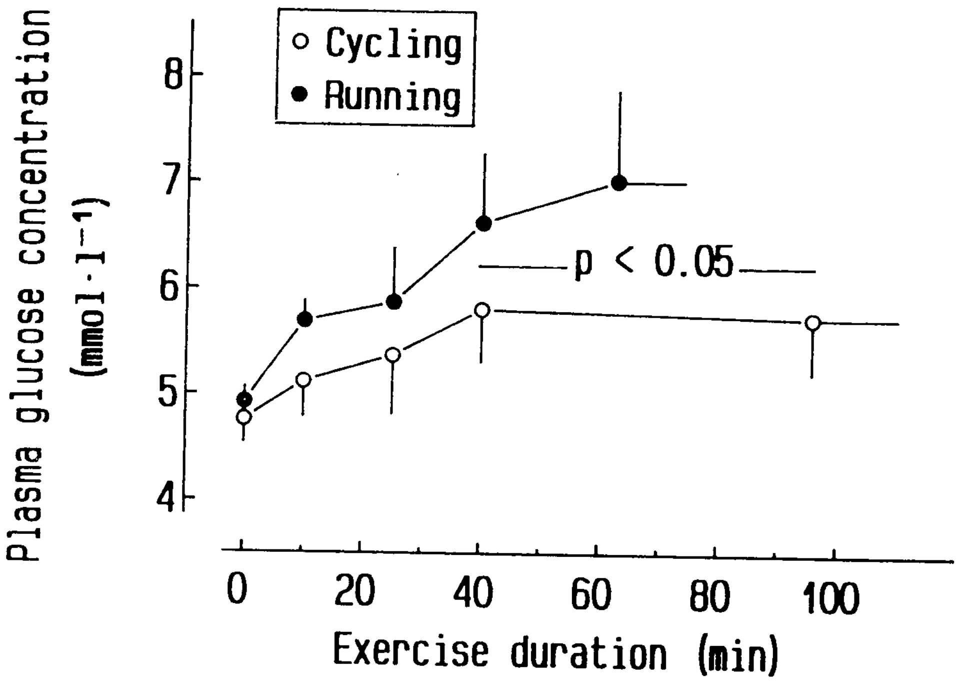

Figure 5 Plasma glucose concentration during cycling and running to exhaustion at 80% of maximal oxygen uptake when ingesting carbohydrate

4.4 Ingested and total glucose oxidation

Rises in the rates of both total glucose and ingested glucose oxidation were also similar in the two trials (Figure 6). During the cycle rides, rates of total glucose oxidation increased to 5.8 ± 0.5 mmol.min^{-1} at exhaustion and, in the runs, they rose to 4.6 ± 0.7 mmol.min^{-1} at the end of exercise. Lower final rates of total glucose oxidation in the runs than in the cycle rides were a reflection of the shorter duration of the running trial. At 40 min, the rates of total glucose oxidation were both ~ 3.9 mmol.min.^{-1}

Rates of ingested glucose oxidation also followed a similar time course in the two trials (Figure 6). Peak rates of ingested glucose oxidation at exhaustion were 3.0 ± 0.3 mmol.min^{-1} in the cycle ride and 2.2 ± 0.3 mmol.min^{-1} in the run. In each case, the peak rates of ingested glucose oxidation accounted for ~50% of the final rates of total glucose oxidation.

4.5 Total carbohydrate oxidation

In contrast, rates of total CHO oxidation were significantly greater in the cycling than in the running trial (Figure 6). Mean rates of total CHO oxidation were 24.0 ± 0.8 mmol $C_6.\text{min}^{-1}$ during the cycle ride and 21.7 ± 1.4 mmol $C_6.\text{min}^{-1}$ during the run ($P < 0.01$).

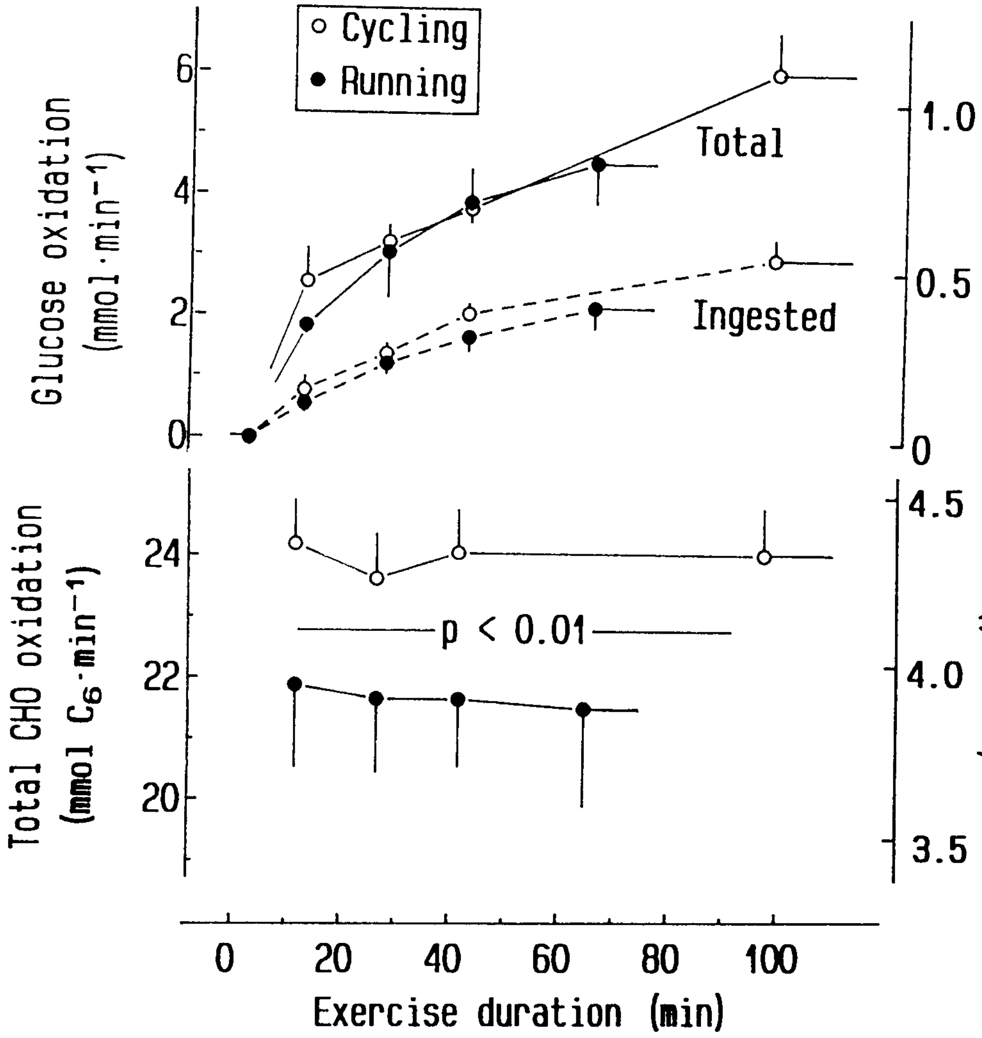

Figure 6 Blood glucose oxidation and ingested carbohydrate oxidation (*top panel*), and total carbohydrate oxidation (*lower panel*) during cycling and running to exhaustion at 80% of maximal oxygen uptake when ingesting carbohydrate

4.6 Fat oxidation

Higher rates of total CHO oxidation in cycling than in running were associated with lower rates of fat oxidation (Figure 7). Whereas the contribution to energy production from fat oxidation increased to 17 ± 4% mJ at the end of the running trial, it rose to only 10 ± 2% mJ in the cycling trial (P<0.01).

In contrast, the higher rates of total CHO oxidation during cycling than during running did not influence the rate of energy production from glucose oxidation (Figure 7). In both trials, the contribution to energy production from glucose oxidation increased at a similar rate and rose to 22 ± 2% mJ at exhaustion in the cycle ride.

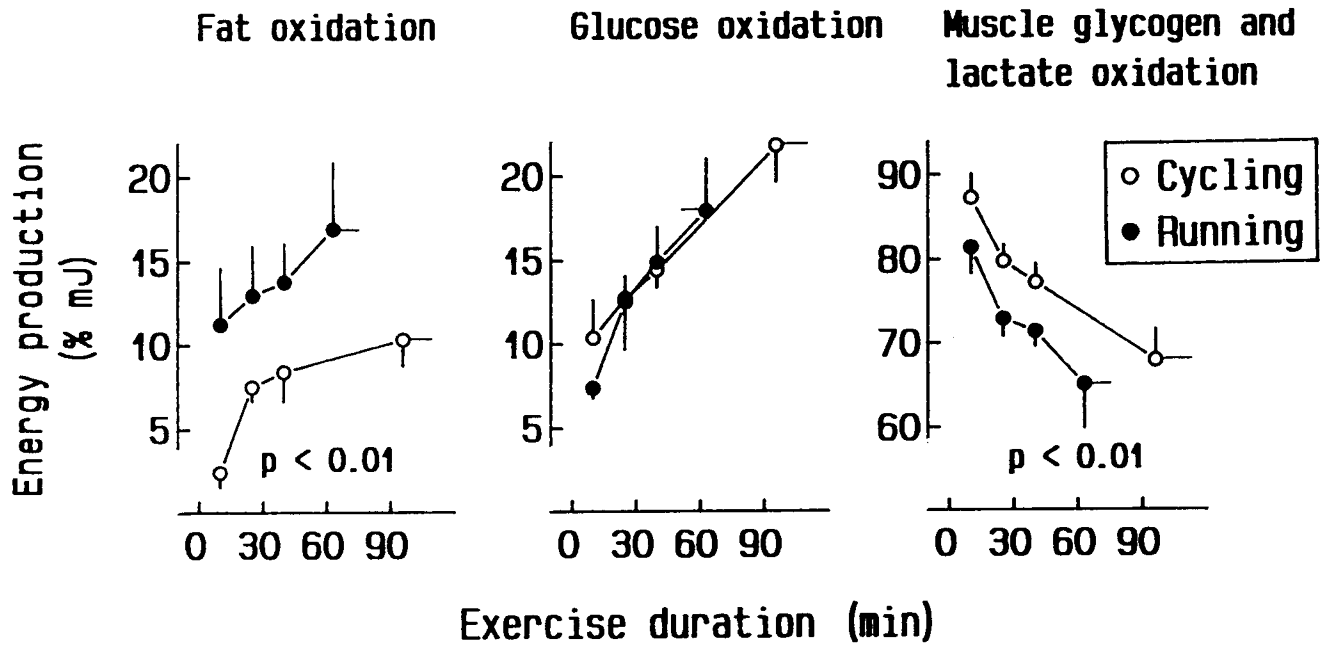

Figure 7 The contribution to total energy production from fat oxidation, glucose oxidation and muscle glycogen and lactate oxidation during cycling

and running to exhaustion at 80% of maximal oxygen uptake when ingesting carbohydrate

4.7 Estimated muscle glycogen and lactate oxidation

Instead, differences between the rates of total CHO oxidation and rates of glucose oxidation suggested that the direct or indirect (via lactate) oxidation of muscle glycogen or both, was greater in cycling than in running (Figure 4). During the first 40 min of exercise, the estimated contributions to energy production from muscle glycogen oxidation decreased from 87 ± 3% to 77 ± 2% mJ in the cycling trial and from 81 ± 3% to 71 ± 2% mJ in the running trial. All between trial differences for estimated contributions to energy production from muscle glycogen oxidation were highly significant ($P < 0.01$).

At exhaustion, however, the estimated contribution to total energy production from muscle glycogen oxidation was similar in the two trials. Subjects could no longer continue running or cycling when ~65% of their energy came from muscle glycogen and lactate oxidation.

CHAPTER FIVE

DISCUSSION

5.0 DISCUSSION

The first finding of the current study was that well trained triathletes exercising at the same *absolute* intensity (80% of VO_{2peak}) became exhausted significantly earlier when running compared to cycling ($P < 0.05$; Figure 1). Traditionally, VO_{2max} values for cycling are 7-11% lower than those recorded for running (Hermansen and Saltin 1969; Koyal et al. 1976, Roalstad, 1989). However, it is possible that the subjects chosen in this study were equally well trained in both disciplines, as exhibited by their identical VO_{2peak} values. The lower VO_{2peak} values recorded for cycling than for running in single-sport athletes are typically due to a smaller muscle mass being recruited when cycling than running (Hermansen and Saltin 1969). The values presented in this study are in keeping with others, who have shown differences in VO_{2peak} values of only three to six percent between running and cycling (Roalstad 1989).

The exercise time to fatigue while running for the subjects in the current study (~60 min) is shorter than that reported by Wilber and Moffat (1992) for ten male distance runners exercising at the same *relative* intensity while ingesting either water or CHO (92± 27 min versus 115± 25 min respectively). However, it should be noted that Wilber and Moffat (1992) utilised one minute rest periods during their exercise protocol. It is possible that this could be the cause for the prolonged running times to fatigue at high intensities (80% of VO_{2peak}) reported in that study. Others (Madsen et al. 1990) have reported similar running times (~70 min) to exhaustion at ~80% of VO_{2max} as that found in the current investigation. Indeed, only when running at a much lower exercise intensity (70% of VO_{2max}) do performance times appear to approach 2 hr (Tsintzas et al. 1993b).

Previous studies have shown that exhaustion during prolonged, continuous, moderate-intensity (<75% of VO_{2max}) exercise often coincides with hypoglycaemia in overnight fasted subjects (Coyle et al. 1983, 1986; Felig et al. 1982) and critically low muscle glycogen concentrations in fed subjects (Bosh et al. 1993a). It has been proposed that CHO ingestion during cycling postpones the onset of fatigue primarily by maintaining plasma glucose concentrations and high rates of CHO oxidation late in exercise (Coggan and Coyle 1991). However, CHO ingestion has failed to improve exercise performance during some (Noakes et al. 1988b; Riley et al. 1988; Sasaki et al. 1987) but not all (Gordon et al. 1925; Tsintzas et al. 1993a; Williams et al. 1990) studies of endurance running.

In the present study, subjects became exhausted during cycling in a euglycaemic state (~5 $mmol.l^{-1}$), whilst during running they experienced a gradual increase in plasma glucose concentration throughout exercise, with modest hyperglycaemia (7.0 $mmol.min^{-1}$) at the point of exhaustion (Figure 5). Such hyperglycaemia during intense running has been reported by others (Edwards and Hopkins, 1993; Wilber and Moffat 1992), and is partially due to a mismatch between hepatic glycogenolysis and muscle glucose uptake (Ahlborg et al. 1974), particularly at the onset of exercise (Wahren et al. 1971). Coggan (1991), hypothesised that athletes performing cycle ergometer exercise appear to be more susceptible to a decline in plasma glucose concentration than athletes performing prolonged running.

Despite the significantly higher plasma glucose concentrations during the running than the cycling trial there were no differences in either the rates of total blood glucose oxidation, or ingested glucose oxidation (Figure 6). The rates of total blood glucose oxidation in the present study (~1 g.min^{-1}) are highly comparable with other investigations with trained subjects cycling at either 55% (Rauch et al. 1995) or 70% of $V0_{2max}$ (Bosch et al. 1993a; Broberg and Sahlin 1989; Hawley et al. 1994). However, the peak rates of ingested glucose oxidation observed in the current study (~0.5 g.min^{-1}) are much lower than those typically measured during prolonged, moderate-intensity exercise (Bosch et al. 1993a; Hawley et al. 1992, 1994; Rauch et al. 1995). In those studies ingested glucose oxidation typically peaks at ~1.0 g.min^{-1} (for review see Hawley, Dennis and Noakes 1992) and contributes approximately 20% of total CHO oxidation. It is likely that the higher relative work rate of the subjects in the current study may have reduced splanchnic blood flow and intestinal absorption as has been previously proposed (Barclay and Turnberg 1987; Maughan et al. 1990; Williams et al. 1964). In support of this hypothesis, Pirnay et al. (1982) observed that as exercise intensity increases above ~50% of VO_{2max}, ingested CHO oxidation plateau's, suggesting that the delivery of ingested glucose to the blood becomes limiting during intense exercise.

While none of the subjects complained of severe gastro-intestinal (GI) discomfort during the cycle ride, five of the six subjects felt nauseous at the end of the run. Although distance runners often complain of GI problems during competition, with the prevalence often being as high as 50% (Rehrer et al. 1989, 1990), there is little documentation of these symptoms in cyclists (Rehrer et al. 1990; Peters et al. 1993). However, it is unlikely that differences in gastric emptying are responsible for the higher incidence of GI distress in runners. Houmard et al. (1991) reported similar rates of gastric emptying for well-trained biathletes during intense (75% of VO_{2max}) running and cycling. This suggests that the running action *per se* brings about mechanical disturbances in normal GI function, compared to cycling, where the body remains relatively stable (Rehrer et al. 1991) and intra abdominal pressures are not increased to the same degree as during running (Noakes 1992).

With no significant difference in either the total or ingested rates of glucose oxidation, it is somewhat paradoxical that the rate of total CHO oxidation was significantly higher during cycling than running (Figure 6). As would be expected, the elevated CHO oxidation associated with cycling was accompanied by a concomitant reduction in the rate of fat oxidation (Figure 7), which strongly suggests that the direct or indirect (via lactate) oxidation of muscle glycogen, or both, may have been greater in cycling than in running. Interestingly during both the running and cycling trials, subjects were no longer able to maintain the desired work rate when the estimated contribution to the overall energy requirements of exercise from muscle glycogen and lactate oxidation fell below 65% of total energy production. However, without muscle biopsy data we are able only to speculate as to the mechanism(s) causing exhaustion during such sustained high-intensity exercise.

Previous investigations which have employed prolonged, moderate-intensity cycling (Bosch et al. 1993a; Coyle et al. 1986) and running (Tsintzas et al. 1993b) have observed that fatigue occurs when muscle glycogen concentration reaches critically low levels (usually <40 $mmol.kg^{-1}$ w.w.), despite the maintenance of euglycaemia (a plasma glucose concentration of

5 mmol.l^{-1}). Thus, there may be an obligatory utilisation of a certain amount of endogenous CHO to maintain citric acid cycle intermediates at a level needed to support the oxidative capacity of the working muscle as has been hypothesised by Spencer et al. (1992).

CHAPTER 6

CONCLUSION

6.0 Conclusion

In conclusion, the results of this thesis show that well trained triathletes exercising at the same absolute intensity (80% of VO_{2max}) became fatigued significantly earlier when running compared to cycling, despite high rates of blood glucose oxidation and the maintenance of high rates of CHO oxidation at the point of exhaustion. Further, during both the running and cycling trials, subjects were no longer able to maintain the desired exercise intensity when the estimated contribution from muscle glycogen (and lactate) oxidation to the overall energy requirements of the exercise task fell to below ~65% of total energy production. Although the oxidation of blood glucose may be able to sustain high rates of CHO oxidation during prolonged, moderate-intensity (70% of VO_2max) exercise, it may well be that some small, but critical requirement for muscle glycogen exists during sustained, high-intensity exercise. Further research will be required to test this hypothesis, and to elucidate the mechanisms of fatigue during running and cycling when blood glucose concentration is maintained by feeding CHO during such exercise.

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CHAPTER 8

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