The effect of fructose and maltodextrin vs glucose and maltodextrin formulated sports beverages on mountain-bike race performance

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Abstract

Background: Exogenous carbohydrate improves performance during prolonged high-intensity exercise. When ingested together, fructose and glucose polymers are oxidised at rates 1.5-1.7 higher than isocaloric glucose solutions. As fructose and glucose are transported across the intestine via different mechanisms, the capacity for exogenous-carbohydrate absorption is greater with composite carbohydrate mixtures. Therefore, since the effect of ingesting multi-transportable carbohydrate on field-based performance has to our knowledge not been investigated, we examined their effect on mountain bike race performance. Finishing time was expected to be substantially reduced when multi-transportable carbohydrates were ingested.

Method: Ten; male (7) and female (3), mountain bikers aged 32.9 ± 8.7 years, weighing 68.8 ± 9.4 kg and training for at least 8 hours per week and racing regularly participated in a double-blind crossover study. Following a standardised training and diet regimen cyclists completed two Olympic-distance (target winning time of 2h 15m), cross-country mountain bike races during which they ingested either a 11.25% maltodextrin and fructose solution (MF) or an isocaloric, equi-volumetric, isosmotic control solution containing maltodextrin and glucose (MG). Performance times, ratings of perceived exertion, gastro-intestinal discomfort and measurements of hydration status were recorded and compared. Data was analysed using appropriate mixed models in SAS.

Results: Cyclists were 1.8% (2mins 31s) faster in MF compared to MG (90% confidence interval: ±1.8%; 72% likelihood of a substantial benefit). The effect solution composition on the increase in time from the first the final lap (fatigue) was 9.7% (±2.8%) in MF and 10.7% (±2.8%) in MG; which corresponded to a 0.9% reduction (±3.5%; unclear) in the fatigue in MF. Abdominal cramps were reduced by 8.1% in MF relative to MG (±6.6%; likely benefit) and for every 1% change in abdominal cramp rating, lap time increased by 0.14% (±0.10%). There we no clear effects of MF on ratings of perceived exertion and hydration status compared with MG.

Conclusion: Cross-country mountain bike race performance was substantially enhanced following ingestion of a maltodextrin and fructose solution. This outcome was related to reduced gastro-intestinal distress supporting the theory that solutions containing multiple-transportable carbohydrates increase the availability of carbohydrate for metabolism. Further investigation with a larger sample size is recommended to establish whether the performance effect is genuinely beneficial or trivial.

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Introduction

Fluid and carbohydrate both independently and together improve exercise performance (Below, et al., 1995). According to Williams, (1989), exercise performance refers to the time taken to perform a set task or distance, whereas exercise capacity refers to the time taken to exercise to exhaustion at a constant intensity. A lack of fluid intake during exercises causes dehydration which may impair thermoregulation (Fortney, et al., 1981; Nadel, et al., 1980; Sawka, et al., 1985), cardiovascular function (Hamilton, et al., 1991; Montain and Coyle, 1992b; Walsh, et al., 1994) and exercise performance (Armstrong, et al., 1985; Barr, et al., 1991; Walsh, et al., 1994); however, when fluid is ingested these impairments can be attenuated (Barr, et al., 1991; Below, et al., 1995; Hamilton, et al., 1991; Maughan, et al., 1996).

Under normal post-absorptive metabolic conditions, exercise at a high intensity relies heavily on endogenous-carbohydrate stores (Gollnick, 1985; Saltin and Karlsson, 1971). As muscle glycogen is depleted, more glucose is extracted from the blood (Gollnick, et al., 1981) and there is an increase in hepatic-glucose output to maintain blood-glucose concentrations (Astrand and Rodahl, 1986). Depleted muscle- and liver-glycogen and an inability to maintain blood glucose are considered to be primary causes of fatigue under these circumstances (Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983). Ingestion of carbohydrate during prolonged exercise, however, can delay fatigue and improve both endurance capacity (Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; Fielding, et al., 1985; Hargreaves, et al., 1984; Maughan, et al., 1989; Mitchell, et al., 1989; Tsintzas, et al., 1996b; Yaspelkis, et al., 1993) and the performance of constant-work tasks (Tsintzas, et al., 1995b) by reducing endogenous-carbohydrate oxidation (Bosch, et al., 1996; Coyle, et al., 1983; Hargreaves, et al., 1984; Jeukendrup, et al., 2006; Jeukendrup, et al., 1999b; McConnell, et al., 1994; Nicholas, et al., 1999; Palmer, et al., 1999; Tsintzas, et al., 1995a, 1996a; Yaspelkis, et al., 1993). Carbohydrate ingestion may also improve performance by reducing central fatigue (Dalsgaard, et al., 2002; Davis, et al., 1992; Nybo, et al., 2005; Nybo, et al., 2003; Snow, et al., 2000). The peak oxidation rates of different types of

exogenous carbohydrate vary with the highest rates of ~1.0 $g \cdot min^{-1}$ (Jeukendrup, et al., 1999b; Rehrer, et al., 1992b; Wagenmakers, et al., 1993) observed following ingestion of glucose or glucose polymers at a rate of 1.2 $g \cdot min^{-1}$ or greater (Hawley, et al., 1992a; Jentjens, et al., 2004c; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Jeukendrup, et al., 1999b; Wagenmakers, et al., 1993; Wallis, et al., 2005). Fructose is oxidised at lower rates than glucose (Adopo, et al., 1994; Guezzenec, et al., 1989; Massicotte, et al., 1986; Massicotte, et al., 1989) when ingested alone. However, fructose absorption appears to be facilitated when it is co-ingested with glucose (Adopo, et al., 1994; Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Shi, et al., 1997; Shi, et al., 1995; Wallis, et al., 2005). Exogenous-carbohydrate oxidation rates are 40-65% higher following co-ingestion of glucose or maltodextrin and fructose at rates of 1.2 $g \cdot min^{-1}$ and 0.6-1.2 $g \cdot min^{-1}$ respectively, relative to an isocaloric quantity of glucose or maltodextrin (Jentjens and Jeukendrup, 2005; Wallis, et al., 2005). However, ingestion of large quantities of carbohydrates without increasing fluid intake increases the solution concentration and osmolality which may reduce fluid uptake (Brouns and Kovacs, 1997; Rehrer, 1994) and increase the likelihood of gastro-intestinal discomfort (Tsintzas, et al., 1995b; Wagenmakers, et al., 1993).

Most studies into the performance effects of carbohydrate supplementation during prolonged endurance exercise have been completed in the laboratory when the athlete has fasted for a prolonged period of time (Jeukendrup, 2004) giving them initially depleted liver-glycogen stores. These conditions do not accurately represent the demands and preparation surrounding competition. Furthermore, to my knowledge no research has looked at the affect of co-ingestion of fructose and glucose-based carbohydrate at high rates on performance. Therefore, the first aim of this study is to investigate the effect of ingesting maltodextrin and fructose at high rates during prolonged, high-intensity exercise performance in the field under normal competition conditions compared to an equicaloric maltodextrin and glucose solution. Performance will be evaluated during an Olympic-distance, cross-country mountain bike race which due to its intermittent and high-intensity nature (Impellizzeri, et al., 2002;

Lee, et al., 2002; Stapelfeldt, et al., 2004) likely relies on carbohydrate as the predominant fuel source (Saltin and Karlsson, 1971). Carbohydrates will be ingested at rates similar to those used previously in laboratory studies to attain high exogenous-carbohydrate oxidation rates (Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Wallis, et al., 2005) and mixed with a quantity of fluid that meets normal recommended rates of fluid ingestion for prolonged exercise (Noakes, 1995; Speedy, et al., 2000). An appropriate quantity of salt will also be included to aid in the stimulation of thirst (Astrand and Rodahl, 1986) and increase both fluid (Wemple, et al., 1997) and carbohydrate (Leiper, 1998; Olsen and Ingelfinger, 1968) uptake. A second aim will be to examine any effect of the different drink compositions on ratings of perceived exertion and gastro-intestinal discomfort. Thirdly, we will investigate whether ingestion of these drink formulations affects dehydration by recording measurements of hydration status (urinary indices and change in body mass). Urine colour has been shown to correlate to more accurate urinary indices of hydration status (Armstrong, et al., 1994) and can be used to monitor hydration in the field to help prevent dehydration. Therefore the final aim is to compare the validity of measurements of urine colour, urine specific gravity and urine osmolarity with those of a previous benchmark study (Armstrong, et al., 1994).

We expect to observe a reduction in performance time and less symptoms of gastro-intestinal discomfort with ingestion of the maltodextrin and fructose drink compared to the maltodextrin and glucose drink. Additionally, we do not expect the cyclists to become unusually dehydrated.

Literature Review

Dehydration and low levels of endogenous carbohydrate are associated with decrements in exercise capacity and performance and when fluids and carbohydrate are ingested these impairments can be attenuated (Armstrong, et al., 1985; Below, et al., 1995; Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; Hamilton, et al., 1991; Hargreaves, et al., 1984; Maughan, et al., 1996; Tsintzas, et al., 1993; Walsh, et al., 1994). This review will cover the mechanisms by which carbohydrate and fluid ingestion facilitate exercise capacity and performance and the degree to which they are enhanced. The review will also discuss the present state-of-understanding of the physiology of carbohydrate digestion and absorption and the resulting current recommendations for the composition of fluids to be taken during prolonged exercise. As the intention of this study is to test a carbohydrate drink formulation on performance during a cross-country mountain bike race, I will start by reviewing the physiology and nutritional demands of cross-country mountain bike racing to show why this mode of exercise is particularly relevant in testing the effectiveness of a sports drink formulation containing multi-transportable carbohydrates.

1. Mountain Bike Racing

There are two disciplines of mountain bike racing: downhill and cross-country racing. Downhill mountain biking involves cyclists starting from a given high point and riding as fast as possible down steep and technical terrain which often involves a narrow single track with rocky areas, tree roots, tight corners, trees, branches and drop-offs all of which must be negotiated successfully with a minimal loss of speed. The winner is the person that makes it to the bottom of the run in the shortest period of time. Races often take between 45 seconds and 2 minutes and there is often transport to the top of the run such as a ski lift or vehicle access. Downhill riders wear almost complete body protection including shin guards, thigh pads, arm guards, chest plates and helmets to

protect the head, face and jaw. As with downhill skiers, downhill mountain bikers start one person at a time.

Cross-country mountain biking takes place on an undulating course, of which the rider usually completes several laps. Races usually involve a mass-start and the winner is the rider who completes the course in the shortest time. The race duration varies with course and race conditions but an Olympic Cross-country course according to the International Cycling Union (UCI) is characterised by a winning time of 2.00-2.15 hours and 1.45-2.00 hours for elite men and women, respectively, is between 5 and 9km in length and involves significant ascending and descending (International Cycling Union, 2006). Different countries often host longer endurance-type races that are not part of National or World Cup series endurance events; for example, the 2.5–4 hour Karapoti, New Zealand and the longer 12-24 hour ultra-endurance events. Uphill sections of cross-country races can be steep and last from seconds to several minutes and downhill sections, whilst not generally as technical as sections of downhill race course; do demand a significant skill level of participants. Cross-country racers must have high levels of aerobic and anaerobic endurance in addition to technical ability (Impellizzeri, et al., 2002; Lee, et al., 2002; Stapelfeldt, et al., 2004).

1.1 Physiological demands of cross-country mountain bike racing

As mentioned earlier, downhill mountain bike racing whilst being physically demanding in its own right, relies to a small degree on physiological endurance capacity. Because this paper is concerned with the effects of carbohydrate ingestion on endurance performance, the focus for this review in respect to mountain bike racing will be on cross-country mountain biking. The physiological requirements of cross-country mountain bike racing emphasise the role of carbohydrates as the main substrate for energy metabolism.

Competitive mountain bike racing is a highly intensive sport demanding the production of high power outputs and requires a high aerobic capacity and anaerobic tolerance. Stapelfeldt et al. (2004) measured power output of 11 elite

mountain bikers over a series of races in one competitive season. Power outputs varied with the profile of the courses but average power output was 246W (3.6 $W \cdot kg^{-1}$) for males and 193W (3.1 $W \cdot kg^{-1}$) for females. The highest power outputs measured during the climbs were between 250 and 500W lasting from a few seconds to several minutes. These power outputs were above those measured during laboratory testing at the individual's anaerobic threshold for most cyclists. In comparison, professional road cyclists produce average power outputs between 325 and 450W for time trial events of 5-70km in length and during the long (~200 km) stage rides of mass start road races, observations of average power outputs between 150 and 300W have been noted (Jeukendrup, et al., 2000).

Several studies have measured the physiological characteristics of off-road cyclists (Baron, 2001; Impellizzeri, et al., 2005; Impellizzeri, et al., 2002; Lee, et al., 2002; Stapelfeldt, et al., 2004; Wilber, et al., 1997) and road cyclists (Lucia, et al., 2000; Padilla, et al., 1999). The direct comparison of values from these studies is difficult as each used different methodology to establish power outputs and different levels of cyclist varying from those at a National level to World class. Two studies (Lee, et al., 2002; Wilber, et al., 1997) in which off-road and road cyclists have been compared directly show similar absolute values for maximal power between off-road and elite road cyclists (420W and 470W (Wilber, et al., 1997) and 413W and 431W (Lee, et al., 2002) respectively). The relative maximal power of off-road cyclists is also similar to that of road cyclists (5.9 $W \cdot kg^{-1}$ and 6.5 $W \cdot kg^{-1}$ (Wilber, et al., 1997) 6.3 $W \cdot kg^{-1}$ and 5.8 $W \cdot kg^{-1}$ (Lee, et al., 2002) for off road and road cyclists respectively).

High VO_{2max} values together with an ability to maintain exercise at a high relative intensity as indicated by parameters such as the lactate threshold, respiratory compensation point or the anaerobic threshold, are also essential to mountain biking. Where the VO_{2max} of elite mountain bike racers has been measured (Baron, 2001; Impellizzeri, et al., 2005; Impellizzeri, et al., 2002; Lee, et al., 2002; Stapelfeldt, et al., 2004; Wilber, et al., 1997), values have ranged between 66.5–78.3 $ml \cdot kg^{-1} \cdot min^{-1}$. These values are again similar to those of elite and professional road cyclists (Faria, et al., 2005; Lucia, et al., 2000; Padilla, et

al., 1999), which are considered to be amongst the highest recorded values. It is difficult to compare values of the highest relative sustainable intensity because most of the authors have used different measurements. The off-road cyclists in these studies had average measures of 75.3-83.8% VO_{2max} for lactate threshold (Impellizzeri, et al., 2002; Wilber, et al., 1997), 87.0% VO_{2max} for the onset of blood lactate accumulation (Impellizzeri, et al., 2002), 73.8% VO_{2max} for ventilatory threshold (VT) (Impellizzeri, et al., 2005) and 87% VO_{2max} for respiratory compensation point. These values compare to those of professional road cyclists; ~77 and 86% VO_{2max} for LT and OBLA respectively (Mujika and Padilla, 2001) and 72.6 ~90% VO_{2max} for VT_1 and VT_2 respectively (Lucia, et al., 2000).

In two studies (Impellizzeri, et al., 2002; Stapelfeldt, et al., 2004) the physiological stress experienced during elite mountain bike racing has been measured by comparing power (Stapelfeldt, et al., 2004) and heart rate (Impellizzeri, et al., 2002; Stapelfeldt, et al., 2004) readings from performance with data collected in the laboratory. Stapelfeldt et al. (2004) measured the time spent at different intensities by elite mountain bike racers, during the competitive season. Intensities were divided into 4 zones; zone 1 was below aerobic threshold (AT), zone 2 was between AT and individual anaerobic threshold (IAT), zone 3 was between IAT and exhaustion (max) and zone 4 was above max. The average time spent in each zone was 39%, 19%, 20% and 22% in zones 1-4 respectively and values were similar between individuals and races. Throughout the races the average heart rate was 104% of the heart rate at individual anaerobic threshold and 91% of heart rate registered at exhaustion during laboratory testing. The latter finding is similar to that of Impellizzeri et al. (2002) in which the average heart rate during races was 90% of the maximum heart rate which corresponded to 84% VO_{2max}. These measured characteristics provide evidence that the physiological demands on mountain bike racers require high levels of aerobic and anaerobic conditioning and are illustrated by a high threshold power.

1.2. Nutritional demands of mountain bike racing

From the evidence explained above, it is clear that cross-country mountain-bike racing is high-intensity intermittent-type exercise. High-intensity exercise requires carbohydrate because transport saturation kinetics (Jeukendrup, 2002) and possibly an inadequate oxygen supply limit the oxidation of fatty acids (Astrand and Rodahl 1986) to 0.6-1.0 $g \cdot min^{-1}$ in trained cyclists (Rowlands and Hopkins, 2002) and the oxidation of pyruvate is limited by mitochondrial respiratory capacity to perhaps on average 4-5 $g \cdot min^{-1}$ (Rowlands and Hopkins, 2002); the remaining energy for work above these rate equivalents must come from carbohydrate-dependent anaerobic glycolysis. Anaerobic glycolysis causes rapid and significant depletion of muscle glycogen (Astrand and Rodahl, 1986). Mountain bike racing at an intensity represented by an average of 90% maximum heart rate, would equate to approximately 82% VO_{2max} (Londeree, et al., 1995) and would therefore rely on muscle glycogen as the predominant fuel source (Saltin and Karlsson, 1971). Because muscles with low glycogen content extract more glucose from the blood (Gollnick, et al., 1981) as glycogen stores become depleted, there will be a greater reliance on blood glucose to fuel exercise. As the rate of blood-glucose oxidation increases, liver glycogenolysis or gluconeogenesis increase hepatic-glucose output in order to maintain blood-glucose concentrations (Astrand and Rodahl, 1986). If hepatic-glucose output is unable to maintain blood-glucose concentration due to depleted liver glycogen or a reduced rate of gluconeogenesis, fatigue may ensue (Astrand and Rodahl, 1986).

Since both glycogen depletion and reduced blood-glucose concentrations are considered to be a primary cause of fatigue in events longer than 1 hour, maximising the availability of ingested carbohydrate should be an important determinant of performance for the elite mountain biker.

1.3. Reliability of mountain bike racing performance

The performance of an athlete randomly varies between races and this variation is known as the within athlete variation or standard deviation of performance (Hopkins, et al., 1999). When expressed as a percentage of the mean performance, the within-athlete standard deviation is known as the coefficient of variation. For an experimental intervention to be beneficial to the performance of mountain bikers or indeed any other athlete, the smallest worthwhile effect must be at least about half (0.3-0.7) the magnitude of the coefficient of variation (Hopkins, et al., 1999). Paton and Hopkins (2006) collected data from the 2001 World Cup cross-country mountain bike races and using mixed linear modelling calculated the within-subject coefficient of variation to be 2.4 and 2.5% in the top quarter of male and female competitors respectively. The smallest worthwhile effect of any intervention in mountain-bike races was therefore calculated to be ~1.2% (Paton and Hopkins, 2006). This value is not only important in guiding interpretation into the effectiveness of an intervention but also in determining the minimum sample size necessary for research studies (Hopkins, 2000).

2. Exogenous Carbohydrate Ingestion and Exercise

2.1 The effect of exogenous-carbohydrate ingestion on exercise capacity and performance

Numerous studies have examined the effect of exogenous carbohydrate ingested during exercise and have shown improvements in both exercise capacity (Bjorkman, et al., 1984; Carter, et al., 2003; Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; El-Sayed, et al., 1997; Fielding, et al., 1985; Hargreaves, et al., 1984; Maughan, et al., 1989; Mitchell, et al., 1989; Tsintzas, et al., 1996b; Wilber and Moffatt, 1992; Wright, et al., 1991; Yaspelkis, et al., 1993) and performance (Angus, et al., 2000; Below, et al., 1995; Jeukendrup, et al., 1997; Tsintzas, et al., 1993; Tsintzas, et al., 1995b; Williams,

et al., 1990). For a summary of studies see Tables 1 and 2. Whilst many of the studies using exercise of longer than two hours duration (Angus, et al., 2000; Carter, et al., 2003; Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; Fielding, et al., 1985; Hargreaves, et al., 1984; Maughan, et al., 1989; Tsintzas, et al., 1993; Tsintzas, et al., 1995b; Tsintzas, et al., 1996b; Williams, et al., 1990; Wright, et al., 1991; Yaspelkis, et al., 1993) have shown the beneficial effects of carbohydrate ingestion, the ergogenic effects for shorter duration exercise are less clear (Below, et al., 1995; Clark, et al., 2000; El-Sayed, et al., 1997; Flynn, et al., 1987; Jeukendrup, et al., 1997; McConnell, et al., 2000; Mitchell, et al., 1989; Palmer, et al., 1998; Wilber and Moffatt, 1992).

Angus et al. (2000) observed a 6.7% improvement in performance when cyclists completed a 100-km time trial during which they ingested a 6% carbohydrate drink at a rate of 1 $L \cdot h^{-1}$ providing 60 $g \cdot h^{-1}$ of carbohydrate. Time to complete the time trial was 166 and 178 minutes when carbohydrate and placebo drinks were ingested respectively. In contrast, an earlier study resulted in no beneficial effect of carbohydrate ingestion at a rate of 66 $g \cdot h^{-1}$ during 100-km cycling time trial (Madsen, et al., 1996). Angus et al. suggested that a difference in pace over the first 2 hours of exercise resulted in a lower contribution of exogenous-carbohydrate to total-carbohydrate oxidation in the study of Madsen et al. Participants of Angus et al. averaged an initial exercise intensity of 75% VO_{2max} compared to less than 70% VO_{2max} by those of Madsen et al. The lower intensity would reduce the reliance on carbohydrate for energy provision as was evidenced by respiratory exchange ratios recorded (0.95 and 0.89 for Angus and Madsen respectively) in turn decreasing the effectiveness of any additional carbohydrate ingested.

In the studies where carbohydrate ingestion has benefited exercise capacity (time to exhaustion at a constant submaximal power) and performance, there has been a range of improvement time in minutes between 1.6 - 33% and 2 - 7% respectively. Studies which have used an exercise pre-load methodology (Below, et al., 1995; Coggan and Coyle, 1987; Fielding, et al., 1985; Hargreaves, et al., 1984; Murray, et al., 1987a; Murray, et al., 1989) such as exercise-time to exhaustion or time to complete a sprint following a previous

bout of exercise show much larger improvements (46–160% and 4.4-15.2% in exercise capacity and performance respectively) however, these improvements do not reflect the total workload completed as they are calculated using data from the final bout of exercise alone.

Major difficulties exist in comparing most of these studies due to differences in exercise modes used, the type, quantity and timing of carbohydrate provision and the methods by which improvements were evaluated. Different authors have measured exercise capacity by sprint time following 3-4 hours exercise, time to exhaustion at a constant work rate and the amount of work completed in a set time period (Table 2). Researchers investigating cycling performance have generally used simulated races and time trials over set distances (Table 1). Other aspects of the methodologies such as pre-exercise diet, pre-exercise carbohydrate stores, environmental conditions, competitive and training status of participants, feedback given to participants, motivation of participants, the number of participants involved in the study and methods of statistical analysis may also contribute to the outcomes reported. However, results from the majority of studies indicate a facilitative effect of exogenous carbohydrate on both exercise capacity and performance.

2.2. Forms of carbohydrate

It is common for athletes to ingest carbohydrate in the form of solid food (fruit, energy bars) during exercise, particularly that of long duration (Burke, 2002; Clark, et al., 1992; Garcia-Roves, et al., 2000; Glace, et al., 2002; Kimber, et al., 2002; Rehrer, et al., 1992a; Saris, et al., 1989; White, et al., 1984). Solid carbohydrate may be psychologically more appetising, may be carried with greater ease than large quantities of fluid as solid food have are more energy dense and may provide a steadier supply of carbohydrate as they are gradually digested in the intestine. However, both the potential increased time of digestion which could reduce the availability of the carbohydrate as a ready substrate and the additional requirement for fluid may make solid food a less attractive carbohydrate source for most high-intensity endurance sports.

Table 1. The effect of carbohydrate ingestion during exercise on performance (effect magnitude is improvement in minutes expressed as %)

Study	Exercise	Type of CHO	Ingestion rate $g \cdot h^{-1}$	n	Performance effect vs placebo	Effect (%)
Less than 120mins						
Below et al. (1995)	50mins at 80% VO_{2max} prior to set amount of work as fast as possible	40% MD-e	79	8	10.2min (CHO), 10.9min (no-CHO)	Y – 6.8%
Jeukendrup et al. (1997)	Set amount of work as fast as possible	7.6% CHO-e	~73.2	19	58.7min (C-e), 60.2min (P)	Y – 2.5%
Palmer et al. (1998)	20km Cycle Time trial	6.8% CHO	20	14	28min (CHO), 28min (P)	N
Clark et al. (2000)	40km Cycle Time trial	7.6% GP	90	43	Decrease in power of 0.3%	N
More than 120mins						
Murray et al. (1987a)	Intermittent cycling (55-65% VO_{2max}) followed by time to complete a sprint	5% GP	24	13	400s (GP), 432 s (P)	N
		4% S + 2% G	29		384s (S+G), 432 s (P)	Y – 12.5%s
		5% GP + 2% F	34		375s (GP+F), 432s (P)	Y – 15.2%s
Murray et al. (1989)	3 bouts of 20mins cycling at 65% VO_{2max} followed by time to complete a sprint	6% S	23	12	13.03s (6%), 13.6s (P)	Y – 4.4%s
		8% S	30		13.30s (8%), 13.6s (P)	N
		10% S	38		13.57s (10%), 13.6s (P)	N
Williams et al. (1990)	30km Running as fast as possible	70g G	35	9	124.8min (G), 129.3min (W)	Y – 3.5%
		70g F	35		125.9min (F), 129.3min (W)	Y – 2.6%
Tsintzas et al. (1993)	30km Running as fast as possible	50g CHO (GP+F)	1 x 50g	7	128min (CHO), 131min (W)	Y – 2.3%
Tsintzas et al. (1995)	42.2km (Marathon) Running as fast as possible	5.5% CHO	19.5	7	190min (5.5), 194min (W)	Y – 2%
		6.5% CHO	24.5		192min (6.5), 194min (W)	N
Madsen et al. (1996)	100km Cycle Time trial	5% MD + G	66	9	160min (CHO), 160min (P)	N
Angus et al. (2000)	100km Cycle Time trial	6% G + S	60	8	166min (CHO), 178min (P)	Y – 6.7%

CHO-Carbohydrate, GP-Glucose Polymer, MD-Maltodextrin, G-Glucose, F-Fructose, S-Sucrose, W-Water, P-Placebo, w-watts, h-hour, Y- reported as statistically significant, $p<0.05$, N- Reported as not statistically significant, $p>0.05$, TT-Time trial, s - seconds

Table 2. The effect of carbohydrate ingestion during exercise on exercise capacity (effect magnitude is improvement in minutes expressed as %)

Study	Exercise	Type of CHO	Ingestion rate $g.h^{-1}$	n	Performance effect vs placebo	Effect (%)
Less than 120mins						
El-Sayed et al. (1997)	Cycling as far as possible in 1 hour	8% G Glucose	25	8	277W (G), 269W (P)	Y – 1.6%
Flynn et al. (1987)	120 min Cycling, trying to complete as much work as possible	3% GP + 2% G	45		184w (CHO), 186W (P)	N
		5% GP + 5% F	90		178w (CHO), 186W (P)	N
		7.7% GP + 2.3% F	90		189w (CHO), 186W (P)	N
Mitchell et al. (1989)	105min cycling at 70% VO_{2max} + 15min TT	6% CHO	37	10	213 kJ (6%), 201 kJ (P)	N
		12% CHO	74		228 kJ (12%), 201 kJ (P)	Y
		18% CHO	111		217 kJ (18%), 201 kJ (P)	N
Wilber & Moffat (1992)	Running at 80% VO_{2max} to exhaustion	7% G	41	10	115.4min (G), 92.0min (P)	Y – 25.4%
McConnell et al. (2000)	Cycling at 294W / 83%VO_{2max} to exhaustion	6% G	81	13	68min (G), 70min (P)	N
More than 120mins						
Coyle et al. (1983)	Cycling at 74% VO_{2max} to exhaustion	GP	60	10	157min (GP), 134min (P)	Y – 17.2%
Hargreaves et al. (1984)	Intermittent intensity cycling for 4 hours followed by sprint to exhaustion	Solid bar: 43gS , 9g fat, 3g protein & water	43	10	127s (Bar), 87s (P)	Y - 46%s
Bjorkman et al. (1984)	Cycling at 68% VO_{2max} to exhaustion	7% Glucose	53	8	137min (G), 116min (P)	Y – 18%
		7% Fructose			114min (F), 116min (P)	N
Fielding et al. (1985)	Intermittent intensity cycling for 4 hours followed by sprint to exhaustion	5% CHO in solid form with water	22	9	121s (CHO), 81s (P)	Y – 49%s
Coyle et al. (1986)	Cycled at 71% VO_{2max} to exhaustion	10% GP	101	7	4.02h (GP), 3.02h (P)	Y – 33%
Coggan & Coyle (1987)	2 x cycling at 70% VO_{2max} to exhaustion	50% CHO	1 x 210g	7	26min (CHO), 10min (P)	Y – 160%
Maughan et al. (1989)	Cycling at 70% VO_{2max} to exhaustion	G Syrup	213	6	79min (G), 70.2min (W)	N
		G-F Syrup	213		79.5min (G-F), 70.2min (W)	N
		F Syrup	213		65.6min (F), 70.2min (W)	N
		G-electrolyte	24		90.8min (G-e), 70.2min (W)	Y - 29%
D.A. Wright et al. (1991)	Cycling at 70% VO_{2max} to exhaustion	5% GP + 3% F	40	9	266min, 201min (P)	Y – 32%
Yaspelkis et al. (1993)	3 hours of intermittent intensity exercise followed by ride to exhaustion	10% CHO drink	54	7	223min (CHO), 202min (P)	Y – 10.4%
		25g CHO bar	50		224min (bar), 202min (P)	Y – 10.9%
Tsintzas et al. (1996)	Running at 70% VO_{2max} to exhaustion	5.5% CHO-e	1 h x 48	11	125 min (CHO), 110 min (P)	Y – 13.6%
Carter et al. (2003)	Cycled at 60% VO_{2max} to exhaustion	6.4% MD	75	8	14.5% improvement	Y
	Cycled at 73% VO_{2max} to exhaustion				13.5% improvement	

CHO-Carbohydrate, GP-Glucose Polymer, MD-Maltodextrin, G-Glucose, F-Fructose, S-Sucrose, W-Water, P-Placebo, w-watts, h-hour, Y- reported as statistically significant, $p<0.05$, N- reported as not statistically significant, $p>0.05$, TT- Time trial

Murdoch et al. (1993) examined the metabolic and performance effects of ingesting either solid or slurried carbohydrate food between two prolonged exhaustive exercise bouts. No differences in the blood-glucose response or time to exhaustion were found following the ingestion of either solid or slurried bananas and no differences were found in blood-glucose concentrations or performance times. Slurrying the bananas was expected to lower the glycaemic response to the bananas. It was suggested, however, that the high water content of bananas (~75%) makes them semi-solid rather than solid which may have made them structurally too similar to the solid banana to elicit a significantly different response and that further work should be completed with foods such as bars and cookies (Murdoch, et al., 1993).

Lugo et al. (1993) compared the effects of ingesting a commercially available sports bar, a carbohydrate solution and a mixture of the bar and the drink. It was postulated that the solid form of the bar may take a longer time to digest and additionally since the carbohydrate contained in a bar is also combined with fat and protein, that the rate of gastric emptying may also have been delayed (Lugo, et al., 1993). These factors may have reduced the availability of exogenous carbohydrate for oxidation. The energy derived from protein and fat varied between treatments although they were iso-energetic in respect to carbohydrate. Additionally, the volume ingested was equated with deionised water to account for differences in densities between the forms of carbohydrate. Results showed that blood-glucose and insulin responses were the same for all treatments as was total carbohydrate oxidation during 120 minutes of cycling at 70% VO_{2max}. Furthermore, there was no difference in time-trial performance which followed. The study concluded that with equal liquid ingestion, the metabolic and performance responses when carbohydrate is consumed as a liquid, a solid or both are similar during prolonged moderate intensity cycling. Yaspelkis et al. (1993) also found no difference in time to exhaustion when solid carbohydrate was ingested compared to a solution containing a similar amount of carbohydrate. Both the liquid and solid supplements increased time to fatigue following 200 minutes of variable intensity exercise compared to a flavoured water placebo. It would appear that the availability of carbohydrate when

ingested at rates of up to 60 $g \cdot h^{-1}$ is similar when ingested in either a solid or liquid form during exercise.

2.3. Oxidation rates of different types of carbohydrate

Numerous studies have shown that when carbohydrate is ingested during exercise, a proportion is absorbed and oxidised providing alternative substrate to endogenous-carbohydrate stores (Bosch, et al., 1994; Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Pallikarakis, 1986; Rehrer, et al., 1992b; Wallis, et al., 2005). The oxidation of ingested carbohydrate can be measured by carbon isotopic tracers (^{13}C, ^{14}C) enabling researchers to establish the ingestion rates that lead to peak oxidation rates. The presumption is that increasing the rates of exogenous-carbohydrate oxidation will improve performance by sparing endogenous-carbohydrate stores, maintaining glucose homeostasis or by some other unknown effect.

2.3.1. Glucose

Researchers have found differing oxidation rates of glucose ingested during exercise. Peak oxidation rates have varied between studies as a result of differing types and quantities provided, exercise intensity and duration. Exogenous-glucose oxidation rates between 0.7 and 1.18 $g \cdot min^{-1}$ have been observed in a number of different studies (Hawley, et al., 1992a; Jentjens, et al., 2004c; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Jeukendrup, et al., 1999b; Pallikarakis, 1986) and it has been suggested that there is an upper physiological limit for exogenous-glucose oxidation (Duchman, et al., 1997; Hawley, et al., 1992a). In a study during which 180 g glucose was ingested during 90 minutes cycling at 70% VO_{2max} the rate of exogenous-carbohydrate oxidation reached a plateau at approximately 1 $g \cdot min^{-1}$ despite an increasing carbohydrate concentration in the intestine (Hawley, et al., 1992a). The peak oxidation rate was 0.9 $g \cdot min^{-1}$ similar to that of Palikarakis et al. (1986). More recently, Jentjens et al. (2004a) observed peak exogenous-carbohydrate oxidation rates of 0.8 $g \cdot min^{-1}$ and 0.83 $g \cdot min^{-1}$ when medium-glucose (1.2 $g \cdot min^{-1}$) and high-glucose (1.8 $g \cdot min^{-1}$) solutions were ingested during 120 minutes of cycling at ~63% VO_{2max}. These results confirm a maximal rate of glucose

oxidation exists, after which additional glucose ingestion does not further increase exogenous-carbohydrate oxidation rates.

2.3.2. Maltose

There have been few studies examining the oxidation of the two-glucose disaccharide maltose but in those available it appears that maltose is oxidised at the same rates as glucose, implying rapid hydrolysis. Hawley et al. (1992a) recorded the oxidation rates of 180 g of maltose or glucose during 90 minutes of cycling at 70% VO_{2max}. Both the maltose and glucose were oxidised at similar rates, 0.9 and 1.0 $g \cdot min^{-1}$ respectively and both accounted for ~20% of total carbohydrate oxidation. These findings are similar to those of Jentjens et al. (2004b) in which exogenous-carbohydrate oxidation rates were measured following ingestion of a combined glucose and maltose (1.2 and 0.6 $g \cdot min^{-1}$ respectively) solution compared to an isoenergetic amount of glucose with participants cycling at ~60% VO_{2max} for 150 minutes. The exogenous-carbohydrate oxidation rates were similar for the two solutions throughout exercise. These findings would suggest that maltose and glucose are digested and absorbed at the same rate and are equally available for oxidation. An application of maltose over glucose is a halving of solution osmolality.

2.3.3. Fructose

Fructose is a monsaccharide that is commonly used in sports drinks on account of its sweetness and flavour-enhancing properties. The insulin response to fructose is less that that of glucose (Samols and Dormandy, 1963) and therefore reduces the suppression of lipolysis. For these reasons and because of it's abundant cheap supply researchers and industry have been particularly interested in investigating its significance as a substrate in sports drinks.

Massicotte et al (1986) observed decreased exogenous-carbohydrate utilisation when fructose was ingested compared to glucose during 180 minutes cycling at 50% VO_{2max}. Blood-glucose concentrations were lower in the fructose trial than the glucose trial and it was suggested that this represented a slow rate of

conversion of fructose to glucose by the liver. However, blood glucose was similar in the last 60 minutes. As in the report of Samols and Dormandy (1963), the insulin response was lower and fat utilisation higher during the fructose trial. Endogenous-carbohydrate oxidation was similar in both trials indicating that greater fat use made up for the reduction in exogenous-carbohydrate oxidation during the fructose trial. Of the 140 g ingested, 106 g of the glucose and 79 g of the fructose were oxidised.

Further investigation (Massicotte, et al., 1989) compared the oxidation of fructose with that of glucose and a glucose polymer over 120 minutes of cycling at ~50% VO_{2max}. A 7% carbohydrate solution was ingested every 20 minutes throughout exercise. The fructose provided significantly less of the total energy requirement (~13.0%) compared to glucose (~17.6%), with only 53 g of the initial 98.9 g being oxidised compared to 70 g and 64 g of the glucose and glucose polymer respectively. These results are in agreement with those of Adopo et al. (1994) in which participants were given 50 or 100 g of glucose or fructose in 500 ml water during 120 minutes cycling at 60% VO_{2max}. The proportion of the 50 and 100 g oxidised was significantly lower in the fructose trial; 32.2 and 45.8 g than the glucose 37.8 and 58.3 g respectively.

Fructose has consistently been shown to be oxidised at lower rates than glucose and this has been attributed to a slower rate of fructose conversion to glucose by the liver (Adopo, et al., 1994; Guezzenec, et al., 1989; Massicotte, et al., 1986; Massicotte, et al., 1989) and a lower rate of absorption by the intestine (Burelle, et al., 2006) although there is no direct evidence currently available to support this lesser rate of intestinal absorption.

2.3.4. Galactose

Two studies have investigated the oxidation of the monosaccharide galactose during exercise. Leijssen et al. (1995) compared the rates of exogenous- and endogenous-carbohydrate oxidation in participants cycling at 70% VO_{2max} for 2 hours whilst ingesting an 8% solution of either glucose or galactose. The mean oxidation rate of galactose (0.37 $g \cdot min^{-1}$) was only half the rate of intake (0.82

$g \cdot min^{-1}$), whereas, glucose was oxidised at almost the same rate as that of the intake (0.79 $g \cdot min^{-1}$). These results are in agreement with those of Burelle et al. (2006) in which the oxidation of galactose was ~40% lower than glucose during cycling at ~65% VO_{2max}. 100 g of glucose and galactose were ingested in separate trials and over 120 minutes, 23.7 (SE3.5) g of the galactose was oxidised compared to 40.5 (SE3.4) g of glucose. Again, although there is no evidence to explain the difference in oxidation rates of glucose and galactose, it was attributed to slow absorption of galactose by the intestine (Burelle, et al., 2006; Leijssen, et al., 1995), the conversion of galactose to glucose by the liver (Burelle, et al., 2006; Leijssen, et al., 1995) and a high rate of galactose loss in urine (Burelle, et al., 2006).

2.3.5. Sucrose

Sucrose is the disaccharide comprised of glucose and fructose. In a review of studies investigating the oxidation of carbohydrates, it was concluded that sucrose is oxidised at similar rates to glucose and the efficacy of these two carbohydrates may be similar (Jeukendrup and Jentjens, 2000).

When the oxidation rates of glucose and sucrose were compared during 90 minutes cycling at 70% VO_{2max}, total exogenous-carbohydrate oxidation was the same for both glucose and sucrose solutions (Moodley, et al., 1992). A similar relationship was observed by Wagenmakers et al. (1993) between sucrose and the glucose polymer, maltodextrin. Participants ingested either an 8% sucrose or maltodextrin solution during 120 minutes of cycling at 65% Wmax. No differences were observed in exogenous-carbohydrate, total fat or total carbohydrate oxidation or in the sparing of endogenous carbohydrate. The mean carbohydrate intake was ~1.2 $g \cdot min^{-1}$ and produced peak exogenous-carbohydrate oxidation rates of ~0.9 $g \cdot min^{-1}$ in both trials. These treatments significantly reduced oxidation of endogenous carbohydrates compared with the control.

2.3.6. Glucose polymers – Maltodextrins

Maltodextrins are glucose polymers derived from starches that can vary in chain length depending on the degree of hydrolytic processing. Massicotte et al. (1989) compared the ingestion of glucose to that of an isocaloric glucose polymer solution during 120 minutes of cycling at ~50% VO_{2max}. No significant differences were found between the rate of oxidation of the glucose and the glucose polymer (72% and 65% of the 98.9 g ingested). Similar observations were made by Rehrer et al. (1992b) in which 17% glucose and maltodextrin solutions were compared. Participants exercised at 70% VO_{2max} for 80 minutes and ingested 220 g of carbohydrate. Peak oxidation rates of the two carbohydrates were similar; 0.78 $g \cdot min^{-1}$ for glucose and 0.75 $g \cdot min^{-1}$ for maltodextrin demonstrating no differences in the oxidation of maltodextrin and glucose.

Further evidence for the comparable oxidation trends of glucose and maltodextrin was observed by Wagenmakers et al. (1993) in which an upper limit of maltodextrin oxidation was identified as being similar to that previously suggested of glucose (Hawley, et al., 1992a). The oxidation rates of four different maltodextrin solutions (4%, 8%, 12% and 16%) were compared. Maximal oxidation rates were reached with the 8% solution (148 g of carbohydrate). Further increases in carbohydrate ingestion did not significantly increase exogenous-carbohydrate oxidation and it was concluded that with intakes of 1.2 $g \cdot min^{-1}$ or greater, oxidation rates of exogenous carbohydrate reached values between 0.9 and 1.1 $g \cdot min^{-1}$. Further agreement is found in a recent study; Wallis et al. (2005) observed peak oxidation rates of 1.1 $g \cdot min^{-1}$ when an 11.25% maltodextrin solution was ingested at a rate of 1.8 $g \cdot min^{-1}$.

Moodley et al. (1992) appears to be the only study to have found differences in the oxidation of exogenous glucose when glucose and maltodextrin solutions are ingested during exercise. However, it has been suggested (Hawley, et al., 1992b) that the differences were small and were unlikely to be of physiological significance to carbohydrate availability during exercise. There appears to be little difference in oxidation rates when glucose or maltodextrin are ingested.

However, due to the lower osmolarity of maltodextrin, there seems to be some benefit to using maltodextrin in sports drinks where water absorption is also important (Rehrer, et al., 1992b).

2.3.7. Starch

Starches vary widely in structure and composition but all granules are composed of two major components: amylopectin and amylose. Starches with a relatively large amount of amylopectin are rapidly digested and absorbed, whereas those with high amylose content have a slow rate of hydrolysis (Jeukendrup and Jentjens, 2000). Guezzenec et al. (1989) found the rate of glucose delivery to be similar following glucose or pure corn starch ingestion during 120 minutes of cycling at 60% VO_{2max}. Both trials resulted in similar plasma glucose and insulin responses. Hawley et al. (1992a) compared the ingestion of a soluble starch with a glucose polymer solution. Participants ingested 1200 ml of 15% solutions during 90 minutes of cycling at 70% VO_{2max}. Despite similar rates of gastric emptying, the rate of exogenous-carbohydrate oxidation from the soluble starch solution was significantly greater than from the glucose polymer solution. In contrast Saris et al. (1993) found that amylopectin (branched chain) was oxidised at a higher rate than a combined amylopectin and amylose (long polymer chain) when participants ingested 316 g whilst cycling at 68% VO_{2max} for 2.5 hours. The amount of carbohydrate delivered to the intestine was not significantly different suggesting that digestion and absorption could have limited oxidation. It was postulated that the physical characteristics of the two starches affected the rate at which they are digested and therefore oxidised. Because the number of amylase binding sites available determines the hydrolysis rate, the branched chain structure of amylopectin gives it preferential characteristics for digestion. Saris et al. also suggested and confirmed that methodological errors had resulted in the high oxidation rates of the soluble starch in the Hawley study.

As soluble starch (amylopectin) is oxidised at a greater rate during exercise, its use should be preferred in sports drinks over amylose (Saris, et al., 1993).

However, the ingestion of starch may be as effective as free-glucose in maximising carbohydrate availability during exercise.

To summarise, it would appear that there is little difference between the carbohydrate available for oxidation during exercise when glucose, maltose, sucrose, glucose polymers and starch are ingested. Fructose whilst having a blunted insulin response and reduced inhibition of lipolysis seems to result in a lower oxidation rate relative to the ingestion rate together with galactose.

In a review of the oxidation of carbohydrate feedings during prolonged exercise (Jeukendrup and Jentjens, 2000), maximal exogenous-carbohydrate oxidation rates were plotted against ingestion rates from different carbohydrates oxidation studies. It was suggested from the graph that exogenous-carbohydrate oxidation may be optimal at rates of ingestion around 1.0-1.5 $g \cdot min^{-1}$. This is in agreement with studies in which even when very large amounts of carbohydrate are ingested, peak exogenous-carbohydrate oxidation is still ~1.0 $g \cdot min^{-1}$ (Jeukendrup, et al., 1999b; Rehrer, et al., 1992b; Wagenmakers, et al., 1993). Therefore, peak exogenous-carbohydrate oxidation rates reach 0.9-1.1 $g \cdot min^{-1}$ when glucose is ingested at a rate of 1.2 $g \cdot min^{-1}$ or greater (Hawley, et al., 1992a; Jentjens, et al., 2004c; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Jeukendrup, et al., 1999b; Wagenmakers, et al., 1993; Wallis, et al., 2005).

2.4. Multi-transportable carbohydrates

When two or three different types of carbohydrate are ingested together to create a facilitative effect on carbohydrate absorption, they are known as multi-transportable carbohydrates. Adopo et al. (1994) found that adding fructose to a glucose solution increases exogenous-carbohydrate oxidation by 21% compared with an isoenergetic glucose solution. Participants ingested 100 g of glucose or fructose or a combined drink containing 50 g glucose and 50 g fructose. The amount of exogenous carbohydrate oxidised was 58.3, 45.8 and 73.6 g in the glucose, fructose and combined trials respectively. However, solutions containing multi-transportable carbohydrates often have a higher osmolarity. As monosaccharides, glucose and fructose do not have to be

digested prior to absorption in the intestine, however, their short chain length increases the osmolality of solutions of which they are a part. Solution osmolality has been an important consideration in sports drinks design as it has been suggested that hypertonic solutions can reduce fluid and electrolyte absorption (Rehrer, et al., 1992b).

In a study by Shi et al. (1995) varying concentrations and combinations of glucose, maltodextrin, fructose and sucrose were ingested in solutions, to measure the affect of osmolality and carbohydrate form on water, sodium and carbohydrate absorption. Despite the increased osmolality of the solutions containing different substrates, both carbohydrate and water absorption increased following the ingestion of the solutions containing two transportable substrates. The increased water and solute absorption was attributed to the stimulation of multiple transport mechanisms by multiple substrates (Shi, et al., 1995).

More recently, a series of studies (Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Wallis, et al., 2005) has investigated peak exogenous-carbohydrate oxidation rates during exercise with ingestion of multi-transportable carbohydrates. Jentjens et al. (2004a) compared multi-transportable carbohydrate ingestion with an iso-energetic quantity of glucose and glucose at a rate thought to saturate intestinal transporters during 120 minutes cycling at ~62% VO_{2max}. The rates of carbohydrate ingestion were 1.8 $g \cdot min^{-1}$ (1.2 $g \cdot min^{-1}$ glucose and 0.6 $g \cdot min^{-1}$ fructose) for the multi-transportable carbohydrate mixture, 1.8 $g \cdot min^{-1}$ glucose in the high glucose trial and 1.2 $g \cdot min^{-1}$ glucose in the medium glucose trial. The mixture of glucose and fructose resulted in ~55% higher (1.26 $g \cdot min^{-1}$) exogenous-carbohydrate oxidation rates compared with the isoenergetic glucose trial (0.83 $g \cdot min^{-1}$). Furthermore, the average exogenous-glucose oxidation was similar for all three carbohydrate trials (0.75 $g \cdot min^{-1}$, 0.75 $g \cdot min^{-1}$ and 0.77 $g \cdot min^{-1}$ for the medium, high and mixed trials respectively). These results indicate that the higher oxidation rates in the mixed trial could be completely attributable to the additional fructose and confirm previous suggestions that glucose transporters may become saturated when glucose is

ingested at rates greater than 1.2 g·min^{-1} (Jentjens, et al., 2004a). A second study compared exogenous-carbohydrate oxidation rates when a mixture of glucose and sucrose (1.2 and 0.6 g·min^{-1}) were ingested compared to an isoenergetic amount of glucose (1.8 g·min^{-1}). The mixed solution resulted in peak exogenous-carbohydrate oxidation rates of ~1.25 g·min^{-1}, almost 20% higher than the 1.06 g·min^{-1} attained with an isoenergetic amount of glucose (Jentjens, et al., 2004b).

A further study compared a glucose, fructose and sucrose mix which provided 1.2, 0.6 and 0.6 g·min^{-1} of each of the carbohydrate types respectively (Jentjens, et al., 2004c). Peak exogenous-carbohydrate oxidation rates reached 1.7 g·min^{-1} when participants ingested 288 g carbohydrate in the mixed trial. This peak oxidation rate was ~50% higher than the peak exogenous-carbohydrate oxidation rate in the isocaloric glucose trial (1.17 g·min^{-1}). These high rates are in agreement with those of Hawley et al (1994) in which glucose was infused intravenously bypassing the stomach, intestines and liver. A peak oxidation rate of 1.8 g·min^{-1} was measured suggesting that the muscle can oxidise exogenous carbohydrate at high rates providing that sufficient amounts of exogenous carbohydrate is delivered into the blood stream. Unpublished data from cited in Jentjens and Jeukendrup (2005) showed that ingestion of equal amounts of glucose and sucrose at a rate of 2.4 g·min^{-1} produced relatively low oxidation rates (1.2 g·min^{-1}) and it was suggested that this may be due to inhibition of sucrose hydrolysis when glucose transporters are saturated (Jentjens, et al., 2004b).

Following on an attempt was made to quantify the rate at which fructose should be ingested in order to saturate fructose transporters and maximise oxidation rates from combined glucose and fructose (Jentjens and Jeukendrup, 2005). The ingestion of glucose at a rate of 1.2 g·min^{-1} was compared to that of glucose and fructose combined at a rate of 1.2 g·min^{-1} each. The combined ingestion of glucose and fructose resulted in a peak oxidation rate of 1.75 g·min^{-1} suggesting that an increase in the rate of fructose ingestion from 0.6 g·min^{-1} (Jentjens, et al., 2004a) to 1.2 g·min^{-1} in the present study leads to higher peak oxidation rates (1.26 v 1.75 g·min^{-1} respectively). The highest amount of

fructose ingested previously was 0.9 g·min^{-1} (0.6 g·min^{-1} directly from fructose and 0.3 g·min^{-1} from the hydrolysis of sucrose) (Jentjens, et al., 2004c). The peak oxidation rate when 1.2 g·min^{-1} fructose was ingested was similar to that when 0.9 g·min^{-1} was ingested which may indicate that a rate of intake of 0.9 g·min^{-1} may be sufficient to saturate fructose transporters and maximise exogenous-carbohydrate oxidation rates (Jentjens and Jeukendrup, 2005). Since it appears that the upper limit of exogenous-glucose and -fructose oxidation is ~1.1 and 0.6 g·min^{-1} respectively (Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jeukendrup and Jentjens, 2000) it was suggested that 1.75 g·min^{-1} may represent the upper limit of exogenous-carbohydrate oxidation that is physiologically possible when ingesting multi-transportable carbohydrates orally (Jentjens and Jeukendrup, 2005).

Wallis et al. (2005) used a protocol similar to that of previous studies (Jentjens, et al., 2004c; Jentjens, et al., 2004b) and compared exogenous-carbohydrate oxidation rates following the ingestion of maltodextrin and fructose combined (1.2 and 0.6 g·min^{-1} respectively) with an isocaloric maltodextrin (1.8 g·min^{-1}) solution. Oxidation rates of 1.5 g·min^{-1} were observed during the combined maltodextrin and fructose trial, approximately 40% higher than those observed during the maltodextrin only trial (~1.1 g·min^{-1}). Whilst the peak oxidation rates during the maltodextrin trial are in agreement with the findings of Jentjens et al. (2004b,c, 2005), the peak oxidation rate in the glucose and fructose trial was higher than that reported during the similar trial of Jentjens et al. (2004a) (1.5 g·min^{-1} compared with 1.26 g·min^{-1}). It was postulated (Wallis, et al., 2005) that this discrepancy may be due to differences in the osmolality of the solutions (866 mOsm·kg^{-1} in Jentjens et al. vs 260 mOsm·kg^{-1}). Alternatively the slightly higher exercise intensity of Wallis et al. (55% Wmax compared to 50% Wmax of Jentjens) may have demanded higher rates of oxidation.

In addition to confirmation of an upper limit of glucose absorption, these studies confirm that when solutions containing both glucose and fructose are ingested, high rates of exogenous-carbohydrate oxidation can be achieved. The combined ingestion of glucose and fructose at rates of 1.2 g·min^{-1} each has resulted in the highest recorded peak exogenous-carbohydrate oxidation rates

during exercise (Jentjens and Jeukendrup, 2005). However, a mixture of maltodextrin ingested at 1.2 $g \cdot min^{-1}$ with fructose at either 0.9 or 1.2 $g \cdot min^{-1}$, may lead to even greater oxidation rates but this has yet to be tested.

2.4.1. Mechanisms by which multi-transportable carbohydrates increase exogenous-carbohydrate oxidation rates

By considering the absorption and oxidation mechanisms of glucose and fructose, we begin to understand how their combined ingestion increases exogenous-carbohydrate oxidation.

2.4.1.1. Glucose Kinetics

Glucose is transported from the small intestine to the blood in two stages. Initially glucose is co-transported with sodium by SGLT1 from the intestinal lumen across the brush border membrane (Wright, et al., 2006). The sodium-potassium pump works across the basolateral membrane to maintain low enterocyte intracellular sodium concentrations compared to that of the lumen. This creates a sodium electrochemical potential gradient across the brush border membrane of the small intestine. SGLT1 couples glucose transport to the sodium gradient and acts as the co-transporter for glucose into the enterocytes (Wright, et al., 2006). Following the sodium-dependant transport across the brush border, glucose transport is facilitated across the basolateral membrane via a concentration gradient by GLUT2. This facilitated diffusion across the basolateral membrane is sodium independent (Wright, et al., 2006).

It has been postulated that SGLT1 transporters may become saturated when glucose ingestion exceeds the rate of 1.2 $g \cdot min^{-1}$ and therefore intakes above this to do not alter exogenous-glucose oxidation rates (Jentjens, et al., 2004a). Recent studies as mentioned earlier would appear to confirm that the maximal rate of glucose absorption across the intestine is between 1.0-1.1 $g \cdot min^{-1}$, (Jentjens, et al., 2004c; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Wallis, et al., 2005).

2.4.1.2. Fructose Kinetics

In 1964, Holdsworth et al. measured the absorption of glucose and fructose across the intestine and because no evidence of competition between the sugars was found, it was concluded that their absorption must occur via different transport mechanisms. Furthermore, fructose absorption has been found to be unaffected by mutations in the small intestinal Na^+/Glucose co-transporter (SGLT1) which cause glucose uptake to cease, providing further evidence of a separate transport protein (Wright, et al., 1991). However, fructose uptake was stimulated when GLUT5 mRNA was injected into oocytes which confirmed that GLUT5 is responsible for the uptake of fructose from the lumen of the small intestine across the brush border membrane (Burant, et al., 1992). The process of absorption is mediated by GLUT2 which is present on the basolateral membrane of the enterocytes because little, if any, GLUT5 is found on the basolateral membrane (Burant, et al., 1992). GLUT2 is responsible for transporting fructose and glucose across the basolateral membrane (Wright, et al., 2006).

2.4.1.3. Multi-transportable carbohydrate Kinetics

As mentioned earlier, fructose ingestion results in lower exogenous-carbohydrate oxidation rates when compared to glucose and this has been attributed to lower absorption of fructose through the intestine (Adopo, et al., 1994; Guezzenec, et al., 1989; Massicotte, et al., 1986; Massicotte, et al., 1989). Fructose is absorbed from the intestine by GLUT5 transporters, (Burant, et al., 1992; Davidson, et al., 1992; Ferraris and Diamond, 1997) providing a different transport mechanism to that of glucose. However, fructose absorption is facilitated by the presence of glucose (Holdsworth and Dawson, 1964; Rumessen and Gudmand-Hoyer, 1986).

Shi et al. (1997) investigated the mechanism for enhanced fructose absorption with co-ingestion of glucose and hypothesised three possible mechanisms of fructose transport across the small intestine: facilitated diffusion (GLUT5), a disaccharide-specific transport mechanism and paracellular transport. Using the triple lumen technique, different carbohydrate solutions were diffused consisting of combinations of glucose, fructose, sucrose, mannitol, acarbose, and

lactulose. Acarbose was used to block the disaccharide specific transport without affecting facilitated diffusion or paracellular pathways. The addition of mannitol would allow only facilitated diffusion, lactulose was used to examine paracellular transport and sucrose would primarily stimulate any disaccharidase-related transport. Fructose absorption was greatest in both the fructose-glucose (FruGlu) solutions and fructose-glucose-acarbose (FruGluA) solutions, providing evidence that any disaccharidase transport system is not used for free fructose and glucose. Fructose absorption from the fructose-mannitol (FruMann) solution was 71% of the fructose absorption from the FruGlu solution, indicating that the major route of fructose absorption is through facilitated diffusion. Finally, no lactulose was absorbed from solutions with only fructose and sucrose with acarbose, indicating that free glucose or the glucose produced from sucrose hydrolysis is essential for transport through the paracellular pathways (Shi, et al., 1997). This indicated that fructose absorption was enhanced when co-ingested with glucose. It was suggested that when fructose is co-ingested with glucose, the glucose opens tight junctions between epithelial cells and fructose absorption is enhanced. The opening of these junctions would allow small solutes including fructose to move passively with water by solution drag (Shi, et al., 1997).

In summary, glucose and fructose are absorbed by SGLT1 and GLUT5 respectively across the brush border membrane with GLUT2 transporting both across the basolateral membrane. The different transport mechanisms allow them to be co-ingested whilst not competing for absorption transporters allowing increased carbohydrate absorption. Furthermore, it is postulated that fructose transport is further enhanced in the presence of glucose which opens paracellular channels. In this way the absorption of carbohydrate is highest when glucose and fructose are ingested together.

2.5. Osmolality and Concentration

It could be considered that whatever factors limit gastric emptying could potentially limit exogenous-carbohydrate oxidation by limiting the delivery of

carbohydrate to the intestine for absorption. Whilst a number of factors such as carbohydrate concentration (Maughan, et al., 1989; Rehrer, et al., 1992b), calorific content (Jandrain, et al., 1989), and the volume and timings (Noakes, et al., 1991) of fluid ingested clearly have an important effect on gastric emptying, it has become apparent that gastric emptying itself does not affect exogenous-carbohydrate oxidation.

Rehrer et al. (1992b) compared the effect of the concentration and osmolality of ingested solutions on exogenous-carbohydrate oxidation. Gastric emptying, water and carbohydrate absorption and exogenous-carbohydrate oxidation were measured during 80 minutes of cycling at 70% VO_{2max} with ingestion of 4.5% glucose, 17% glucose and 17% maltodextrin solutions. Gastric emptying rates of the 17% solutions were slower than the 4.5% solution and water; however, this did not affect exogenous-carbohydrate oxidation rates. These results agree with those of Moodley et al. (1992) who also found that whilst ingestion of a 15% carbohydrate solution did reduce gastric emptying compared to a 7.5% solution, total exogenous-carbohydrate oxidation was significantly greater with the 15% solution. As with other studies (Hawley, et al., 1991; Hawley, et al., 1992a; Jeukendrup, et al., 1999a; Saris, et al., 1993), this was attributed to increased carbohydrate delivery to the intestine and an increased percentage of that delivered being oxidised, confirming that gastric emptying does not limit exogenous-carbohydrate oxidation.

Independent of the effect on gastric emptying, solution osmolality and concentration may affect intestinal absorption and therefore exogenous-carbohydrate oxidation. Different solution concentrations affect exogenous-carbohydrate oxidation in respect to the quantity of carbohydrate they provide. Solutions of low concentration may provide less carbohydrate than can be absorbed by the intestine and in that respect it is the quantity rather than the concentrations that affects absorption and oxidation. Jandrain et al. (1989) investigated the oxidation of 50 g glucose dissolved in 200, 400 or 600 ml of water (1204, 644 and 439 $mmol \cdot l^{-1}$ respectively). Although the concentrations and osmolality of these drinks was different, no differences were observed in blood glucose response or exogenous-carbohydrate oxidation during 4 hours of

exercise at 45% VO_{2max}. These results suggest that the total amount of carbohydrate seems to be a more important determinant of exogenous-carbohydrate oxidation than osmolality or carbohydrate concentration (Jeukendrup and Jentjens, 2000).

Studies comparing glucose and glucose polymer solutions which have different osmolalities have generally shown both to have similar exogenous-carbohydrate oxidation rates (Hawley, et al., 1991; Hawley, et al., 1992a; Massicotte, et al., 1989; Rehrer, et al., 1992b; Wagenmakers, et al., 1993). These results indicate that solution osmolality is not important when large amounts of single carbohydrates are ingested during exercise. However, as mentioned earlier, it has been suggested that solution osmolality may become an important factor in determining exogenous-carbohydrate oxidation rates when large amounts of multi-transportable carbohydrates are ingested during exercise (Wallis, et al., 2005).

The protocol of Wallis et al. (2005) was similar to that used in a series of studies by Jentjens et al. (2004b,c) but substituted glucose with maltodextrin to significantly reduce the osmolality (866 $mOsm \cdot kg^{-1}$ to 260 $mOsm \cdot kg^{-1}$) of a solution containing glucose and fructose at rates aimed to saturate intestinal transporters. The peak oxidation rate of 1.5 $g \cdot min^{-1}$ exceeded that of 1.26 $g \cdot min^{-1}$ observed in the similar study of Jentjens et al. (2004a). Although it is possible that a slight difference in exercise intensity between the studies (55% Wmax, Wallis vs 50% Wmax, Jentjens) may have affected peak oxidation rates, it was postulated that the discrepancy was due to differences in the osmolality of the solutions (Wallis, et al., 2005). These findings are in contrast to those of Shi et al. (1995) in which solutions of differing osmolality and concentration were compared in respect to their affect on water and carbohydrate absorption. Osmolality and concentration had no direct correlation with carbohydrate absorption and it was concluded that in solutions where two or three carbohydrates are ingested, osmolality may be less important than Na+ and carbohydrate transport in maximising water and carbohydrate absorption (Shi, et al., 1995). The differences in the conclusions of Shi et al. and those of Wallis et al. and Jentjens et al. maybe due to differences in the magnitude of the

osmolality of the test solutions. In Shi et al. the highest solution osmolality was 477 $mOsm \cdot kg^{-1}$ and the solutions were perfused in resting subjects, which may not have been great enough to show a significant effect. The combined solution of Jentjens et al. (2004a) was considerably higher (866 $mOsm \cdot kg^{-1}$) than that of Wallis et al. (2005) (260 $mOsm \cdot kg^{-1}$) leading to the suggestion that the higher exogenous-carbohydrate oxidation rates of Wallis may be due to the lower solution osmolality.

In conclusion, while the concentration and osmolality of a solution do not appear to directly influence intestinal absorption and exogenous-carbohydrate oxidation rates, osmolality could be a more important determinant when large quantities of multi-transportable carbohydrate are ingested although this needs to be investigated further.

2.6. Physiological Mechanisms by which Carbohydrate Affects Endurance Exercise

It is well established that carbohydrate feedings during exercise can delay fatigue following prolonged exercise (Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; Tsintzas, et al., 1996a; Tsintzas, et al., 1996b; Yaspelkis, et al., 1993), improve performance of set distance events (Tsintzas, et al., 1993; Tsintzas, et al., 1995b) and improve sprint performance following prolonged exercise (Fielding, et al., 1985; Hargreaves, et al., 1984). However, the manner in which supplemental carbohydrate improves performance is still a source of debate with findings suggesting both that performance is improved through the maintenance of blood glucose and high rates of glucose oxidation (Coggan and Coyle, 1987; Coyle, et al., 1986; Hargreaves and Briggs, 1988), the sparing of muscle- (Hargreaves, et al., 1984; Nicholas, et al., 1999; Tsintzas, et al., 1995a, 1996a; Yaspelkis, et al., 1993) and liver-glycogen (Bosch, et al., 1994; Jeukendrup, et al., 1999a; Jeukendrup, et al., 1999b; McConnell, et al., 1994), the resynthesis of glycogen during low-intensity exercise (Kuipers, et al., 1987) and through the attenuation of central fatigue (Dalsgaard, et al., 2002; Davis, et al., 1992; Nybo, et al., 2005; Nybo, et al., 2003; Snow, et al., 2000).

2.6.1. Maintenance of Blood Glucose

In a study by Bergstrom and Hultman (1967), muscle glycogen was shown to be spared during exercise when carbohydrate was infused. However, at 13-30 $mmol \cdot l^{-1}$, blood-glucose concentrations were abnormally high following infusion compared to 3.5-5.5 $mmol \cdot l^{-1}$, the normal levels during exercise without infusion (Bergstrom and Hultman, 1967). This makes it difficult to draw conclusions as to what sparing occurs under normal physiological conditions.

Coyle et al. (1983) observed participants cycling at ~70% VO_{2max} for 180 minutes or until fatigue whilst ingesting either a glucose polymer solution or a placebo. In the carbohydrate trial performance was improved due to delayed fatigue. Fatigue did not correspond to hypoglycaemia but participants commented on a severe weariness in the working muscles. Respiratory exchange ratio in both trials was the same indicating that the proportion of energy derived from carbohydrate oxidation was equal and since blood glucose and insulin concentrations were higher in the carbohydrate trial by 20-40% and 50% respectively, it was suggested that the rate of glucose uptake by working muscles was increased. It was postulated that the carbohydrate feeding spared muscle glycogen. A further study (Coyle, et al., 1986) took direct measurements of muscle glycogen. Whilst carbohydrate feeding during exercise improved time to fatigue, increased blood glucose concentrations and carbohydrate oxidation compared to the placebo trial, glycogen was not spared. It was concluded that late in exercise, when muscle glycogen is low, blood-glucose oxidation is increased and that exogenous-carbohydrate maintains these high rates of carbohydrate oxidation and delays fatigue. To test this hypothesis a follow-up study was conducted (Coggan and Coyle, 1987). Participants cycled to fatigue at 73% VO_{2max} (bout 1), rested for 20 minutes and ingested either water, glucose or had intravenous glucose supplementation and then continued to exercise to exhaustion (bout 2). Low levels of blood glucose and a subsequent decrease in RER at fatigue suggested that the fatigue was due to an inadequate supply of carbohydrate to the working muscles (Coggan and Coyle, 1987). The decline in carbohydrate oxidation was reversed and participants

were able to continue exercise for 26 minutes when euglycaemia was restored and maintained with glucose ingestion and 43 minutes with glucose infusion. There were no changes in muscle glycogen. These results supported the hypothesis that the maintenance of carbohydrate oxidation late in exercise is important in delaying fatigue. Other studies (Hargreaves and Briggs, 1988; Mitchell, et al., 1989) have also shown no glycogen sparing effect in moderate to heavy (65-75% VO_{2max}) continuous cycling exercise.

These studies show that carbohydrate feedings improve performance without changes in muscle glycogen, however, Wagenmakers et al. (1993) observed significantly reduced endogenous-carbohydrate oxidation with 8, 12 and 16% maltodextrin solutions compared with a water control providing indirect evidence that either muscle or liver glycogen is spared during exercise when carbohydrate is ingested.

2.6.2. Sparing of Muscle Glycogen

Several authors (Hargreaves, et al., 1984; Nicholas, et al., 1999; Tsintzas, et al., 1995a, 1996a; Yaspelkis, et al., 1993) have found direct evidence that muscle glycogen is spared during exercise when carbohydrate is ingested. It would appear that carbohydrate ingestion has an ergogenic affect on continuous cycling and that this is related to the maintenance of blood glucose and carbohydrate oxidation rather than glycogen sparing. However, a series of studies by Tsintzas et al. (1993, 1995a,b, 1996a,b) has found that fatigue in prolonged running exercise does not coincide with hypoglycaemia yet carbohydrate ingestion does improve performance (Tsintzas, et al., 1993; Tsintzas, et al., 1995b) and capacity (Tsintzas, et al., 1996a; Tsintzas, et al., 1996b). Furthermore, the ingestion of carbohydrate reduced muscle-glycogen use during running (Tsintzas, et al., 1995a, 1996a). Carbohydrate ingestion has also been observed to improve exercise capacity (Hargreaves, et al., 1984; Yaspelkis, et al., 1993) and reduce glycogen depletion when ingested during intermittent cycling (Hargreaves, et al., 1984; Yaspelkis, et al., 1993) and running (Nicholas, et al., 1999).

2.6.2.1. Running

Tsintzas et al. (1993) found that endurance-running performance improvements were independent of changes in blood-glucose concentration. Participants completed a 30-km race whilst consuming either a 5% carbohydrate drink or water. Carbohydrate ingestion improved time to complete the race by allowing the runners to maintain their running speed for the duration of the race and there was no indication of hypoglycaemia in the water trial. These results do not substantiate suggestions that carbohydrate ingestion improves performance by maintaining blood-glucose concentrations. It was suggested that low hepatic- and muscle-glycogen stores in the water trial may have resulted in reduced total carbohydrate oxidation rate causing the reduction in running speed over the last 5 km of the race (Tsintzas, et al., 1993). Similar results were obtained when carbohydrate was ingested during marathon-running on a treadmill (Tsintzas, et al., 1995b).

To measure the effect of ingesting water versus a 5.5% carbohydrate electrolyte solution on muscle-glycogen use, biopsies were taken prior to and following 60 minutes running at 70% VO_{2max} (Tsintzas, et al., 1995a). Carbohydrate ingestion resulted in 28% glycogen sparing predominantly in type-1 fibres (42% reduction). Blood glucose and insulin concentrations were higher in the carbohydrate compared to the water trial which because of their potential to facilitate the uptake of glucose into the working muscles, this may explain the reduction in the use of muscle glycogen. These results indicate that the uptake of blood glucose can be increased early on in exercise when muscle-glycogen concentrations are high (Tsintzas, et al., 1995a) not just late in exercise when glycogen is low (Coyle, et al., 1986; Gollnick, et al., 1981). A follow-up study (Tsintzas, et al., 1996b) was completed to see if the carbohydrate provided in the first hour of exercise delayed the onset of fatigue. Participants ingested the same amount of fluid and carbohydrate in the first hour of exercise to exhaustion at 70% VO_{2max}. Fatigue was delayed in the carbohydrate trials and there was no hypoglycaemia at exhaustion. It was surmised that the sparing of muscle glycogen rather than the maintenance of blood glucose and carbohydrate oxidation towards the end of exercise, might have been

responsible for the improvement in endurance running capacity (Tsintzas, et al., 1996b).

A further study by Tsintzas et al. (1996a) measured muscle-glycogen content at rest and at exhaustion from running following ingestion of carbohydrate (CHO) and a placebo (Pl). Muscle-glycogen content was also measured during CHO trial at the same point at which exhaustion was experienced in the placebo trial (TPE). Again carbohydrate ingestion delayed the point of fatigue (104.3 ± 8.6 and 132.4 ± 12.3 minutes) in Pl and CHO trials respectively. The average rate at which muscle-glycogen was used during Pl and to TPE in CHO was 3.2 ± 0.3 and 2.5 ± 0.1 $mmol \cdot kg^{-1} \cdot min^{-1}$ respectively. A rate of 2.6 ± 0.7 $mmol \cdot kg^{-1} \cdot min^{-1}$ was maintained to exhaustion in CHO. At TPE muscle glycogen was 24% higher following carbohydrate ingestion. Total carbohydrate oxidation rate was the same throughout exercise and at exhaustion indicating that a reduction in total carbohydrate oxidation was not responsible for exhaustion. However, muscle-glycogen content at exhaustion was very low in both the placebo and carbohydrate trials with approximately 90% having been used suggesting that low levels of muscle glycogen are associated with fatigue during prolonged running. Because the rate of total carbohydrate oxidation was the same in both trials, but the rate of glycogen use was lower in CHO, blood glucose must have made a larger contribution to energy metabolism in CHO.

This series of studies provides strong evidence that in running exercise a reduction in muscle-glycogen content of type-1 fibres is associated with fatigue. Furthermore, ingestion of a carbohydrate-electrolyte solution during running will elevate blood-glucose and insulin concentrations which increase the uptake of glucose by the working muscle and spares muscle glycogen. This increased uptake occurs early in exercise and delays the point at which muscles are depleted of glycogen, increasing time to exhaustion. This carbohydrate ingestion and glycogen sparing also allows running speeds to be maintained preventing a reduction in performance time over distances such as 30- or 42.2-km.

2.6.2.2. Intermittent Exercise

Hargreaves et al. (1984) fed carbohydrate to participants during 4 hours of intermittent exercise. Glycogen measurements were taken at 0, 1 and 4 hours and time to exhaustion of sprint performance at the end of the 4 hours was measured. Over the 4 hours, blood-glucose concentrations decreased in the control group but not beyond that considered normal. Blood-glucose concentrations were maintained following carbohydrate ingestion. Total glycogen utilisation was lower following carbohydrate ingestion and time to exhaustion in the final sprint was longer, 126.8 and 87.2 s in the carbohydrate and control groups respectively. A follow-up study by Fielding et al. (1985) used the same exercise protocol and observed elevated and maintained blood-glucose levels with carbohydrate ingestion but no sparing of muscle glycogen. The quantity of carbohydrate however, was less than half that used in the previous study (21.5 and 45 $g \cdot h^{-1}$ respectively) and this may have been insufficient to affect the use of muscle glycogen (Tsintzas and Williams, 1998).

These findings agree with those of Yaspelkis et al. (1993) in which reduced muscle glycogen and increased time to exhaustion following ingestion of a carbohydrate solution was also observed. Participants followed an exercise regimen that alternated between periods of low and medium intensity bouts (45 and 75% VO_{2max} respectively) for 200 minutes and then cycled at 80% VO_{2max} to exhaustion. At 190 minutes the carbohydrate treatment had maintained muscle-glycogen concentration at a level that was ~35% greater than that of the placebo treatment. In addition, time to exhaustion was ~33 and ~2 mins in the carbohydrate and placebo trials respectively. The glycogen concentration was significantly greater in type-1 fibres in the carbohydrate trial compared to the placebo (393 and 314 $\mu mol \cdot g^{-1}$ respectively) and there was no difference between treatments in type-2 fibres. It was postulated that the mechanism which induced the reduced glycogenolysis was hyperglycaemia and hyperinsulineamia which would have promoted glucose uptake to the working muscles and spared muscle glycogen.

A recent study by Palmer et al. (1999) has also shown a trend towards greater muscle-glycogen sparing following carbohydrate ingestion during variable

intensity (VI) versus continuous intensity cycling (C). Muscle-glycogen content was reduced by 65% following 140 minutes of exercise during C compared with 49% during VI. Again the sparing occurred in type-1 fibres. Total carbohydrate oxidation was the same in both trials, however, a greater amount of blood glucose was oxidised in the VI trial than in the C trial (99.2 ± 5.3 and 83.9 ± 5.2 $g \cdot 140min^{-1}$ respectively). The difference between the amounts of blood glucose used between the trials (15 g) was not considered to be sufficient to account for the differences in muscle-glycogen content and it was suggested that glycogen resynthesis may have taken place in type-1 fibres during low-intensity periods of the VI trial. Blood insulin responses were similar in the two trials.

Glycogen sparing has also been observed during intermittent running. Nicholas et al. (1999) found that carbohydrate ingestion during 90 minutes of intermittent, high intensity running reduced muscle glycogen utilisation by 22%. Glycogen sparing occurred in both type-1 and -2 muscle fibres though it more so in type 1. The greater use of glycogen from type-2 muscle fibres shows the heavy demands of the type of exercise used.

The sparing seen during intermittent exercise could be due to hyperglycaemia and hyperinsulineamia promoting glucose uptake to the working muscles and sparing glycogen in type-1 fibres as suggested (Yaspelkis, et al., 1993) or for muscle-glycogen resynthesis to be occurring in the type-2 fibres during periods of low intensity exercise (Kuipers, et al., 1987) or it may be a combination of both.

A study by Mitchell et al. (1989) found no difference between glycogen use during intermittent and continuous exercise when carbohydrate was ingested. In this study however, although exercise was intermittent in nature the intensity remained constant at 70% VO_{2max}. This may have prevented any resynthesis from occurring in type-2 fibres due to the absence of periods of low-intensity exercise as has been suggested necessary for resynthesis to occur (Kuipers, et al., 1987).

2.6.3. Sparing of Liver Glycogen

As exercise time continues muscle-glycogen stores are gradually depleted and the muscles increase their uptake of blood glucose to maintain oxidation rates (Gollnick, et al., 1981). It is the role of the liver to maintain blood-glucose concentrations and a number of tracer studies (Bosch, et al., 1994; Jeukendrup, et al., 1999a; Jeukendrup, et al., 1999b; McConnell, et al., 1994) have investigated the effect of carbohydrate ingestion on liver glucose kinetics. Bosch et al. (1994) investigated the effect of carbohydrate ingestion during prolonged exercise on liver-glucose turnover, exogenous- and endogenous-glucose oxidation and muscle-glycogen utilisation. During 180 minutes of cycling at 70% VO_{2max} liver glucose production was reduced in those consuming a 10% carbohydrate drink (CI). This reduced hepatic output was due to a significant contribution of ingested carbohydrate to the total rate of appearance (Ra) of glucose. Mean endogenous Ra of the CI participants was 65% of the placebo subjects (PI) (72 vs 111 g) giving a total of 46 g glycogen spared. The blood-glucose oxidation was higher in CI as a result of exogenous-glucose oxidation; however, lower rates of blood-glucose oxidation from endogenous glucose in the CI trial indicate a liver-glycogen sparing effect (Bosch, et al., 1994). In a similar study, McConnell et al. (1994) observed comparable results. Glucose ingestion decreased hepatic-glucose production by 51% and a further study of Bosch et al. (1996) confirmed a liver-glycogen sparing effect was independent of initial muscle-glycogen levels.

Jeukendrup, et al. (1999a) found that whilst liver glycogenolysis was reduced, muscle-glycogen utilisation remained unchanged when glucose was ingested during prolonged exercise. Trained cyclists exercised at 50% VO_{2max} for 120 minutes in the fasted state whilst ingesting either no glucose or a total of 70 g or 350 g in fasting, low- and high-glucose trials respectively. Muscle-glycogen oxidation was similar in all the trials even when large amounts of glucose were ingested. However, the large amounts of exogenous carbohydrate ingested in the high trial appeared to completely suppress liver glycogenolysis. The rate of appearance of glucose in the low trial was greater than the amount ingested (0.84 $g \cdot min^{-1}$ and 0.3 $g \cdot min^{-1}$ respectively) suggesting that the liver was still

producing glucose. However, in the high-glucose trial the rate of appearance was less than the rate of ingestion (1.36 $g \cdot min^{-1}$ and 1.5 $g \cdot min^{-1}$ respectively) suggesting that hepatic-glucose production could be completely suppressed. A comparison of the rate of appearance of glucose from [6,6-^{2}H2]- and [U^{13}C]-glucose tracers provided an indication of gluconeogenesis which was found to be negligible and was not reduced by carbohydrate feedings.

A further study (Jeukendrup, et al., 1999b) used a dual tracer approach to measure total R_a and R_{agut} (rate of appearance of ingested glucose) allowing hepatic-glucose production to be quantified. When 175 $g \cdot h^{-1}$ glucose was ingested, endogenous-glucose production was completely blocked, all glucose appearing in the plasma was that of the ingested glucose. Although the tracer method used was unable to establish the difference between glycogenolysis and gluconeogenesis. As a previous study (Jeukendrup, et al., 1999a) had found gluconeogenesis to be negligible during exercise when glucose is ingested, it was suggested that glucose ingestion mainly affected glycogenolysis and that this may have been due to high insulin levels. These findings are significant in that they suggest that exogenous-carbohydrate feedings may prevent liver glycogenolysis. In the same study, the disappearance of glucose was closely matched by the plasma-glucose oxidation rate in fasting, low- and high-glucose trials indicating that muscle-glycogen was not being synthesised and all glucose taken up by the muscles was used as fuel for work. Muscle-glycogen concentration was not different between the low- and high-glucose trials. This finding is in agreement with those studies which took direct measurement of muscle glycogen using biopsy techniques (Bosch, et al., 1994; Coyle, et al., 1986; Hargreaves and Briggs, 1988).

Recently, Jeukendrup et al. (2006) has shown that during ultra-endurance exercise, when presumably muscle-glycogen concentrations are low following 2-4 hours of exercise, there is increased demand on hepatic-glucose output for fuel and that exogenous carbohydrate provides a substantial proportion of the carbohydrate oxidised. During 5 hours of cycling at 58% VO_{2max}, there was a linear rise in the R_d of blood glucose which would be expected as it has been

shown that blood glucose uptake is increased as muscle-glycogen stores are reduced (Gollnick, et al., 1981) and blood-glucose oxidation increases with the duration of exercise (Ahlborg and Felig, 1982). However, as the oxidation of exogenous carbohydrate levelled off after 120 minutes, an increase in hepatic-glucose output must have occurred. Participants ingested either glucose (glu) or glucose and fructose (glu+fru) at the rate of 1.5 $g \cdot min^{-1}$. The glu+fru provided a greater contribution to the total amount of carbohydrate oxidised (77%) compared to glu (65%) without a concurrent rise in R_d or R_a glucose. It was suggested that the additional carbohydrate from glu+fru may have been transported to the muscles or liver in a non-glucose form such as lactate or that extra glucose could have been oxidised in the liver.

These studies suggest that exogenous carbohydrate appears to reduce hepatic-glucose output, sparing liver-glycogen stores. When exercise is prolonged, muscle glycogen is gradually reduced resulting in an increased glucose uptake. If this increased uptake causes hypoglycaemia, exercise intensity must be reduced or exercise must be discontinued. Since the liver maintains blood glucose, this increased uptake must be compensated for by liver gluconeogenesis or glycogenolysis or exogenous-carbohydrate ingestion. If sufficient glucose can be provided by exogenous sources, liver-glycogen stores can be maintained delaying fatigue due to depleted liver-glycogen stores.

2.6.4. Sparing of Liver and Muscle Glycogen

Recent studies (Jentjens, et al., 2004c; Wallis, et al., 2005) suggest that when large amount of carbohydrate is ingested, muscle-glycogen in addition to liver-glycogen, may be spared. Jentjens et al. (2004c) indicates a strong possibility that muscle glycogen may be spared as a result of exogenous-carbohydrate ingestion. Participants cycled for 150 minutes at ~62% VO_{2max} whilst ingesting either water, glucose or a mixed glucose, sucrose and fructose solution (MIX). Ingestion of MIX resulted in almost 30% lower endogenous-glucose oxidation. Although, the methodology did not allow the source of the spared glucose to be identified as either muscle- or liver-derived glucose, it could be assumed that

reduced endogenous-carbohydrate oxidation spared muscle glycogen. Jeukendrup et al. (1999b) found that when carbohydrate was ingested at an average rate of 3.0 $g \cdot min^{-1}$ during prolonged cycling, hepatic-glucose output (HGP) was completely blocked. Ingestion at a rate of 0.6 $g \cdot min^{-1}$ had suppressed HGP to ~0.16 $g \cdot min^{-1}$ from 0.4 $g \cdot min^{-1}$ in the fasting trial a difference of 0.24 $g \cdot min^{-1}$. If this was extrapolated onwards it would suggest that ingestion of carbohydrate so that exogenous R_a was 1 $g \cdot min^{-1}$ would be sufficient to block HGP. The average rate of ingestion in Jentjens study was 2.4 $g \cdot min^{-1}$, slightly less than the 3.0 $g \cdot min^{-1}$ of Jeukendrup, and peak exogenous-carbohydrate oxidation rate was 1.7 $g \cdot min^{-1}$. If liver-glucose production was suppressed to the same extent during MIX as in that of Jeukendrup et al. then it would indicate that the endogenous carbohydrate being spared is that of the muscle in addition to the liver.

2.6.5. Glycogen Resynthesis during Exercise

The relationship between exogenous-carbohydrate supplementation and muscle-glycogen sparing is not completely clear. The ability to make judgments could be complicated by glycogen resynthesis during periods of lower intensity exercise.

Glycogen resynthesis was initially observed during exercise in trained rats (Constable, et al., 1984; Kuipers, et al., 1986) and then male cyclists (Kuipers, et al., 1987). In a study by Kuipers et al. (1987) male cyclists completed intermittent exercise to exhaustion and then consumed a carbohydrate drink whilst either resting or continuing to cycle at 40% Wmax for 3 hours. As in the rat study, glycogen resynthesis occurred in glycogen-depleted muscle during mild exercise. Whilst glycogen depletion occurred in types-I and -IIA fibres during the intensive exercise, repletion occurred mainly in type-II fibres. It was suggested that this was the result of the relative inactivity of type-II fibres during moderate exercise (Kuipers, et al., 1987) as it has been shown that type-II fibres are largely inactive during low to moderate-intensity exercise (Gollnick, et al., 1974). The resynthesis of glycogen during moderate exercise was not observed in non-endurance-trained, glycogen-depleted subjects with high

carbohydrate intakes suggesting that the ability to do this may be an adaptation response to endurance training (Kuipers, et al., 1989).

2.7. Effect of Carbohydrate Ingestion on Central Nervous System

In addition to the peripheral mechanisms discussed, carbohydrate may delay central nervous system (CNS) fatigue by reducing serotonin synthesis (Davis, et al., 1992), maintaining cerebral glucose uptake (Nybo, et al., 2003), preventing the depletion of brain glycogen (Dalsgaard, et al., 2002) and preventing cerebral hyperammonemia (Nybo, et al., 2005; Snow, et al., 2000).

2.7.1. Serotonin Synthesis

Tryptophan (TRP) is a precursor to serotonin (5-Hydroxytryptamine, (5-HT)) and normally circulates in plasma loosely bound to albumin. However, free tryptophan (f-TRP) can be transported across the blood-brain barrier via a specific mechanism that TRP shares with the branched chain amino acids (BCAA's) (Chaouloff, 1989). Consequently when the concentration ratio of f-TRP to BCAA's increases there is an increase in the uptake of f-TRP by the brain (Chaouloff, et al., 1986). During exercise the delivery to the brain of f-TRP increases and the synthesis of serotonin increases (Chaouloff, et al., 1985; Chaouloff, et al., 1987). It is postulated that this leads to impaired CNS function during prolonged exercise (Newsholme, et al., 1987). It has been proposed that f-TRP increases during prolonged exercise firstly, because BCAA's are oxidised for energy by the working muscle which would reduce their concentration in the plasma and increase the f-TRP:BCAA. Furthermore, during prolonged exercise, plasma free fatty acids (FFA's) increase displacing TRP from its binding site on albumin, increasing f-TRP (Curzon, et al., 1973). It was suggested that carbohydrate supplementation during exercise would attenuate f-TRP and f-TRP/BCAA and delay CNS fatigue by suppressing the mobilisation of FFA's (Davis, et al., 1992).

However, although studies using rats (Bailey, et al., 1992, 1993a, 1993b; Chaouloff, et al., 1985; Chaouloff, et al., 1986; Chaouloff, et al., 1987) have shown increases in TRP, 5-HT and 5 HIAA (a major metabolite of 5-HT) in the brain are associated with exercise, fatigue and reduced exercise performance, results from human studies are not conclusive. The use of 5-HT agonists has shown exercise time to exhaustion to be both improved (Struder, et al., 1998; Wilson and Maughan, 1992) and unaffected (Strachan, et al., 2004). Furthermore, there appears to be no improvement in exercise performance following administration of tryptophan (Stensrud, et al., 1992; van Hall, et al., 1995). Van Hall et al. (1995) fed participants drinks containing carbohydrate with either tryptophan or BCAA's added. Cycle time to exhaustion at ~70% W_{max} was not altered despite six- to seven-fold increases in plasma tryptophan (van Hall, et al., 1995). These results are in agreement with those of Stensrud et al. (1992) who also found no effect of L-tryptophan on running performance. In contrast an earlier study found running time to exhaustion at 80% VO_{2max} was improved by 49.4% with ingestion of L-tryptophan (Segura and Ventura, 1988). However, it has been suggested (Stensrud, et al., 1992) that the results of Segura et al. may have been due to poor methodology and a very heterogenous participant group.

These results appear to suggest that the role of serotonin in central fatigue during exercise may not be significant, however, because TRP uptake by the brain is expected to increase with increased plasma f-TRP during prolonged exercise, the suggestion has been made that brain serotonin increases due to exercise may become involved in central fatigue during physical activities lasting several hours (Nybo and Secher, 2004).

2.7.2. Maintenance of Cerebral Glucose Uptake

Another possible mechanism for central effect of carbohydrate ingestion has been postulated to be related to a direct increase in glucose availability to the brain (Nybo, et al., 2003). Under euglycaemic conditions, glucose is the

predominant substrate of the brain and even during hypoglycaemia the brain uses almost the same amount of glucose for energy (Wahren, et al., 1999). In the case of severe hypoglycaemia, possibly during prolonged exercise, the glucose supply to the brain could reach critically low levels causing cerebral dysfunction and central fatigue.

Nybo et al. (2003) measured the cerebral metabolism and blood flow of participants cycling at ~60% VO_{2max} for 180 minutes ingesting either a non-calorific placebo (Pl) or 6% carbohydrate (CHO) drink. Blood glucose was maintained during CHO but it decreased from 5.2 to 2.9 $mmol \cdot L^{-1}$ in Pl over the exercise period. In Pl glucose uptake decreased and although there was a small increase in the uptake of β-hydroxybutyrate, a decrease in oxygen consumption and carbon dioxide production suggested that uptake of ketone bodies and other substrates was insufficient to compensate for the reduced glucose uptake. RPE increased in Pl (~17 and ~13.5 in Pl and CHO respectively at 180 minutes) and in addition two subjects felt dizzy and struggled to maintain visual focus. This reduced glucose uptake together the increased RPE indicate that substrate availability for the brain was insufficient. This study provides evidence that glucose ingestion during prolonged exercise increases endurance by maintaining cerebral glucose uptake and decreasing central fatigue (Nybo, et al., 2003).

2.7.3. Depleted brain glycogen

Associated with the decrease in glucose availability to the brain, the depletion of brain glycogen may also be important in the development of central fatigue (Dalsgaard, et al., 2002). The metabolic ratio of oxygen uptake to glucose uptake by the brain is usually ~6:1; however, this ratio is reduced when glucose uptake increases in response to exhaustive exercise (Ide, et al., 2000). It has been suggested (Dalsgaard, et al., 2002) that since brain glycogen is reduced in rats following cerebral activation achieved by sensory stimulation through stroking the face (Madsen, et al., 1995; Madsen, et al., 1999; Swanson, et al., 1992), the additional glucose taken up by the brain may be used to replenish

brain glycogen. Dalsgaard et al. (2002) found that the metabolic ratio of the human brain only decreased when the will to exercise was intense and speculated that intense activity in cerebral regions caused the energy demand to exceed energy production, depleting the glycogen reserves. It was postulated that the depleted glycogen in turn increases the metabolic ratio as central fatigue ensues (Dalsgaard, et al., 2002). It has also been noted that the metabolic ratio normalises within a few minutes following exhaustive exercise and the time span relates to that in which it takes an athlete to regain motivation (Dalsgaard, 2006).

2.7.4. Hyperammonemia

A final possible mechanism of carbohydrate reducing central fatigue is through a reduction in hyperammonemia. Ammonia production results from a variety of pathways including amino acid catabolism (Wagenmakers, 1998) and production is increased when glycogen is depleted (Wagenmakers, et al., 1991) and/or systemic glucose availability is low (Snow, et al., 2000). Elevated levels of ammonia are toxic to the brain and can cause cerebral disruption and performance decrements during prolonged endurance exercise (Banister and Cameron, 1990).

Snow et al. (2000) found that prolonged sub-maximal exercise caused increases in muscle- and plasma-ammonia concentrations. Carbohydrate ingestion attenuated these increases by reducing the degradation of amino acids. Furthermore, carbohydrate ingestion has also been shown to reduce cerebral ammonia concentrations (Nybo, et al., 2005). Nybo et al. (2005) investigated the effect of 3 hours cycling at ~60% VO_{2max} on cerebral ammonia uptake with either carbohydrate or no carbohydrate supplement. Cerebral ammonia uptake and CSF and plasma ammonia concentration increased with exercise duration and the response was augmented in the placebo compared to when carbohydrate was ingested. Those subjects with high levels of circulating ammonia experienced most difficulty in completing the exercise trials. Furthermore, glucose ingestion decreased the RPE at the same time as it

attenuated the exercise-induced systemic and cerebral ammonia responses supporting the suggestion that cerebral ammonia accumulation may be related to central fatigue. However, the RPE's of some endurance trained participants increased later in exercise with no or negligible cerebral ammonia uptake demonstrating that other factors are involved in fatigue.

Because it is difficult to separate the contribution of central and peripheral factors to fatigue, it remains difficult to establish conclusively the means by which carbohydrate delays fatigue or enhances endurance performance.

3. Hydration, Fluid Intake and Endurance Performance

During exercise or heat exposure, total body water is lost even when water is readily available for drinking ad libitum (Greenleaf and Sargent II, 1965). Greenleaf and Sargent (1965) observed that subjects at rest maintained hydration status by drinking ad libitum but subjects resting in the heat and exercising in the cool and in the heat were hypohydrated at the end of a 4 hour experimental period despite having water available ad libitum. This phenomenon, known as voluntary dehydration has also been observed in soldiers walking in the desert (Adolph, 1947).

Daily water loss is ~2.6 L (Astrand and Rodahl, 1986), however this rate can increase considerably during exercise or exposure to warmer climates (Sawka and Pandolf, 1990). Physical exercise increases the body's metabolic rate and therefore heat production. This excess heat must be dissipated to maintain heat balance. Heat is lost from the body via radiation, conduction and evaporation and the relative contribution of each depends on the environmental conditions (Sawka and Pandolf, 1990). The main sources of heat loss during exercise are through an increase in cutaneous blood flow which allows heat to be transported from the core to the skin for dissipation and through the evaporation of sweat. In hot environments where radiation maybe limited or reversed large amounts of water can be lost through sweating to facilitate cooling by evaporation (Wenger, 1972).

Although sweat rates during exercise vary according to the ambient temperature, humidity, the velocity of the air and the intensity and duration of exercise, they frequently exceed 1 $L \cdot h^{-1}$ during prolonged exercise in temperatures of 20-25°C (Coyle and Hamilton, 1990). The highest sweat rate recorded in literature is 3.7 $L \cdot h^{-1}$ (Armstrong, et al., 1986). These high sweat rates, in addition to voluntary dehydration, lead to a reduction in body fluids and a state of hypohydration.

Hypohydration during exercise adversely affects the cardiovascular (Below, et al., 1995; Hamilton, et al., 1991; Montain and Coyle, 1992a, 1992b; Nadel, et al., 1980) and thermoregulation (Fortney, et al., 1981; Fortney, et al., 1984; Montain and Coyle, 1992a, 1992b; Nadel, et al., 1980; Sawka, et al., 1985) systems of the body and adversely affects exercise capacity (Caldwell, et al., 1984; Saltin, 1964a, 1964b) and performance (Armstrong, et al., 1985; Barr, et al., 1991; Maughan, et al., 1996; Walsh, et al., 1994; Webster, et al., 1988).

3.1. Measurement of Hydration Status

The hydration status of an athlete can be easily ascertained in the laboratory or field setting by measuring urine specific gravity (Oppliger, et al., 2005) and urine colour chart (Armstrong, 2000, 2005; Armstrong, et al., 1994).

Armstrong et al. (1994) measured urine colour (U_{col}) urine osmolality (U_{osm}) and urine specific gravity (U_{sg}) in different situations providing varied environmental factors. U_{osm} was found to be linearly related ($r=0.97$, $p<0.0001$) to U_{sg} and U_{col} was strongly related to both U_{osm} and U_{sg} ($r=0.82$ and 0.80 respectively and both $p<0.0001$). It was concluded that in research settings U_{osm} and U_{sg} could be used interchangeably for accurate measurements of hydration status and U_{col} could be used in field-type settings where close estimates are acceptable.

3.2. Effect of Hypohydration on Exercise Capacity and Performance

A number of studies have found the maximal work capacity (Caldwell, et al., 1984; Saltin, 1964a, 1964b) and exercise performance (Armstrong, et al., 1985; Maughan, et al., 1996; Walsh, et al., 1994; Webster, et al., 1988) of athletes to be reduced when in a state of hypohydration. These performance decrements exist across different exercise modalities and regimens.

Most studies have used a heated environment to make any effects of hypohydration on performance and physiological responses more pronounced. In an ultra-endurance study, seven out of eight participants cycling at 55% VO_{2max} had higher RPE's and heart rates and were unable to complete six hours of exercise when no fluid (NF) was ingested compared to water (W) or saline (S) ingestion (Barr, et al., 1991). However, all but one of the participants completed the ride in both W and S. Furthermore, Walsh et al. (1994) studied the effect of exercise-induced dehydration on high-intensity cycling performance. Participants cycled for 60 minutes of exercise at 70% VO2peak before completing a ride to exhaustion at 90% VO2peak whilst ingesting fluid or no fluid. In the no fluid trial, participant's experienced low levels (1.8%) of dehydration which resulted in increased RPE and a reduced cycle time to exhaustion during the performance ride.

Decrements in exercise capacity and performance due to hypohydration are also observed in a more neutral environmental temperature. Armstrong et al. (1985) examined the effect of diuretic-induced dehydration on competitive running performance in non-warm environmental conditions of 15.7°C and 31.8% humidity. A 2% decrease in bodyweight resulted in decreased running velocity and increased performance time in a 5000m, 10,000m and maximal treadmill test. It was suggested that hyperthermia altered anaerobic function, increased perception of effort or a combination thereof and was responsible for the decrements. Webster et al. (1988) found that typical dehydration practises of wrestlers reduced body weight by 4.9% and resulted in deleterious effects on strength, anaerobic power and capacity, aerobic power and lactate

measurements. Additionally, Maughan et al. (1996) observed an improved exercise time to exhaustion at 70% VO_{2max} when fluids were ingested compared to no fluids.

The detrimental effects of dehydration on exercise performance and capacity are widely accepted but the level of dehydration at which these effects occur has not yet been established (Noakes, 2007). Difficulty exists in interpretation of studies since many have not used exercise as the stimulus for dehydration but rather dehydration has been induced prior to exercise by diuretics, exercise and heat which may themselves have been the real cause of the impaired performance (Noakes, 2007). The extent to which performance is affected may also depend on exercise intensity and the environment conditions which in turn affect heat balance; the quantity of heat produced and heat lost. Furthermore, an estimation of the level of dehydration is difficult to quantify since it is regularly expressed in terms of weight loss during exercise (Armstrong, et al., 1985; Barr, et al., 1991; Murray, et al., 1987a; Walsh, et al., 1994). This weight loss cannot be solely attributed to water loss as a proportion of it will result from fuel that is irreversibly oxidised during metabolism (Noakes, 2003). However, it is clear that fluid ingestion does produce physiological effects that could aid performance and that these benefits increase with the duration of exercise (Noakes, 2007).

3.3. Physiological Effects of Hypohydration

3.3.1. Impaired Thermoregulation

Body temperature increases during exercise when the body is an a state of hypohydration due to a failure to replace fluids (Below, et al., 1995; Fortney, et al., 1981; Fortney, et al., 1984; Hamilton, et al., 1991; Maughan, et al., 1996; Montain and Coyle, 1992a). Exercise-induced dehydration impairs the body's thermoregulatory mechanisms by reducing skin blood flow (Fortney, et al., 1984; Montain and Coyle, 1992a, 1992b; Nadel, et al., 1980; Saltin, 1964b) and sweat rate (Fortney, et al., 1981; Fortney, et al., 1984; Sawka, et al., 1985)

which limits heat loss from evaporation and further increases body temperature (Sawka, et al., 1985).

Fortney et al. (1981) observed significantly reduced sweating rates (33.9 ± 6.0 and 25.5 ± 4.8 $g \cdot min^{-1}$ in control and hypovolemia respectively) in participants with a diuretic-reduced blood volume during exercise. The slopes of the sweating rate to oesophageal temperature relationship were also reduced over inactive muscle sites indicating reduced for each increase in temperature. Sweat rates during 140 minutes of exercise were also reduced in participants' hypohydrated by an exercise-heat regimen (Sawka, et al., 1985). Participants in the study of Sawka et al. (1985) were hypohydrated to 3, 5 and 7% of their body weight. Sweating rates were systematically decreased at any given core temperature with increasing levels of hypohydration. Other studies (Montain and Coyle, 1992a, 1992b) have shown no changes in sweat rate with increasing levels of hypohydration, however, this may be due to sweat collection methodology that may not have been sensitive enough to detect changes.

In addition to a reduction in sweat rate which impairs thermoregulation by reducing heat dissipation via evaporation, hypohydration also attenuates cutaneous blood flow. Nadel et al. (1980) observed an upward shift in the threshold for cutaneous dilation of participants exercising whilst hypohydrated (36.9 and 37.3°C for normally- and hypo-hydrated conditions respectively). Furthermore, maximal cutaneous blood flow was also much lower in the hypohydrated trial (11.5 $ml \cdot min^{-1} \cdot 100ml^{-1}$) compared to control conditions (18.6 $ml \cdot min^{-1} \cdot 100ml^{-1}$). Because an increase in the rate of temperature increase accompanied a reduction in cutaneous blood flow it was concluded that the hyperthermia that occurs in hypohydrated individuals during exercise in the heat is due, at least partly, to changes in skin blood flow. Participants exercising whilst dehydrated also exhibited reduced maximal cutaneous blood flow in the study of Fortney et al. (1984). This was accompanied by a reduction in the slope of the cutaneous blood flow to oesophageal temperature relationship indicating a reduced cutaneous blood flow for any increase in body temperature. Montain and Coyle (1992a) measured forearm blood flow (FBF) as an indication of skin blood flow. Fluid replacement during 2 hours of cycling at

~65% VO_{2max} increased FBF 16% between 30 and 105 minutes of exercise and was 17-20% higher compared with the no fluid trial after 105 minutes. This reduced cutaneous blood flow when hypohydrated can increase heat storage by decreasing heat transfer from the body core to the periphery and reduce the evaporation of sweat from the skin surface.

3.3.2. Impaired Cardiovascular Function

A reduction in body fluid affects cardiovascular function during exercise causing a reduced stroke volume and increased heart rate (Hamilton, et al., 1991; Montain and Coyle, 1992a, 1992b; Nadel, et al., 1980; Saltin, 1964b; Sproles, et al., 1976) and reduced cardiac output (Hamilton, et al., 1991; Montain and Coyle, 1992a, 1992b; Nadel, et al., 1980).

Nadel et al. (1980) observed increased body temperature and heart rate and decreased stroke volume and cardiac output in participants exercising at 55% VO_{2max} in warm conditions whilst hypohydrated. A similar relationship occurred in lower environmental temperatures in a study by Hamilton et al. (1991) when participants cycled for 2 hours at 70% VO_{2max} in a laboratory maintained at 22°C. Stroke volume and cardiac output were reduced when no fluid was ingested; however, when sufficient fluid was ingested to replace body water losses, euhydration was maintained and reductions in stroke volume and cardiac output were prevented. Montain and Coyle (1992b) examined the effect of graded dehydration on cardiovascular drift and found that the level of hyperthermia and the degree of cardiovascular drift were directly related to the change in bodyweight and dehydration accrued whilst exercising at ~65% VO_{2max}.

Loss of body water through sweating reduces blood volume and therefore stroke volume. With increased body temperature during exercise, the circulatory system must maintain the supply of blood and hence oxygen delivery to the working muscles and simultaneously supplies blood to the skin to enhance heat dissipation. Heart rate increases help maintain cardiac output but as dehydration increases, sweat rate and cutaneous blood flow are reduced

increasing thermal strain on the body and reducing exercise performance. To prevent this increase in circulatory and thermal strain and decreased exercise capacity and performance, dehydration must be avoided by effective fluid replacement throughout prolonged exercise.

3.4. Fluid Replacement during Prolonged Exercise

Dehydration has the potential to reduce exercise performance under normal environmental conditions. Increased temperature and humidity will exacerbate the physiological responses to reduced body fluid. When fluid is replaced during exercise, these negative effects of dehydration can be attenuated (Barr, et al., 1991; Below, et al., 1995; Hamilton, et al., 1991; Maughan, et al., 1996; Montain and Coyle, 1992a, 1992b; Walsh, et al., 1994), however, the composition and quantity of fluid to be replaced has been debated.

3.4.1. Composition of Replacement Drinks

As suggested earlier, both water and carbohydrate facilitate performance by maintaining euhydration and high carbohydrate-oxidation rates respectively. Consequently, various studies have attempted to establish the ideal composition of a fluid replacement drink appropriate for prolonged exercise in terms of fluid, electrolytes and carbohydrate.

3.4.1.1 Carbohydrate content of fluid replacement drinks

It has been suggested that during prolonged exercise in the heat, fluid delivery will be impaired by the carbohydrate content of a fluid supplement (Costill and Saltin, 1974). However, recent evidence suggests that carbohydrate ingested as part of a fluid supplement does not hinder fluid absorption (Below, et al., 1995; Maughan, et al., 1996; Murray, et al., 1987a; Yaspelkis and Ivy, 1991). In a study by Murray et al. (1987a) there were no changes in circulatory or thermoregulatory responses when water and three carbohydrate mixes (5% glucose polymer, 6% glucose/sucrose, 7% glucose polymer/fructose) were ingested during intermittent exercise. No differences occurred between trials in

bodyweight, mean sweat rates, heart rate, rectal and skin temperatures, plasma volume and plasma osmolality. These findings suggest the carbohydrate content of fluid replacement solutions has little effect on the maintenance of fluid homeostasis (Murray, et al., 1987a). Yaspelskis and Ivy (1991) also found no difference in markers of thermoregulation during two hours cycling at ~50% VO_{2max} when water, a 2% and 8.5% glucose polymer solution were ingested. Rectal temperature, heart rate and plasma volume shifts were the same between trials supporting the contention that the carbohydrate content of solutions ingested during exercise does not adversely affect temperature regulation.

Below et al. (1995) found the effects of water and carbohydrate to be independent and additive. Participants completed a performance test following 50 minutes of cycling at 80% VO_{2max} and showed a 6% improvement in the both carbohydrate and water trials. A 12% improvement occurred in the combined carbohydrate and water trial. These findings are in agreement with those of Maughan et al. (1996) who observed improved endurance performance when water was ingested and further increases with the ingestion of glucose-electrolyte solutions.

Several authors suggest that the carbohydrate content of a fluid replacement drink should not exceed 6-8% (Convertino, et al., 1996; Gisolfi and Duchman, 1992) as it has been suggested that solutions greater than 10% carbohydrate will cause a net movement of fluid into the intestinal lumen because of their high osmolality (Maughan, 1992). This suggestion is in agreement with the findings of Rehrer et al. (1992b) in which no differences in the oxidation of the maltodextrin and glucose when participants ingested 17% glucose and maltodextrin solutions whilst exercising at 70% VO_{2max} for 80 minutes. However, net water absorption was observed with the polymer, whereas net secretion was observed with the hypertonic free-glucose solution suggesting that during exercise where water absorption is important, maltodextrin may be a preferential source of carbohydrate in drinks. More recent observations have shown that drink osmolality can be kept well below the osmolality of body fluid with the use of maltodextrins and long-chain glucose polymers (Rowlands, et

al., 2005) which would permit ingestion of large quantities of carbohydrate without increasing solution osmolality. Furthermore, as mentioned earlier in the review, Shi et al. (1995) have found that solutions containing multi-transportable carbohydrates stimulate greater water absorption for a given luminal osmolality and the effect of hyperosmolality on water absorption is reduced.

3.4.1.2. Salt and Electrolyte Content

Sodium chloride is the main salt lost in sweat and although the quantity varies between and within individuals depending on collection methodology, state of training and heat acclimation, the normal concentration is ~20-80 and ~20-60 $mmol \cdot l^{-1}$ of sodium and chloride respectively (Maughan, 1992). Other electrolytes such as potassium and magnesium are lost at a lesser rate and whilst the rate is high compared to plasma levels, they represent a small percentage of total body stores (Costill, et al., 1976). Sodium is therefore considered to be the only electrolyte that it is necessary to include in sports drinks (Coyle, 2004; Horswill, 1998; Maughan, 1992, 1997) and it is usually added in the form of sodium chloride (Maughan, 1992). Sodium stimulates thirst (Astrand and Rodahl, 1986), increases fluid uptake which maintains body fluid balance (Wemple, et al., 1997), and aids in carbohydrate uptake in the intestines (Leiper, 1998; Olsen and Ingelfinger, 1968). Salt is therefore considered an important component of oral rehydration solutions and 20-30 $mmol \cdot l^{-1}$ is considered an appropriate quantity for inclusion (Convertino, et al., 1996; Gisolfi and Duchman, 1992; Wemple, et al., 1997) in sports drinks for exercise lasting more than 3 hours. It should be noted, however, that there is currently no evidence to suggest that the inclusion of sodium in sports drinks is a physiological necessity for exercise of any duration (Noakes, 2007).

3.4.2. Fluid Quantity

Differences between individuals, research methodologies, exercise intensity and duration and environmental conditions appear to make it very difficult to establish a precise guideline for the appropriate quantity of fluid to be replaced during prolonged exercise (Maughan, 1997). The 1996 ACSM position stand on

exercise and fluid replacement suggests that athletes should attempt to consume fluids at a rate that is sufficient to replace all the water lost through sweating, or consume the maximal amount that can be tolerated (Convertino, et al., 1996). The suggestions of other studies also advise that fluid should be ingested at a rate that equals the rate of fluid loss from sweating (Barr, et al., 1991; Coyle and Montain, 1992; Montain and Coyle, 1992b; von Duvillard, et al., 2004) as it is in this state of euhydration that causes least disturbance in thermoregulation and circulatory responses to exercise. However, it has been suggested (Noakes, 2002) that a danger exists in encouraging individual's to drink sufficient to replace sweat losses or as much as can be tolerated as this can lead to hyponatraemia caused by voluntary over drinking. Observations made during the 1996 (Speedy, et al., 1997) and 1997 (Speedy, et al., 1999) New Zealand Ironman Triathlon have shown that during endurance races it is those who gain weight that have the reduced sodium concentration of hyponatremia. During the 1997 New Zealand Ironman ultra-distance triathlon 18% of the race finishers were hyponatremic. Of these, 19% were severely hyponatremic (<130 $mmol \cdot L^{-1}$) and 73% of athletes with severe hyponatremia had either gained or maintained pre-race weight (Speedy, et al., 1999). When fluid intake was limited during these events, the incidence of symptomatic hyponatremia was reduced (Speedy, et al., 2000). Furthermore lab studies have shown that changes in weight during exercise are inversely proportional to changes in plasma sodium concentration (Noakes, et al., 2001; Speedy, et al., 2001). Safer rates of drinking of between 500 and 1000 $ml \cdot h^{-1}$ or ad libitum according to thirst (Noakes, 2007) have been proposed (Noakes, 1995; Speedy, et al., 2000). This is in agreement with Gisolfi and Duchman (1992) who recommend that for exercise greater than three hours in duration, of which endurance mountain biking is an example, that 500-1000 $ml \cdot h^{-1}$ should be ingested.

In summary, a recent review (Loo, et al., 2002) has concluded that the sodium-glucose co-transporter acts as a low conductance water channel and may be responsible for 35% of the steady state water flow across the brush border membrane of the small intestine. This underlines the fact that any solution

aimed at rehydration must contain both glucose and sodium. A solution containing water, carbohydrate and sodium will be effective in meeting the nutritional and fluid requirements of the body during prolonged exercise. The relative quantities of each in any given solution will depend upon factors such as environmental conditions, exercise intensity and duration and the individual's nutritional status.

For prolonged endurance exercise fluid may be ingested at a rate of 500-1000 $ml \cdot h^{-1}$ and should contain 20-30 $mmol \cdot l^{-1}$ sodium. Although several authors suggest that the optimal carbohydrate content of a solution should be 6-8%, in view of the recent research into multi-transportable carbohydrates, it may be preferential to ultra-endurance performance to ingest multi-transportable carbohydrates a rate which maximises their oxidation as outlined earlier in the review. These findings suggest that fluid replacement solutions containing multi-transportable carbohydrates at rates (1.5 $g \cdot min^{-1}$ and 90 $g \cdot h^{-1}$) estimated to maximise the availability of ingested carbohydrate in a quantity of fluid (800 $ml \cdot h^{-1}$) that will not cause fluid overload, whilst being of greater concentration than recommendations (11.25%) may not limit fluid uptake.

Methods

The investigation comprised two attempts at successful collection of data in the field using two different study designs. The first attempt was a randomised controlled design and was organised to take place at the end of the mountain bike racing season in autumn 2006. However, on the night before and morning of the event a storm occurred with cold and heavy rain and the course conditions were considered unsafe for performance riding with an unclear performance outcome being the likely result. As a result the race was cancelled. In the evaluation of this event and its study design, a preferential design was organised for the second attempt to be held the following summer. Details of the initial controlled study design can be found in Appendix 1.

Study Design

Participants

Fourteen; male (10) and female (4), mountain bikers involved in regular training participated in this study. Cyclists were 32.9 ± 8.7 years, 68.8 ± 9.4 kg, training for at least 8 hours per week and racing regularly. Six cyclists attended a pre-event meeting and course ride 4 days prior to race one and all cyclists attended a pre-race briefing on the day of both races. Those cyclists who were unable to attend the course ride were provided with a map of the course (Appendix 2) and were asked to familiarize themselves with the course by riding around it prior to racing at a time that was convenient to them. All but one of the riders completed this requirement. After reading the study information sheet (Appendix 3), all cyclists completed a General Health Questionnaire (Appendix 4) to screen for prohibiting medical factors. All cyclists were made aware of the physical and psychological discomfort associated with competition, the possibility of gastro-intestinal discomfort from the ingestion of high quantities of carbohydrates and were informed of their right to withdraw from the study at any time. All cyclists signed a written, informed consent form before racing (Appendix 5). This study was approved by the Massey University Ethics Committee: Southern A, 06/04.

Study Protocol

The study had a double-blind, cross-over design with 2 treatments (Figure 1). Cyclists were ranked according to performance in local cross-country events and placed into two matched groups on alternate rankings. The groups were randomly assigned alternate treatments over two cross-country mountain bike events. The two events were 7 days apart. For four days before the event cyclists completed a training diary (Appendix 6). On days four and three before the race, cyclists could complete regular training but were instructed to repeat this exactly on the equivalent days during the following week. On days two and one before the race, cyclists were required to complete either light training or no training at all. A food diary (Appendix 7) was also completed for the day before and the day of the event. For breakfast and lunch on the day before the event, cyclists were able to eat normal foods but were asked to eat exactly the same foods and quantity the following week. A prescribed menu (Appendix 8) was followed for meals within 24 hours of the race. Starting with dinner the day before, a minimum of 6.5 g carbohydrate per kilogram bodyweight (~490 g for 75 kg) was consumed to ensure all cyclists started the race with full glycogen stores. Cyclists were required to abstain from alcohol consumption in the 24 hours prior to the race and on race day to abstain from the consumption of any stimulants such as caffeine-containing drinks. From two hours before the race, only water was consumed.

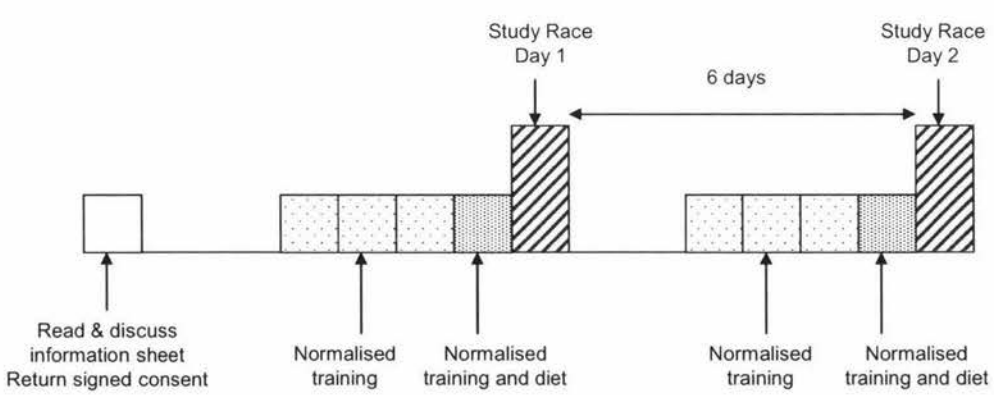

Figure 1 Study design and control of lead-in diet and training

Cyclists arrived at the race venue at approximately 1630 hours on race day. On arrival a urine sample was provided and body mass was recorded in race clothing, following which cyclists were free to prepare their equipment and warm-up as necessary. All cyclists attended a pre-race briefing at 1730 hours which included safety information, a reminder of race etiquette, re-emphasis of experimental protocol to be used and an outline of the course. At 1745 hours cyclists collected their camelbak water carriers which contained the required drink for laps 1 and 2 and at the same time consumed an initial drink bolus of 200 ml. At 1800 hours cyclists assembled on a start line for a mass start. At the end of laps two and four, cyclists entered a feeding area next to the start/finish line and had their camelbak refilled whilst completing scales to measure the ratings of perceived exertion and gastrointestinal discomfort experienced at that time. After crossing the finish line the cyclists completed the scales, provided a post-race urine sample and had body mass measured. Following completion of the experimental protocol, cyclists were free to eat and drink normally and to leave.

Exercise Protocol

The course consisted of medium-grade single track, 4WD track and some short sections of tar seal. Approximately 75% of the course was the same as that used in a UCI (Union of Cyclist International) graded race two months following. The terrain included many steep uphill sections of between five seconds and five minutes duration to facilitate glycogen depletion. The event was run in a similar fashion to local club events, with directional signage, public notification, marshals, and road signs where necessary. Normal mountain bike race rules and etiquette applied and first aid stations were provided. The race course was designed to fit the duration criteria of an international mountain bike event with a target winning time of 2 hours and 15 minutes and an approximate average time of 2 hours and 30 minutes. However, due the mixed gender status (females are generally ~20% slower than males) and fitness ability of the cyclists volunteering (ranging from New Zealand representatives to good regional level Masters grade) we calibrated rider ability with the required number of laps to complete the race in around 2 hours 30 minutes (final range 2 hours 10 minutes

to 2 hours 40 minutes). Consequently, some cyclists completed three, four or five laps based on time differentials generated from results in the 2005 Port Nicholson and Poneke Cycling Club Mountain Bike Cross-country Racing Series and participation in the Karapoti Classic; eight cyclists completed five laps, four cyclists completed four laps and two cyclists completed three laps. Designating a set number of laps to each cyclist ensured that they all completed a set amount of work that was appropriate for their individual capacity, over a specified time in order to obtain a homogenous dose-duration physiological response to the treatment. There was a strong element of competition amongst those cyclists completing the same number of laps.

Both races were performed under similar environmental conditions. At the start of the race (1800 hrs), Wellington Airport readings (3km from the race venue) of air temperature, relative humidity and wind strength (direction) were 15°C, 64%, 6 knots (S) and 15°C, 56%, 5 knots (S) for Race 1 and Race 2 respectively (personal communication, B. McDavitt, NZ Meteorological Service). Overhead, there were scattered, high-level clouds but it was predominantly sunny and the track was hard and dry.

Experimental Drinks

The two drinks were a maltodextrin and fructose (MF) intervention and a maltodextrin and glucose (MG) control. The analytical composition of the drinks is shown in Table 3. Calculated drink quantities were based on the energy and hydration needs of the fastest (five lap) cyclists, but calibrated down accordingly based on pre-established rider ability. Including the initial bolus, the quantity of drink provided to each athlete was 800 $ml \cdot h^{-1}$ per 75 kg bodyweight. This was calculated to be 400 ml per lap per 75 kg of body mass based on the expectation that each lap would take the approximately 30 minutes to complete. The drinks were designed to provide carbohydrate at a rate of 1.5 $g \cdot min^{-1}$ for a 75 kg individual. As the three and four lap cyclists were also provided with fluid on a per lap basis, the actual quantity of solution ingested over a set time period was less, but was relevant to their individual requirements. Approximately 1622

± 463 L of solution was ingested during the race providing 1.38 ± 0.19 g·kg·min^{-1} carbohydrate. Pilot tests were completed on the drinks prior to use in the event. Three competitive cyclists, not involved in the study, sampled both drinks at the required rate during a three-hour training ride and provided comments on the drinks' palatability and any gastro-intestinal discomfort.

Table 3. The analytical composition of the test drinks

	MG	MF
g·L^{-1}		
Maltodextrin	75	75
Glucose	37.5	-
Fructose	-	37.5
NaCl	1.17	1.17
KCl	0.37	0.37
$MgSO_4$	0.24	0.24
Ca_3PO_4	0.43	0.43
Citric Acid	0.4	0.4
Water	884.89	884.89
Lime Flavouring (ml)	40	40
Osmolality (mOsm·kg^{-1})	400	423

Measurements

RPE and GI Scales

Selected perceptual responses such as ratings of perceived exertion (leg muscle strength, tiredness, effort of cycling) and gastro-intestinal discomfort (abdominal cramps, nausea, diahorrea) were measured before, during and after the races. The scales used were modelled on Borg's CR10 scale (1970, 1985, 1988) (cited in Borg 2001) (see Appendix 9 for original and modified Borg CR10 scales). The CR10 scale is based on experiments which showed that the magnitude of perception is positively accelerating and can be described by a power function with an exponent of 1.6 (Borg, 2001). Scale ratings were later converted in a spreadsheet to a linear proportional scale with units 0 (no sensation) to 100 (maximal sensation) for ease of comparison, presentation and statistical appropriateness.

Cyclists were asked to make a horizontal pencil mark on a scale with regularly spaced written verbal anchors. The verbal anchors differed between ratings of perceived exertion and can be seen in Appendix 9 but as an example perceptions of tiredness were rated Fully Fresh, Extremely Fresh, Very Fresh, Fresh, Moderate, Tired, Very Tired and Completely Tired. Cyclists were instructed to treat the scale as continuous and to mark anywhere on the line not just at the divisions assigned by the verbal anchors. During collation of the scales the rider's mark was recorded using subdivisions placed between each of the discrete numerical values to provide fractions.

Hydration Status

The osmolality, specific gravity and colour of both pre- and post-race urine samples were measured and with pre- and post-race body mass were used to give an indication of hydration status. Both before and after racing, body mass was measured using electronic scales (model UC-321, A&D Scales, A&D Co. Tokyo, Japan). Mid-flow urine samples were then collected and a subjective colour rating given using the Urine Colour Chart of Armstrong et al. (1994). The samples were stored on ice for no more than 15 hours before the urine specific gravity was measured using a SUR-NE Refractometer (Atago Co. Ltd. Japan). The samples were then frozen and stored for up to one month. Osmolality was measured by freezing point depression at the Nutrition Laboratory, Institute of Food Nutrition and Human Health, Massey University, Palmerston North Campus.

Race Timing

Cyclist's lap and final race times were recorded using a digital stopwatch (model DT2000Digi, sports instruments, Pesotec Ltd., Hong Kong). Split times and rider's race numbers were taken as they crossed the entrance line to the feed station at the end of each lap and at the end of the race. Cyclists were asked to record the time of any stoppages such as mechanical faults or accidents so that

these could be deducted from their final race time. Time of entry into the feed station was also videoed as a back up.

Statistical Analysis

Sample Size

Although fourteen athletes participated in the races, the results of only ten (7 male and 3 female) were used in the analysis. Data from four athletes was excluded for a variety of reasons; two cyclists had mechanical failure (broken chain and slashed tire), one rider did not complete the study protocol properly and one rider completed the race course at an early time of day due to work commitments and experienced different track conditions to the other cyclists in race two because of light overnight rain.

Statistical Models

The effect of drink formulation on race outcome data was analysed with appropriate mixed linear and other repeated-measures models in the proc mixed utility in SAS (Statistical Analysis Systems, Vers 9.1 Cary, NC). All race time, urine, and hydration data was analysed after log-transformation to reduce the effects of non-uniformity of error; this method generates back-transformed effects expressed as a percentage. The cumulative final race time outcomes were derived from the interaction between the fixed effects race order and treatment (drink formulation). The random effects for this analysis were the participant and the interaction between participant and change in condition (assumed extra variance for the MF condition). The second analysis for the performance outcome was a linear model applied to the effect of lap time by lap, where lap was adjusted to give all cyclists the same start and finish numeric finishing values despite the different number of lap completed. The scale was measured from 0-60 with each corrected lap (lapcorr) value being the proportion of 60 represented by the actual laps completed as a fraction of the total number to be completed. For example, for those cyclists completing five laps, the lapcorr values were 12, 24, 36, 48 and 60 compared to cyclists completing three

laps with lapcorr values of 20, 40 and 60. For this linear model, the fixed effects were the interaction between race order and treatment, with the slope effect derived from interaction with lapcorr coded as a numeric effect. The random effect was subject identity only; because the model would not run with the interaction between subject and change in condition. The perceptual response (ratings of perceived exertion and gastro-intestinal discomfort) to drink formulation was determined using a linear model as described for lap time, but with subject and the interaction between subject and change in condition. Post-pre-race changes in measurements of hydration status (urine measurements and body mass) were analysed within the model; subject identity and the interactions between subject and race order and drink condition were the specified random effects for the appropriate effects.

The measures of centrality and spread for subject descriptive and raw urine data are reported as raw means and standard deviations to allow direct comparison with the results of Armstrong et al. (1994) with respect to the latter. Race time and measurements of hydration status data were reported as back log-transformed least-squared means, with standard deviations as percentage values. For example, for a mean cumulative performance time of 8541.82 seconds with a between-subject standard deviation of 6.97%, the typical variation is 8541.82 ÷ 1.07 to 8541.82 x 1.07 or 7985.25 to 9137.18 seconds. With the ratings of perceived exertion and gastro-intestinal discomfort, the effects are expressed as a proportion of maximal sensation.

As suggested by Batterham and Hopkins (2006), 90% confidence limits (CL) or the ± confidence interval have been used to show the precision of estimates and the statistical inference has been shown using the analysis method and descriptors provided. If both the chance of a substantial benefit and detriment are >5%, then the stated effect is described as being unclear. When this is not the case, the chances of substantial benefit or detriment were inferred as follows: <1%, almost certainly not; 1-5%, very unlikely; 5-25%, unlikely; 25-75%, possible; 75-95%, likely; 95-99%, very likely; >99%, almost certain (Batterham

and Hopkins, 2006). An effect is described as being trivial when the chance that a benefit or detriment is <5% and the chance of the effect being trivial is greater than the >5% opposite effect, detriment or benefit. Use of qualitative inferences in respect to the practical importance of a finding characterised by confidence limits and the smallest worthwhile effect accentuates precision of the estimation rather than the strength of evidence against the null hypothesis (*P* value) (Sterne and Smith, 2001).

Meaningful inferences were obtained using an excel spreadsheet (Hopkins, 2002a). Where a within-athlete coefficient of variation is available, as in mountain bike performance time over a series of races, this value has been halved and used as the threshold for clinical chances (Hopkins, et al., 1999). Paton and Hopkins (2006) have previously calculated the within-athlete coefficient of variation for World Cup cross-country mountain bikers to be 2.4%, therefore the threshold for smallest worthwhile effect used is 1.2%. However, to ensure that the coefficient of variation used in our study would represent as closely as possible the cyclists involved, we calculated a specific coefficient of variation from the official results of the Port Nicholson and Poneke (PNP) Mountain Bike Race Series 2006. The five-race series, in which most of our cohort competed, was similar in nature and time duration to our study races and being held during August-November 2006, immediately preceded the study. Series race times of cyclists who had completed at least three of the five races from the senior and masters 1, 2 and 3 categories, in which most of our cyclists race, were obtained from the published database on the PNP website (Port Nicholson and Poneke Cycling Club, 2007). As a result of these criteria, no female results were used in the analysis.

To determine the coefficient of variation for race time of our local sample, the mixed linear model procedure (Proc Mixed) of SAS (Statistical Analysis Systems, Vers 9.1 Cary, NC) was used based on the method of Paton and Hopkins (2006). Race times were log transformed before the analysis to give the resulting variance in the form of a percent of the mean or the coefficient of

variation as described previously. Changes in mean race time between races due to differences such as distance, terrain, environmental factors and rider category (e.g. Elite, Masters 1) were controlled as fixed effects. The within-athlete variability between races was then determined from the square root of the residual variation from the random effects model, where the specified random effects were rider identity and the interaction between rider identity and race. When describing estimated effects such as those calculated for the ratings of perceived exertion and gastro-intestinal discomfort and measurements of hydration status where the smallest worthwhile change is not known, the method of Cohen was used whereby the smallest worthwhile effect is 0.2 x the standard deviation for the control condition. Meaningful inferences were obtained using the excel spreadsheet provided by Hopkins (2002a).

Pearson Product Moment Correlations were calculated between the raw urine measurements using excel. At the 90% confidence level, meaningful inferences regarding the strength of these relationships were obtained using descriptors (Hopkins, 2002b).

Results

Performance

Race Completion Time

The maltodextrin and fructose (MF) solution enhanced performance time by 1.8% (90% confidence interval: ±1.8%). For this outcome, the likelihood of a substantial benefit to performance based on the smallest worthwhile difference derived from variation in World Cup mountain bike performance is 72%, while harm is 1%. The coefficient of variation for PNP Club Mountain Bikers during the 2006 cross-country series was 3.6%. The value 1.8% may therefore be used as an estimate for the smallest worthwhile effect of an intervention on performance time in this group resulting in a 50/50 chance of beneficial or trivial outcome. With the treatment effect factored out, performance time was 2.8% faster (±1.8%) in race 2 compared with race 1.

Lap Time

The effect of the drink composition on lap time is shown in Figure 2. Consistent with the analysis of race completion time, overall average lap time (position effect) derived from the linear model was 1.5% (90% confidence interval: ±1.2%; 67% likelihood of a substantial reduction) faster in the MF condition, relative to the MG condition. Expressed in terms of the adjusted lap value, the increase in lap time (slope), or the effect of fatigue in slowing race time from completion of the first lap to the end of the race was 10.7% in MG (±2.8%) and 9.7% in MF (±2.8%), which represents a 2.6% and 2.3% slowing (±0.6%) of race lap time for every quarter of the race completed for the respective conditions. The corresponding effect of drink composition was a 0.9% reduction (±3.5%; unclear) in the fatigue rate in the MF condition, relative to the MG condition.

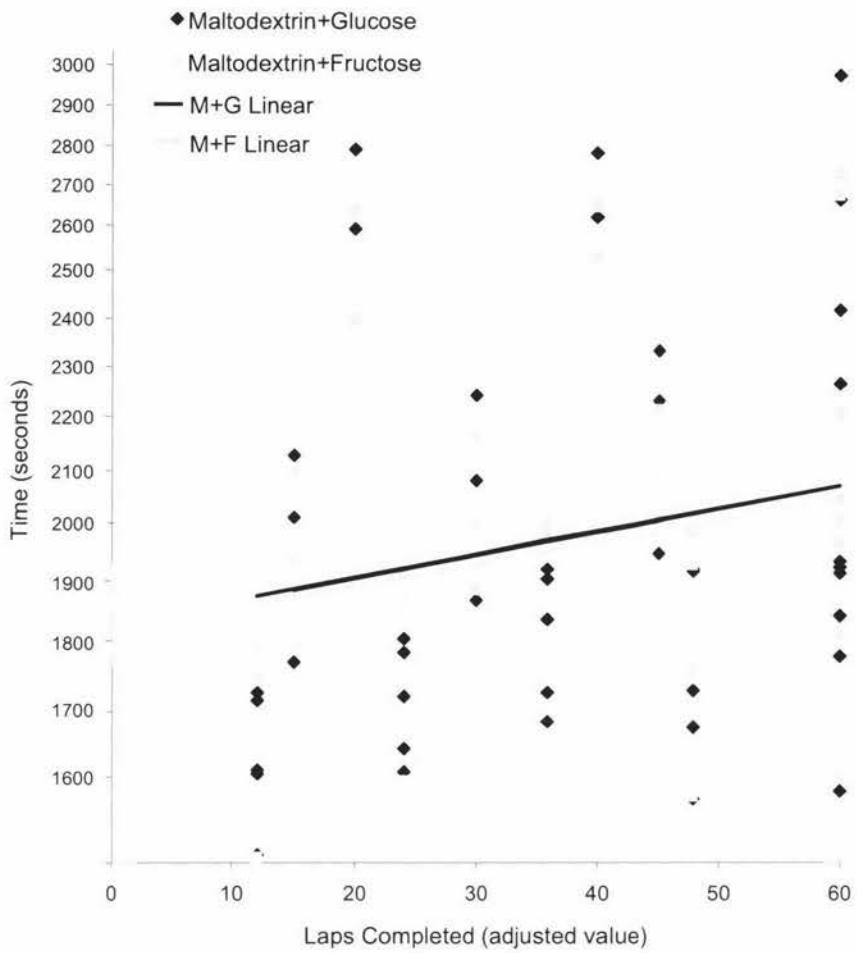

Figure 2. The effect of drink composition on lap time. Data are back log-transformed lap times (average for athletes at the end of each timed lap) with the corresponding overall treatment effect derived from the linear model

Mechanisms Analysis

The relationship between change in abdominal cramp and the change in lap riding time is shown in Figure 3. From the covariate mechanism analysis, for every 1% change in abdominal cramp, lap time increased by 0.14% (±0.10%). The enhancement with MF on lap time performance derived from the average lap time immediately preceding psychometric data collection points, relative to MG, was a 1.3% enhancement (±1.4%, likelihood of substantial benefit 54%). When the influence of abdominal cramp was added as a covariate in the

analysis, the enhancement was reduced to 0.6% (±1.6%, likelihood of substantial benefit 26%).

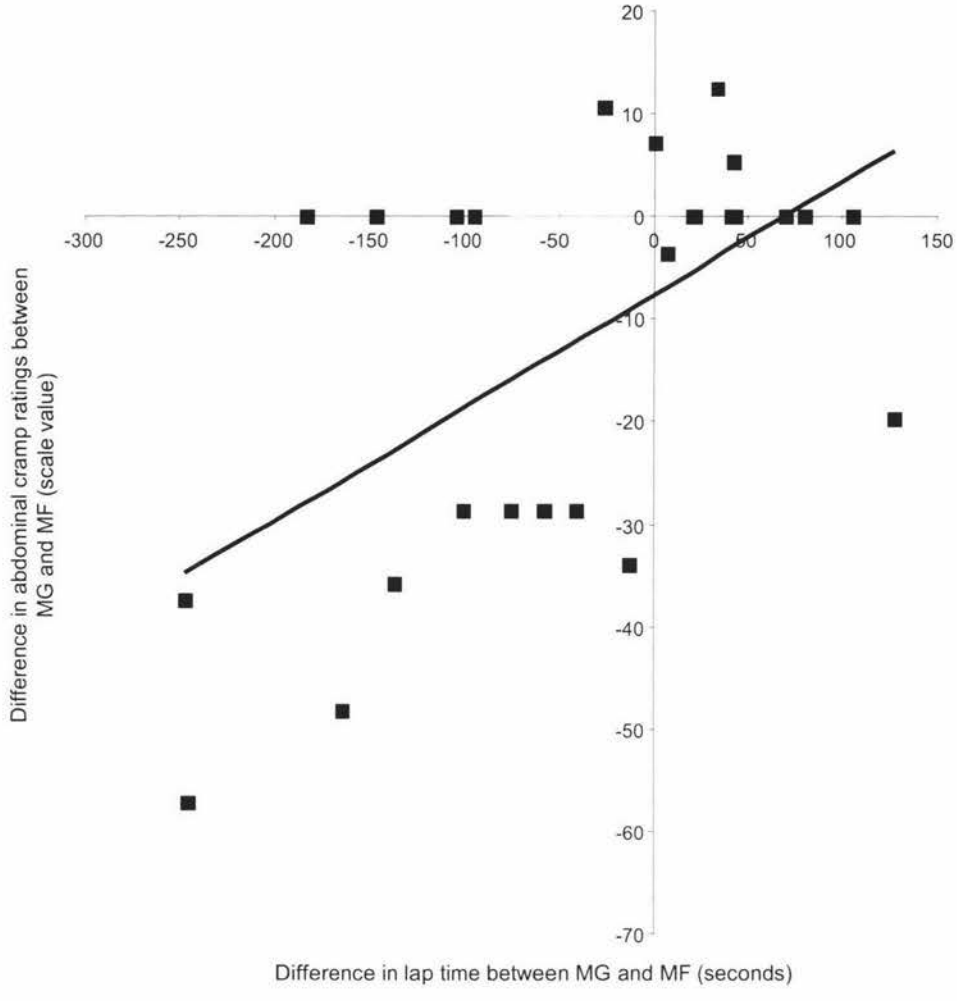

Figure 3. The relationship between ratings of abdominal cramp and changes in lap time

Ratings of Perceived Exertion and Gastro-intestinal Discomfort

Ratings of perceived exertion and gastro-intestinal discomfort are illustrated in Figure 4 and recorded with inferences to the smallest worthwhile effect in Table 4. Although still very low in magnitude, perceptions of abdominal cramps were substantially increased when MG was ingested relative to MF. Cyclist's ratings of abdominal cramps decreased with race order (Appendix 10). There was a

possible increase in feelings of leg strength with MF ingestion. The effect of drink composition on ratings of perceived effort and tiredness was probably trivial, while for nausea and diahorrea the effects were not clear.

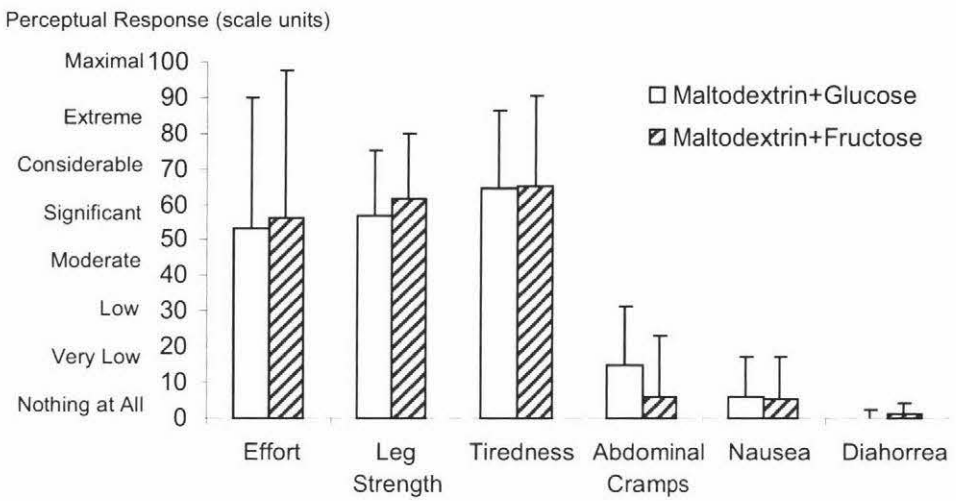

Figure 4: The perceptual response to drink composition

Measurements of Hydration Status

The effects of ingesting MG and MF on changes in measurements of hydration status are shown in Table 5. Ingestion of MF had no clear effect on any of the measurements of hydration status. There was no effect of race on the measurements of hydration status (Appendix 10).

The relationships between urine specific gravity and urine osmolality, urine specific gravity and urine colour and urine osmolality and urine colour are shown in Figures 6, 7 and 8 and were found to have r values (Cohen Effect Size descriptors) of +0.96 (nearly perfect), +0.64 (large) and +0.54 (large) respectively.

Table 4. The effect of ingesting Maltodextrin+Fructose (MF) over Maltodextrin+Glucose (MG) on ratings of perceived exertion and gastro-intestinal discomfort. Markers listed in order of clearest outcome.

	Mean Effect (scale units) of MF-MG ± 90% CL[a]	Chances (%) and Qualitative Inference[b]
Abdominal Cramps	-8.1 ± 6.6	1/10/89 Likely Beneficial
Leg Strength	4.2 ± 5.9	57/41/2 Possibly Beneficial
Effort	0.2 ± 6.7	4/93/3 Probably Trivial
Tiredness	0.8 ± 4.1	7/90/3 Probably Trivial
Nausea	-0.5 ± 3.3	8/74/18 unclear
Diahorrea	0.9 ± 2.2	66/19/15 Unclear

[a] ±90%CL: Add or subtract this number from the mean effect to obtain the 90% confidence limits
[b] Chances that the true effect of the intervention is positive/trivial/negative with qualitative descriptor

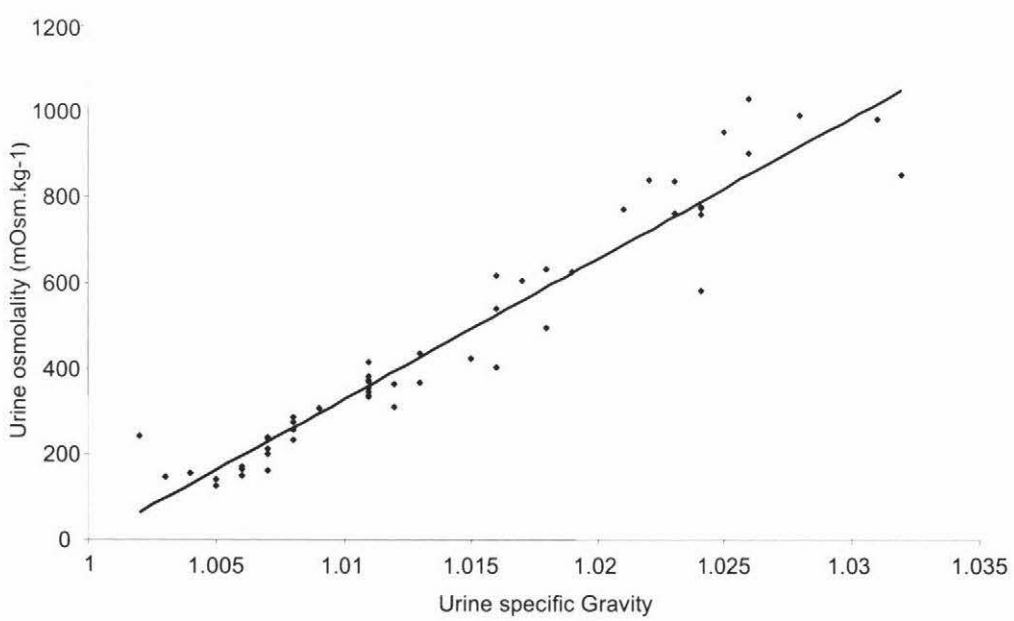

Figure 5. Relationship between urine specific gravity and urine osmolality

Table 5. Measurements of hydration status taken pre- and post-race, the percentage change between post- and pre-race values and the effect of maltodextrin+glucose (MG) and maltodextrin+fructose (MF) ingestion on these changes.

Pre-Race[a]		Post-Race[a]		Relative Change (post-pre)[a]		Effect[b]	Chances[c] (%) and Qualitative Inference
MG	MF	MG	MF	MG	MF		
Body Mass (Kg)							
70.4 ± 9.9	70.3 ± 9.9	69.2 ± 9.6	69.0 ± 9.6	-1.7 ± 0.8	-1.8 ± 0.5	0.1 ± 0.5	0/100/0 Trivial
Urine Specific Gravity (scale value)							
1.015 ± 0.010	1.012 ± 0.007	1.016 ± 0.008	1.014 ± 0.008	0.17 ± 0.52	0.04 ± 0.5	0.1 ± 0.4	50/31/19 Unclear
Urine Osmolality ($mOsm \cdot kg^{-1}$)							
549 ± 370	446 ± 308	511 ± 274	436 ± 229	20 ± 58	25 ± 62	-4 ± 39	26/36/38 Unclear
Urine Colour (scale value)							
2.9 ± 1.4	2.1 ± 1.1	5.8 ± 1.3	5.1 ± 1.7	155 ± 138	207 ± 124	-15 ± 33	11/29/60 Unclear

[a] Data are mean ± SD
[b] ±90%CL: Add or subtract this number from the mean effect to obtain the 90% confidence limits
[c] Chances that the true effect of the intervention is positive/trivial/negative with qualitative descriptor

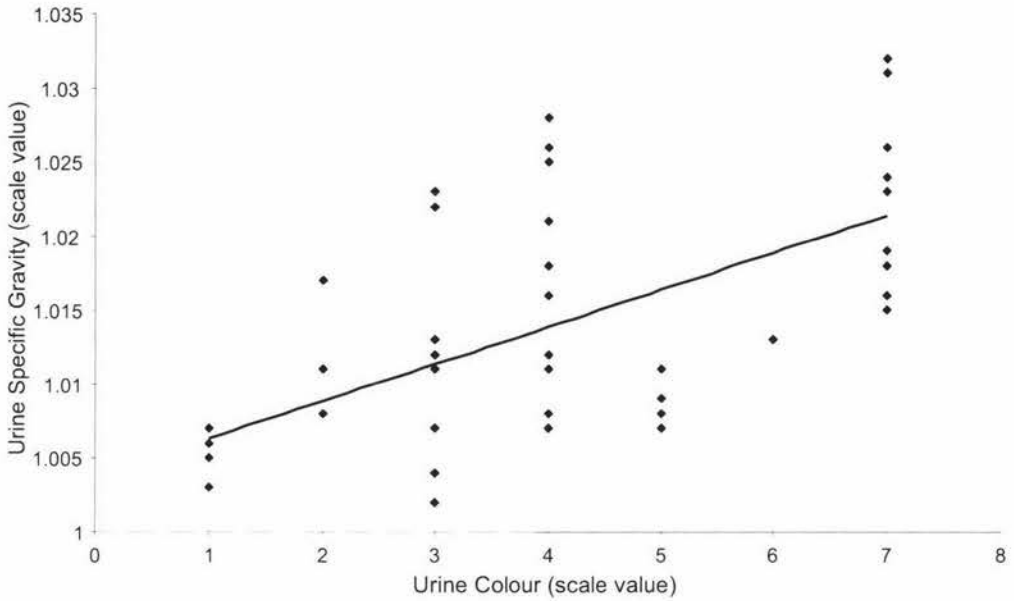

Figure 6. Relationship between urine specific gravity and urine colour

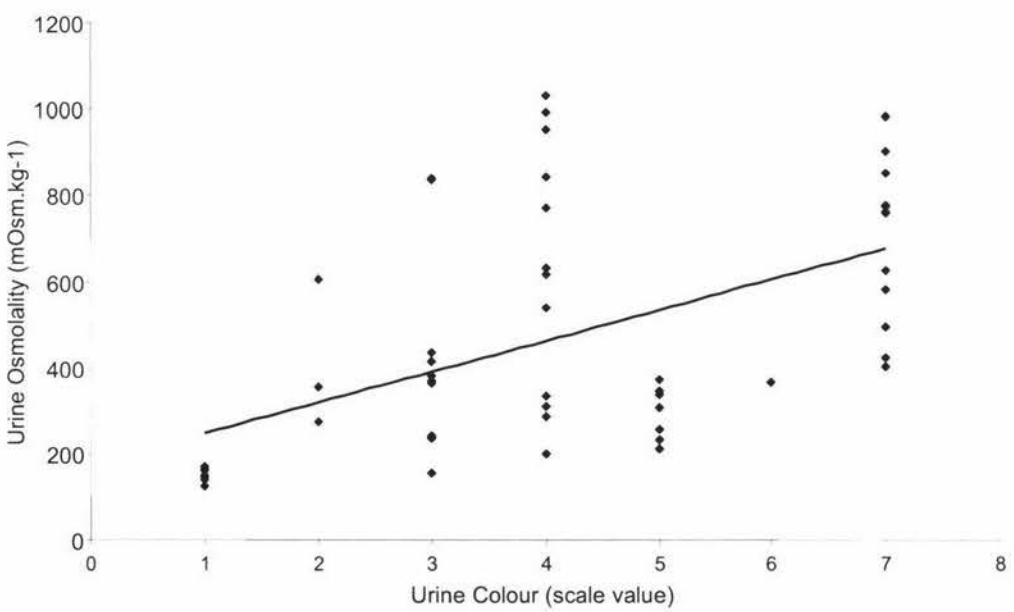

Figure 7. Relationship between urine osmolality and urine colour

Discussion

The main finding of this study is that mean race time decreased by 1.8% in a cross-country mountain bike event following the ingestion of a solution containing maltodextrin+fructose (MF) compared to maltodextrin+glucose (MG). This is the first study, to our knowledge, that has shown improved performance in a field model due to the ingestion of a composite carbohydrate solution. Whilst the effect is statistically described as a possible enhancement when using a coefficient of variation (CV) for race time produced by a similar population sample to determine the smallest worthwhile effect, there is a 72% likelihood of a beneficial effect when using a CV derived from World Cup cross-country mountain bikers. Furthermore there is only a 1% chance of harm which from any athlete's perspective would seem a worthwhile intervention to take in pursuit of improved performance. Only with a sample size of about 90 (Appendix 11), would the confidence interval be narrow enough to establish whether the effect is genuinely trivial or beneficial.

It has been argued that the best method of establishing the smallest worthwhile effect of a performance intervention is to use a value of about half (0.3-0.7) that of the within-subject variance or coefficient of variation for the performance or like performance in question (Hopkins, et al., 1999). We wanted to use a CV calculated from a similar population group to that of our cyclists and derived it from the Port Nicholson and Poneke Cycling Club, 2006 cross-country mountain bike race series. However, it has been suggested that there are two important assumptions that are made when doing this (Paton and Hopkins, 2006), firstly that the athletes compete as individuals and secondly that each athlete attempts to provide their best performance in each race. Whilst both of these assumptions can be confidently made at the elite World Cup level, they provide a possible area of weakness in the use of our PNP Club generated CV. Despite the fact that there are numerous elite riders who compete within the different categories of this club series, the level and consequential importance of the series itself will likely cause some riders to compete in some of the races at below their best. They may do this by using some races as hard training rides in

preparation for more significant events such as the National Series, preparing in a different manner for each race (socially, nutritionally, psychologically and in terms of health, rest and exercise), riding different bikes, incurring mechanical faults or having crashes, all of which would increase variation and reduce the reliability of the calculated CV. Although similar points could be made with the World Cup Series, the consistency and preparation by elite athletes for competition at this level is likely to be much improved, as was also suggested by Paton and Hopkins (2006). In consideration of this, use of the World Cup CV may prove more reliable than that of the PNP club as Hopkins (2000) has stated that "the typical error (also known as within-subject standard deviation) can be estimated from a sample of individuals that is not necessarily representative of a population."

Previous studies which have investigated the effects of carbohydrate on performance have shown performance improvements of between 2-7% (Angus, et al., 2000; Tsintzas, et al., 1993; Tsintzas, et al., 1995b; Williams, et al., 1990). However, to the author's knowledge, only one study has tested the effect of ingested carbohydrate on performance in the field. Tsintzas et al. (1993) observed a 2.3% improvement in time to complete a 30-km road race when participants ingested 1 L of a 5% carbohydrate drink compared to 1 L of non-flavoured tap water. Whilst the 2.3% improvement was gained with ingestion of a total of only 50 g carbohydrate, it was measured against performance without carbohydrate ingestion or blinding, whereas our study shows a 1.8% improvement with ingestion of a carbohydrate drink containing multi-transportable carbohydrates compared to another containing an equal amount of carbohydrate in a placebo-controlled flavoured solution. It is likely that an even greater performance improvement would have been measured against water ingestion alone. Below et al. (1995) observed independent and additive improvements when a water-electrolyte drink and a drink containing carbohydrate and electrolytes were ingested compared with a non-fluid electrolyte-containing placebo during exercise. Performance times in a test designed to simulate a sprint finish at the end of a 40-km time trial were improved by 6% when fluid and carbohydrate were ingested individually and by

12% when the fluid and carbohydrate were taken together. Furthermore, the relatively large performance effect observed by Tsintzas et al. (1993) may have been partly attributable to a placebo effect created by the use of non-flavoured tap water in the control group which has been shown in a lab based setting to be substantial (Clark, et al., 2000). Angus et al. (2000) observed a 6.7% decrease in performance time during a 100-km cycling time trial when a 6% solution was ingested at a rate of 1 $L \cdot h^{-1}$. However, the non-technical nature of a time trial and the controlled conditions of the laboratory setting would be expected to reduce the variance associated with environmental factors and enhance the observed effect.

The proposed mechanism behind the performance benefits of a combined maltodextrin and fructose drink compared to maltodextrin and glucose drink is increased carbohydrate availability and higher exogenous-carbohydrate oxidation rates (Hawley, et al., 1994; Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jeukendrup and Jentjens, 2000; Jeukendrup, et al., 2006; Jeukendrup, et al., 1999a; Wallis, et al., 2005) which spare muscle (Hargreaves, et al., 1984; Yaspelkis, et al., 1993) and/or liver glycogen by maintaining blood-glucose availability (Bosch, et al., 1994; Jeukendrup, et al., 2006; Jeukendrup, et al., 1999b) and possibly allow the re-synthesis of muscle glycogen during periods of low intensity exercise (Kuipers, et al., 1987; Kuipers, et al., 1989; Palmer, et al., 1999). Peak oxidation rates of exogenous carbohydrates are higher when glucose or glucose polymers in combination with fructose are ingested at high levels (Jentjens, et al., 2004c; Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Wallis, et al., 2005) than when either other combined carbohydrate solutions (Jentjens, et al., 2004c; Jentjens, et al., 2004b) or solutions containing only one carbohydrate (Jeukendrup, et al., 1999b; Rehrer, et al., 1992b; Wagenmakers, et al., 1993) are ingested. It has been suggested that this is because glucose and fructose are transported across the small intestine by different mechanisms and that fructose transport is further enhanced by the presence of glucose (Shi, et al., 1997). Several studies have shown that in moderate to heavy continuous cycling exercise (50-80% VO_{2max}) there is no muscle-glycogen sparing but that

fatigue maybe delayed due to maintenance of blood-glucose concentrations (Coggan and Coyle, 1987; Coyle, et al., 1986; Coyle, et al., 1983; Hargreaves and Briggs, 1988; Mitchell, et al., 1989). However, both improvements in performance (Hargreaves, et al., 1984; Yaspelkis, et al., 1993) and muscle-glycogen sparing (Hargreaves, et al., 1984; Palmer, et al., 1999; Yaspelkis, et al., 1993) have been shown in cycling exercise of an intermittent nature. High rates of carbohydrate ingestion have been shown to improve performance by decreasing muscle-glycogen use (Hargreaves, et al., 1984; Yaspelkis, et al., 1993) decreasing hepatic-glucose production (Jeukendrup, et al., 1999a; Jeukendrup, et al., 1999b) which therefore spares liver glycogen (Jentjens, et al., 2004c; Wallis, et al., 2005) and possibly by allowing the re-synthesis of muscle glycogen in endurance-trained athletes (Kuipers, et al., 1987; Kuipers, et al., 1989). It may also be possible that the carbohydrate ingested may have an independent or additional ergogenic effect through a reduction in central fatigue (Dalsgaard, et al., 2002; Davis, et al., 1992; Nybo, et al., 2005; Nybo, et al., 2003; Snow, et al., 2000).

Additionally, abdominal cramps may have contributed to the performance outcome. Abdominal cramp ratings were lower in MF compared to MG (Figure 5). Previous studies (Jentjens, et al., 2004c; Jentjens, et al., 2004a; Jentjens, et al., 2004b; Wallis, et al., 2005) in which high rates of carbohydrates have been ingested in the form of glucose and glucose polymers compared with combined carbohydrate solutions have also recorded incidences of gastro-intestinal discomfort with the former although when experienced, severe nausea was a common complaint. In the current study there was no clear effect from either solution on nausea or diahorrea. It has been suggested (Jentjens, et al., 2004a) that the higher rate of gastro-intestinal discomfort following ingestion of drinks containing large amounts of glucose compared with glucose and fructose combinations may be due to less carbohydrate leaving the gastro-intestinal tract. This would support the contention that exogenous-carbohydrate availability is limited by intestinal absorption (Hawley, et al., 1992a) and that increased exogenous carbohydrate is made available when an equicaloric quantity of carbohydrate is ingested in the form of multi-transportable

carbohydrates because of reduced competition for transporters (Jentjens, et al., 2004a).

We also observed a trend towards greater perceptions of leg strength with ingestion of MF compared to MG although further research with a larger sample size would be required to determine whether this is a conclusive beneficial effect. Previous studies have found no significant relationship between the RPE for legs and ingestion of combined carbohydrate solutions (Jentjens and Jeukendrup, 2005; Jentjens, et al., 2004b). The effect of drink formulation on effort and tiredness was probably trivial in both cases. Likewise, no significant differences in RPE ratings have been reported where the effect of carbohydrate on exercise capacity and performance has been investigated previously (Below, et al., 1995; Tsintzas, et al., 1993; Tsintzas, et al., 1996a; Tsintzas, et al., 1995b; Tsintzas, et al., 1996b; Williams, et al., 1990; Yaspelkis, et al., 1993). This finding should be typical of those situations in which participants are requested to work as hard as possible at a given task such as in racing performance or an exercise capacity test. Regardless of their absolute output due to a given intervention, their relative effort would be expected to remain the same.

The results of this study are relevant to athletes who race at intensities and of a duration in which glycogen stores may become depleted. Cross-country mountain bikers are an example of such athletes because of the high intensity, intermittent nature of their racing. Stapelfeldt et al. (2004) observed that although elite mountain bikers spent 39% of race time below their aerobic threshold, their average heart rate throughout the race was 104% of that at their individual anaerobic threshold and 91% of that at exhaustion. Similar findings were also produced by Impellizzeri et al. (2002). Exercise at these intensities rely on muscle glycogen as the predominant fuel source which when low is associated with exhaustion (Saltin and Karlsson, 1971). As muscle-glycogen stores decrease, more glucose is extracted from the blood (Gollnick, et al., 1981) which leads to increased liver glycogenolysis and gluconeogenesis in

order to increase hepatic-glucose output and maintain blood-glucose concentrations.

Most of the riders involved in this study were highly-trained regional-level athletes and included two recent New Zealand representatives. However, these findings may actually become of greater relevance to elite athletes as it has been suggested (Hopkins, et al., 1999) that there is probably a smaller between-athlete variation at the elite level than that of sub-elite athletes. The elite would therefore benefit from an intervention effect of lesser magnitude. Furthermore, extremely well-trained athletes may oxidise carbohydrates at higher rates than less well trained athletes (Angus, et al., 2002) gaining more benefit from high rates of carbohydrate ingestion through drinks such as this although no studies have as yet researched this idea.

The effect of the composition of the drinks ingested on changes in hydration status measured by urinary indices and change in body mass was unclear. The relative change in body mass over the race period was -1.7% and -1.8% in MG and MF respectively and reflects both fluid loss from respiration and sweating and substrate oxidation. Dehydration as measured by a decrease in body mass has been shown to be harmful to performance (Armstrong, et al., 1985; Walsh, et al., 1994) but a review of the studies measuring hydration status and exercise performance (Shirreffs, 2005) has suggested that when exercise is performed in a temperate environment (20-21°C) which reflects the conditions of this study, that dehydration by 2% of body mass appears to have an insignificant effect on endurance performance. These values indicate that an appropriate quantity of fluid was provided to the riders during the races. This proposition is further supported by the urine measurements which when compared to previously determined recommendations (Armstrong, et al., 1994) indicate that the cyclists were in a euhydrated state both before and after racing.

The final aim of this study was to compare correlations between urinary indices of hydration status with those previously reported by Armstrong et al. (1994). While the correlation coefficient between urine osmolality (U_{osm}) and urine

specific gravity (U_{sg}) in the two studies was similar (+0.97 and +0.96 in Armstrong and the current study respectively), there was a greater difference in the strength of the correlations between urine colour (U_{col}) and U_{sg} (+0.8 and +0.64) and U_{col} and U_{osm} (+0.82 and +0.54). The discrepancies between correlations involving urine colour are likely to reflect the reduced accuracy and subjective nature of the urine colour test but validate the suggestion that urine colour may be used in athletic settings where close estimates of hydration status are acceptable but should not be used in laboratories where greater precision and accuracy are required (Armstrong, et al., 1994).

In conclusion, performance may be improved by the ingestion of a drink formulation containing maltodextrin and fructose compared to maltodextrin and glucose in a cross-country mountain bike event. To confirm the strength of the effect, the use of a larger sample size in a further investigation is recommended. The reduced abdominal cramp ratings in MF compared to MG provide evidence to support the suggestion that intestinal absorption limits exogenous-carbohydrate availability and that increased carbohydrate is made available through the ingestion of multi-transportable carbohydrates. Furthermore, ingestion of a concentrated drink containing large amounts of multi-transportable carbohydrates does not appear to cause dehydration during high intensity exercise of 2-3 hours duration.

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Appendix 1 Controlled Study Methods

Participants

31 mountain bikers involved in regular training participated in this study. Cyclists were 72.9 ± 12.2 kg, training for at least 8 hours per week and racing regularly. Cyclists attended a pre-event meeting seven days prior to the race and were required to attend a pre-race briefing prior to the race itself. After reading the study information sheet, all cyclists completed a General Health Questionnaire (Appendix 2) to assess them for prohibiting medical factors. All cyclists were made aware of the physical and psychological discomfort associated with competition, high intensity exercise and the possibility of gastro-intestinal discomfort from the ingestion of high quantities of carbohydrates and were informed of their right to withdraw from the study at any time. All cyclists signed a written, informed consent form before racing. This study was approved by the Massey University Ethics Committee: Southern A, 06/04.

Preliminary Testing

Seven days prior to the event cyclists were asked to complete a grading event from which results were used to match pairs. The grading event was considered preferential to standard measures of physiological capacity such as maximal power and maximal aerobic capacity due to its ability to account for the high technical skill and psychological components of mountain bike racing in addition to the physiological component. The grading event was one of a series of cross-country mountain bike races run by a Wellington-based mountain bike club. The course terrain was of a similar nature to the proposed event and had a winning time of approximately 1 h 40 min duration ensuring that strength and endurance capabilities provided a significant contribution to the results. Prior to the start of the grading race all cyclists were weighed so that mass could be used to calculate the amount of drinks required for each individual and were familiarized with the scales to be used during the race. The results from the race were used to form two groups of riders with as close as possible average total race time and a negligible effect size statistic. Each of the two groups was then randomly assigned to one of the two drink formulations for the study race.

Study Protocol

The study had a double-blind, controlled design with 2 treatments (Figure A1). Two groups were randomly assigned one treatment drink for consumption during a cross-country mountain bike event. For 2 days before the event cyclists standardised training and diet. Cyclists were required to do either very light training or no training at all to ensure that all athletes started the race in a similar physical state and emphasis was placed on the need to refrain from hard riding on these days. From the morning of the second day before the event food intake was standardized by ingestion of a diet containing a minimum of ~500g carbohydrate. Cyclists were required to abstain from alcohol consumption in the 24 hours prior to the race. On race day a prescribed breakfast, which was considered to provide appropriate pre-race nutrition, was consumed 2hrs prior to the race start. It consisted of two muesli bars, a banana and water *ad libitum* and provided approximately 1800kj; 80g carbohydrate, 7g proteins and 11g fat. Particular emphasis was placed on the importance of refraining from consumption of any stimulants such as caffeine-containing drinks.

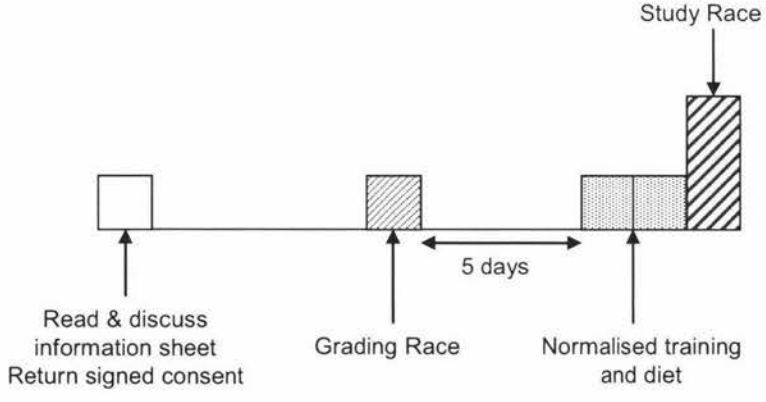


Figure A1. Study design – controlled model

Cyclists arrived at the race venue at approximately 0830 hours. On arrival a urine sample was provided and body mass was recorded in race clothing, following which cyclists were free to prepare their equipment and warm-up as necessary. All cyclists attended a pre-race briefing at 0930 hours which included safety information, a reminder of race etiquette, re-emphasis of

experimental protocol to be used and an outline of the course. At 0945 hours cyclists collected their camelbak water carriers which contained the required drink for laps 1 and 2 and at the same time consumed an initial drink bolus of 200ml. At 1000 hours cyclists assembled on a start line for a mass start. A feeding area was stationed at the start line. At the end of laps two and four, cyclists entered the feeding area and had their camelbak refilled whilst completing a scale to mark the level of perceived fatigue, exercise intensity and gastrointestinal discomfort experienced at that time. After crossing the finish line, cyclists again completed the scale provided a post-race urine sample and had body mass measured. Following completion of the experimental protocol, cyclists were free to eat and drink normally and to leave.

Exercise Protocol

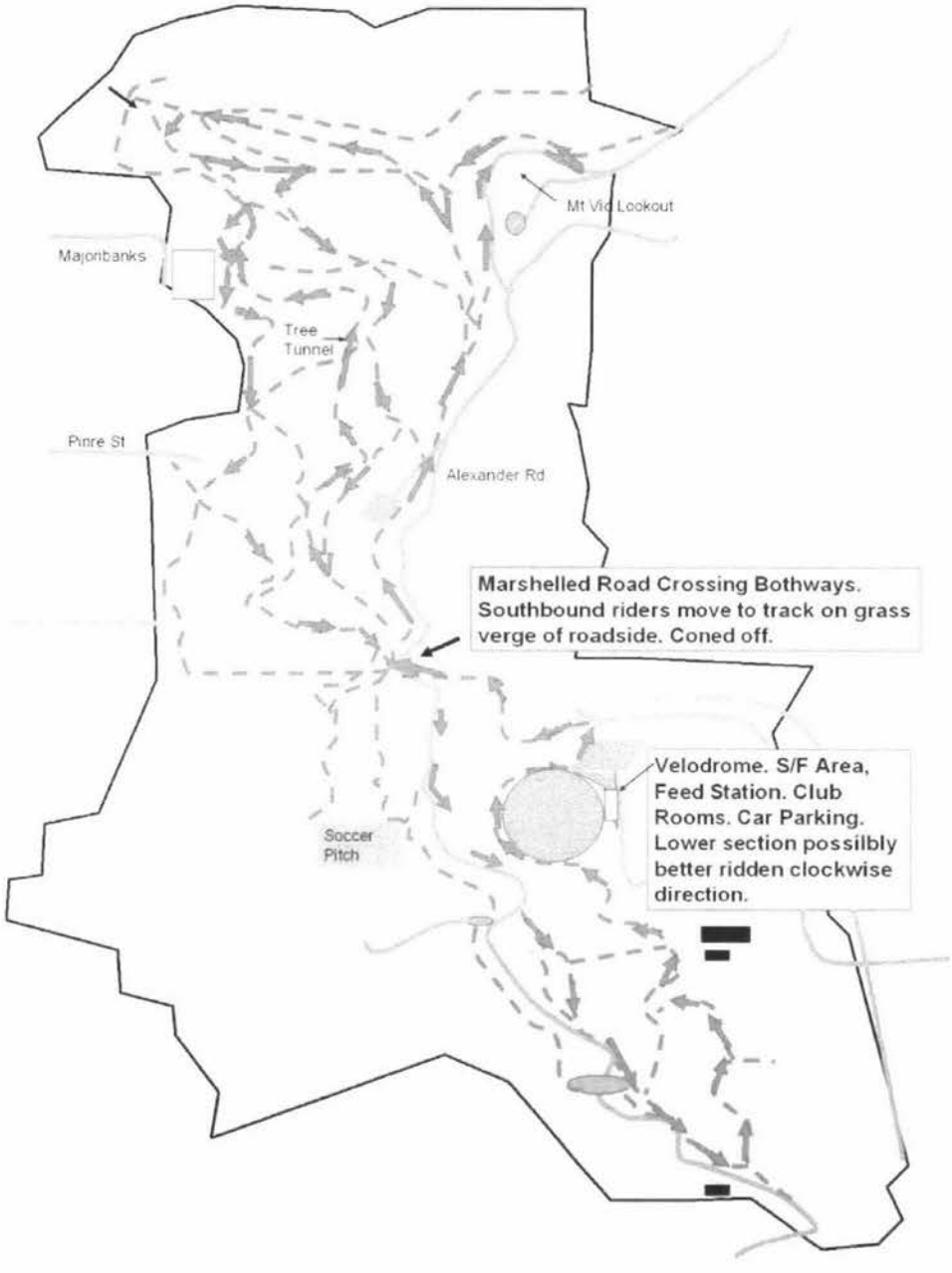
The race was an endurance event designed to be completed in approximately two and a half hours. The course was five laps and consisted of firebreaks, medium-grade single track, 4WD track, shingle and some short sections of seal. The terrain included many steep uphill sections of between five seconds and 10 minutes duration to emphasise glycogen depletion. The event was run in a similar fashion local club events, with directional signage, public notification, marshals, and road signs where necessary. Normal MTB race rules and etiquette applied and first aid stations were provided.

Experimental Drinks

The two drinks were a maltodextrin and fructose (F) intervention and a maltodextrin and glucose (G) control. The analytical composition of the drinks is shown in Table 3 of the Methods section (p60). Including the initial bolus, the quantity of drink provided to each cyclist was 800 $ml \cdot h^{-1}$ per 75kg bodyweight. This was calculated to be 400ml per lap per 75kg based on the expectation that each lap would take approximately 30 minutes to complete. Pilot tests were completed on the drinks prior to use in the event. Three competitive cyclists, not involved in the study, sampled both drinks at the required rate during a three hour training ride and provided comments on the drinks' palatability and any gastro-intestinal discomfort.

Appendix 2 Course Map

Proposed Course for Massey University Study 6 and 13 Dec 2006 as red arrows. Should be no off-bike climbing sections. Measured ride time (D Rowlands on Rigid) 27.5 min (5 x 27.5 min = 2:15min). Need to test ride to determine. Want 23-24 min lap time for first 2-3 laps for faster rider to make wining time 2h 30min.



Appendix 3 Information Sheet

The Institute of Food, Nutrition and Human Health

The Institute of Food, Nutrition and Human Health is part of Massey University. It includes a large team of lecturers and scientists that are interested in human nutrition, sport and exercise science, physiology, and health.

The researchers in this project are Marilla Swift, who will be working on the project for her post-graduate research thesis and Dr David Rowlands, who has a PhD in Exercise Physiology and Sport Nutrition and is a lecturer in sport and exercise science at the Wellington campus. The researchers are interested in the role of diet and training interventions on physiological and performance responses in athletes.

Why Are We Doing This Study?

Muscle glycogen (carbohydrate stored in the muscles) and blood glucose are the most important fuels for the exercising muscles. Fatigue in prolonged endurance events is often associated with muscle-glycogen depletion and reduced blood-glucose concentrations. It is generally believed that the maximal rate at which ingested glucose-based carbohydrate can be converted to energy for use by the muscles is approximately 1.0g per minute during prolonged exercise. Recent studies indicate that ingestion of an alternative type of sugar, fructose, in combination with glucose results in about 1.5-fold higher rates of absorption and oxidation. Glucose and fructose are absorbed by the intestines via different transport mechanisms.

Most studies into the performance effects of carbohydrate supplementation during prolonged endurance exercise have been completed in the laboratory using exercise of about two hours duration when the athlete has fasted overnight, giving them initial depleted glycogen stores. These conditions do not accurately represent the demands and preparation surrounding competition. Furthermore, no research has looked at the affect of combined fructose and glucose (*composite carbohydrate*) on performance. Mountain biking uses large muscle groups for extended periods of time at intensities that are likely to deplete muscle-glycogen stores interspersed with short periods of recovery. Ingesting carbohydrate supplements can slow the rate at which these glycogen stores are depleted and helps maintain the body's ability to perform once stores are depleted. The extended length of an endurance MTB event (e.g. Colville; Karapoti) provides a stimulus that should enhance any potential ergogenic effects of the supplement.

Participant Recruitment

The researchers would like to recruit a large sample of mountain bikers (30+) to compete in two endurance MTB events on Wed 6th and 13th Dec 2006. To participate in the study you should:

- Be a male or female mountain biker in regular training aged 18-55 years. Mountain bikers between the ages of 16 and 18 years may enter with written parental/guardian consent. Regular training, for the purposes of the study, is defined as about 8 or more hours per week for the last 20 weeks or greater. You should also have been racing regularly or been exposed to regular high-intensity

training. Ideally, you should have been training consistently and seriously at the sport for at least two years.

- Must have 1L minimum capacity hydration pack that is not leaking for use during the race – drink bottles may not be used because of risks of falling out. If you do not have a hydration pack, see if you can borrow or purchase one, or discuss with the researchers.
- Please read carefully through this information sheet before deciding whether you would like to participate in this study. When you have done so please complete the health screening questionnaire and entry form (see below), sign the Massey University research ethics consent form (see below) and return to Marilla Swift. If you have preliminary questions, contact Marilla Swift by e-mail or phone.

What is Involved

The objective of the research is to determine if adding fructose to maltodextrin (a glucose-based carbohydrate) in a sports drink will enhance performance, relative to a glucose-maltodextrin control. Both drinks will contain the same amount of calories and salt.

The study involves completion of two mountain bike races, a week apart. All participants will be required to compete in both races. Participants will be split into two groups, with each group consuming one of the two drinks during each of the events. The events will be run at Mt. Victoria from the velodrome on Wed 6th and 13th December 2006. See entry form for more details. The event is an endurance event so will take most about 2.5-3.0 h on the Mt. Victoria tracks. The event will be run in a similar fashion to PNP club events, with directional signage, public notification, marshals, and road signs. Normal MTB race rules and etiquette will apply. Qualified first aid provision will be on course. The study design is illustrated in Figure 1.

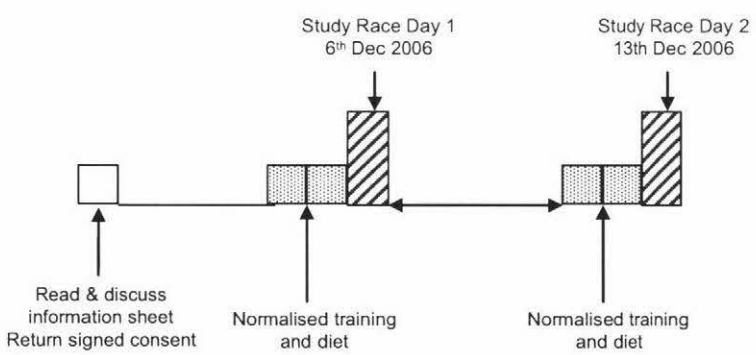


Figure 1. Study design

Details:

- All participants will be required to complete the health questionnaire, race entry form and consent form included in this information sheet. Please feel free to contact Marilla Swift if you have any questions regarding the study. Prior to the events, there will be opportunities to meet the researchers either at the Massey University Sports Science Lab or at the Makara Peak PNP Club Race. At this

time we will provide you with an opportunity to become familiarized with the scales to be used during the race and to clarify any other questions that you might have. We will also record your body weight for the purposes of drink quantity calculation in the proceeding week.

- **Normalised Training.** For 4 days before the event we would like you to standardize your training program. On the Saturday and Sunday before you may exercise as you like but you must complete exactly the same exercise (length and intensity) on both weeks. So if you do a race on the weekend of 3rd Dec you must mimic it during the following weekend. Best not to do a race on the weekend of the 10th Dec, as it will be difficult to predict the nature of the race the weekend prior. On the Mondays and Tuesdays any training should be either *very light training* or no training at all to ensure that all athletes start the race in a similar physical state. It is very important that you *do not do a hard ride the day before the event* or you will compromise the research outcomes.
- **Normalised Diet.** From the morning of the day before the event we would like you to standardize your diet by ingesting a minimum quantity of carbohydrate-rich food. Instructions will be provided. We would also like you to refrain from alcohol consumption in the 24hrs prior to the race.
- Race Days - Registration 4:15-5.15pm. Briefing 5:25pm. Race start 5:45pm. Please don't be late, sunset is 8:45pm!
- **Standardised diet and no caffeine.** We would like you eat a prescribed standardised breakfast and lunch and an afternoon snack, which we will provide and then refrain from eating after 4 PM. Because of the probable and variable effects on your performance, you *may NOT take any stimulants such as caffeine-containing drinks like coffee, coke, or tea on the day or during the event.* So no morning coffee or tea, sorry. It's ok, you will survive.
- **Urine sample.** At or around registration, we would like you to provide a urine sample in a container which we will provide and we will measure your body mass on weighing scales. You will provide a second sample at the end of the event. By measuring body weight and the density of your urine we gain information regarding hydration status.
- **Ingest only water from 4 PM**. *From 4 PM before race start you will ingest only water* as you feel. Please ensure that you are well hydrated throughout the day by drinking about 6-10 full glasses of fluid during the day and ensure urine flow is reasonable and not dark coloured.
- **Experiment drinks**. During the race, the drinks will be lemon/lime flavoured and taste similar to commercial products, such as, Replace. You will not be made aware of which of the solutions you are consuming. Hand in your hydration pack at registration and we will fill it with a specific quantity of drink based on your body weight. This will be the quantity of drink you will be asked to consume completely on a per lap/section basis during the race. You will collect your filled hydration pack at the end of race briefing.
- **Scales.** At the end of each lap/section at the feed station and at the finish you will mark lines on a linear scale providing a rating of how you are feeling in relation to muscular fatigue, general fatigue, the intensity at which you are working and any gastric discomfort. Researchers will present you with a laminated card showing scales relating to these conditions and ask you to mark each scale according to the comfort levels you are experiencing at that time. You will have an opportunity to become familiarised with the scales prior to race day and at registration, any queries that you may have can be clarified at this point.

Scale marking will take only 10 s. Laps will be timed upon entry to the feed zone.

- Following completion of each event, a free BBQ will be provided and following the second event there will be a short prize giving where results and spot prizes will be awarded. Performance Prizes will be awarded on a sealed handicap basis. Some spot prizes will also be awarded. Fingers crossed for good weather.

Other Information:

- The drinks will be formulated to provide what we think from lab studies is the optimal amount of carbohydrate, salts, and fluid to maximise carbohydrate delivery and therefore enhance performance.
- *The total time commitment for the study* from you is approximately 9 h: 4 hours for each event which includes; 1 hour before the Study Event, the length of time you take to complete the race and the time to provide a post-race urine sample and mass measurement (approx. 15 minutes) and a short meeting with researchers prior to the event.
- If for whatever reason, you need to discuss these issues sooner, please feel free to contact Marilla Swift.

Are any of the Procedures Harmful or Painful?

There is often some physical and psychological discomfort associated with competition.

Recent evidence has indicated that even among healthy populations of athletes who exercise strenuously and regularly, there is some risk of sudden death due to heart failure. Though rare, such cases can occur in people who may have an undiagnosed condition. If you have any reason to suspect that you may have a cardiovascular problem, we suggest that you see your physician and get an ECG before you agree to participate.

In previous studies involving the ingestion of high quantities of carbohydrates, a small number of participants have experienced gastro-intestinal discomfort such as abdominal cramps, diarrhea or vomiting. The occurrence and intensity of these complaints varies between individuals, is very rare, and does not usually prevent them from completing the prescribed exercise.

If you have any additional medical concerns associated with this project, please contact your GP, or discuss with the researcher.

What if I Suffer a Personal Injury?

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Injury Prevention, Rehabilitation and Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury.

If your ACC claim is not accepted you should immediately contact the researcher. The researcher will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim.

Benefits of Participation

Through participation in this study you will receive the following benefits:

- Knowledge of the results "Are sports drinks containing both maltodextrin and fructose better than maltodextrin and glucose only for endurance mountain bike performance?"
- Free entry into two MTB events
- Prizes as outlined above
- Massey University Drink Bottles

Participant's Rights

At any time, you will have the right to:

- decline to participate;
- decline to answer any particular question;
- withdraw from the study;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded

If you are Interested in Taking Part

CONTACT:

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This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 06/04. If you have any concerns about the conduct of this research, please contact Dr John O'Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8635, email *humanethicsouth@massey.ac.nz*.

Appendix 4 General Health Questionnairee

Massey University
Sport and Exercise Science

General Health Questionnaire

Name: .....
Address: .....
.....
.....
Phone: .....

Name of the investigator responsible for the study:

.....

Please answer the following questions. This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate and to provide the researchers with descriptive information about the participant. If you have any doubts or difficulty with the questions, please ask the investigator for guidance. These questions are to determine whether the proposed exercise is appropriate for you. Your answers will be kept strictly confidential.

1.	What gender are you?	Male	Female
2.	What is your exact date of birth? Day..... Month.....Year..19..... So your age is..... Years		
3.	Are you currently taking any medication?	YES	NO
4.	Has your GP ever advised you not to take vigorous exercise?	YES	NO
5.	Has your GP ever said you have "heart trouble"?	YES	NO
6.	Has your GP ever said you have high blood pressure?	YES	NO
7.	Have you ever taken medication for blood pressure or your heart?	YES	NO

8.	Do you feel pain in your chest when you undertake physical activity?	YES	NO
9.	In the last month have you had pains in your chest when not doing any physical activity?	YES	NO
10.	Has your GP (or anyone else) said that you have raised blood cholesterol?	YES	NO
11.	Have you had a cold or feverish illness in the last month?	YES	NO
12.	Do you ever lose balance because of dizziness, or do you ever lose consciousness?	YES	NO
13.	a) Do you suffer from back pain b) If yes, does it ever prevent you from exercising?	YES YES	NO NO
14.	Do you have moderate-severe liver or kidney disease?	YES	NO
15.	Do you suffer from asthma? If yes, do you control it with medication?	YES YES	NO NO
16.	Do you have any joint or bone problems which may be made worse by exercise?	YES	NO
17.	a) Has your doctor ever said you have diabetes? b) Do you think you have diabetes?	YES YES	NO NO
18.	Are you accustomed to vigorous exercise (~8h / week)?	YES	NO

I have completed the questionnaire to the best of my knowledge and any questions I had have been answered to my full satisfaction.

Signed: ..... **Date:** .....

Source of Original: School of Sport and Exercise Sciences, University of Birmingham

Appendix 5 Participant Consent Form

Massey University

Sport and Exercise Sciences

Institute of Food, Nutrition and Human Health

College of Sciences

Are sports drinks containing both maltodextrin and fructose better than maltodextrin and glucose only for endurance mountain bike performance?

PARTICIPANT CONSENT FORM

This consent from will be held for a period of five (5) years

I have read the Information Sheet and have had the details of the study explained to me. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: ..... **Date:** .....

Full Name - printed .....

Parent or legal guardian (if participant is between 16-18 years)

Signature: ..... **Date:** .....

Full Name - printed .....

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 06/04. If you have any concerns about the conduct of this research, please contact Dr John O'Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8635, email *humanethicsoutha@massey.ac.nz.*

Appendix 6 Training Diary

Training Diary

Separate training diaries were completed for the days leading up to each race. This is the training diary for race 1.

SATURDAY 2ND DECEMBER					
Session #	Exercise Type	Duration h:min	Intensity		
			Low/Time	Medium/Time	High/Time
SUNDAY 3RD DECEMBER					
Session #	Exercise Type	Duration h:min	Intensity		
			Low/Time	Medium/Time	High/Time
MONDAY 4TH DECEMBER					
Session #	Exercise Type	Duration h:min	Intensity		
			Low/Time	Medium/Time	High/Time
TUESDAY 5TH DEC – **Please remember very light or no training today**					
Session #	Exercise Type	Duration h:min	Intensity		
			Low/Time	Medium/Time	High/Time
WEDNESDAY 6TH DEC – **Please do no exercise other than race today**					
Session #	Exercise Type	Duration h:min	Intensity		
			Low/Time	Medium/Time	High/Time
1	MTB Study Race #1				

Appendix 7 48 Hour Diet Record

48 Hour Diet Record

Separate diet records were completed prior to each race and are exemplified below.

TUESDAY – 5th				
TIME	**BRAND**	**FOOD/DRINK ITEM**	**METHOD OF PREPARATION**	**QUANTITY**

WEDNESDAY – 6th – NO CAFFEINE CONSUMPTION				
TIME	**BRAND**	**FOOD/DRINK ITEM**	**METHOD OF PREPARATION**	**QUANTITY**
Please note: No alcohol consumption 24hrs before racing				

Appendix 8 Instructions for Prescribed Meals

Instructions for Prescribed Meals

Instructions and details for the meal options given to cyclists for their pre-race meals are outlined below. Several versions of this information we created differing only in the quantities of food to be taken. These quantities varied according to body mass to ensure that the quantity of food to be eaten was related to need. The different body mass categories were 50-60kg, 60-70kg, 70-80kg and 80-90kg.

Tuesday night and Wednesday Meal Options – 70-80kg

We would like you to eat the same type and amount of food in your meals and snacks on Tuesday night and Wednesday. This is to standardise the amount of energy stored in the muscle before the race between weeks. We would like you to eat food from the following options so that you eat a minimum of 6.5g carbohydrate per kg bodyweight. This will be achieved if you follow the guidelines below.

We would like you to eat the whole content of any choice box. You must not mix and match from options for any one meal, because each option has a fixed amount of carbohydrate calories. You may, however, eat different options for each meal; for example, Breakfast option 2, Lunch option 2, and Dinner option 1. Weigh or measure the quantities where possible, or record what you have in units you can repeat next week.

This is a minimum food requirement, you may eat more if you wish if hungry – just remember to record it so that you can have it again next week.

Most riders eat similar food on a daily basis, so this standardisation of diet should not be too difficult.

Tuesday night Dinner

Option 1		Option 2		Option 3	
300g	baked potato	135g	Pasta	380g	Boiled Potato (mashed)
410g	baked beans	OR	Mince & Dolmio sauce		Steak
	cheese		Vege's & Dolmio	150g	Sweetcorn
	Salad		Cheese		
			Salad		
205g	fruit salad in light syrup	410g	fruit salad in light syrup	410g	fruit salad in light syrup

Wednesday Breakfast and Lunch

PLEASE REMEMBER NO CAFFEINATED DRINKS

Breakfast option 1		Breakfast option 2		Breakfast option 3	
4	weetbix	75g	Muesli	4	slices of toast
400ml	Milk & dsp sugar	400ml	Milk & dsp sugar	20g	honey/jam
OR	Pottle of yoghurt	OR	OR Pottle of yoghurt		
300ml	Apple/orange juice	300ml	Apple/orange juice	300ml	Apple/orange juice

PLEASE NOTE THAT LUNCH SHOULD BE EATEN BETWEEN 1200-1400h

Lunch Option 1		Lunch Option 2		Lunch Option3	
300g	tomato soup	1	pkt trident noodles	2	Filled sandwich (4 slices of bread) OR
3	slices bread w spread	1	slice of bread w spread	2	Filled Rolls (normal size)
1	apple/banana	1	apple/banana	1	apple/banana
2	Uncle Toby's Chewy Bar	2	Uncle Toby's Chewy Bar	2	Uncle Toby's Chewy Bar
300ml	Apple/orange juice	300ml	Apple/orange juice	300ml	Apple/orange juice

We will provide you with a snack to eat before 4pm on race day. Please eat nothing after 4pm on race day and drink only water.

If you have any concerns, please call Marilla Swift on 021 062 7802

Appendix 9 Borg Scales

Original and Modified Borg CR10 Perception Scales

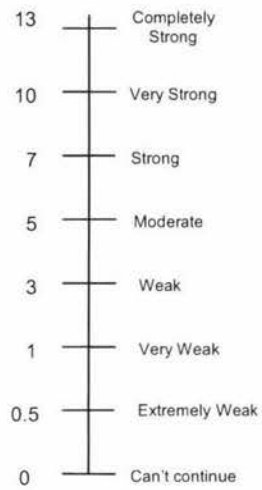
Original CR10 Scale

0	Nothing at all
0.3	
0.5	Extremely weak
0.7	
1	Very weak
0.5	
2	Weak
2.5	
3	Moderate
4	
5	Strong
6	
7	Very strong
8	
9	
10	Extremely strong
11	
•	Absolute Maximum

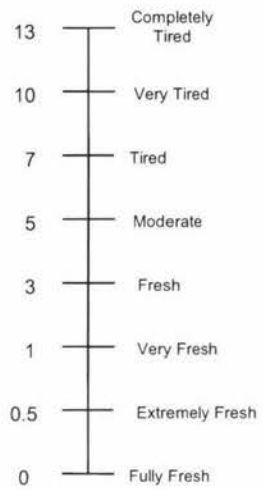
The Borg CR10 Scale (1970, 1985, 1988)

Modified CR10 Scales (this study)

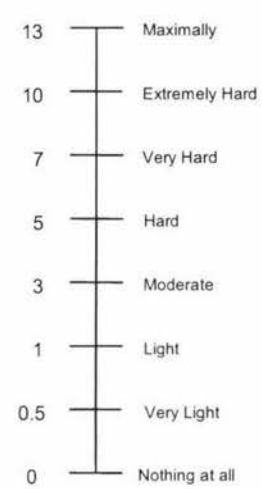
Leg Muscle Strength



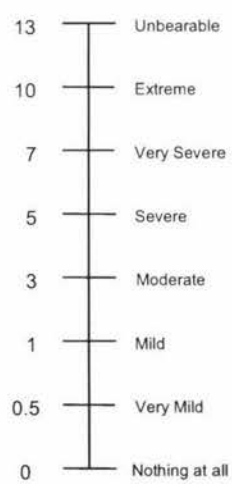
Tiredness?



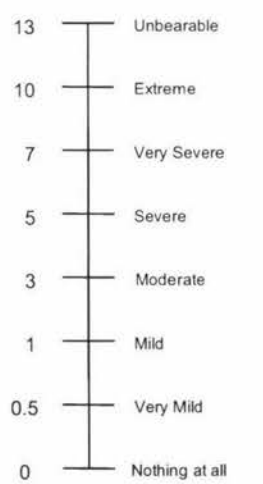
Effort?



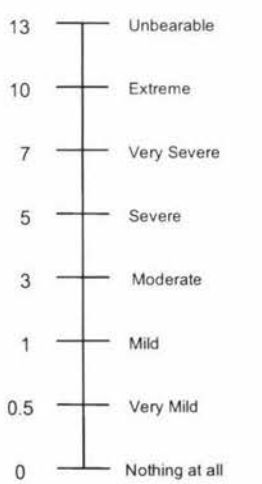
Abdominal cramps?



Nausea?



Diahorrea?



Appendix 10 Effect of race order on ratings of perceived exertion and gastro-intestinal discomfort and measurements of hydration status

Table A1. The mean effect of race order on ratings of perceived exertion and gastro-intestinal discomfort

	Mean Effect (scale units) for Race 2-1 ± 90% CL[a]	Chances (%) and Qualitative Inference[b]
Abdominal Cramps	-2.5 ± 6.6	7/52/41 Unclear
Diahorrea	0.9 ± 2.2	15/19/66 Unclear
Leg Strength	-2.3 ± 5.9	5/60/35 Unclear
Tiredness	0.4 ± 4.1	4/94/2 Trivial
Effort	-0.6 ± 6.7	4/90/6 Trivial
Nausea	-2.6 ± 3.3	1/43/56 Increase possible

[a] ±90%CL: Add or subtract this number from the mean effect to obtain the 90% confidence limits
[b] Chances that the true effect of the intervention is positive/trivial/negative with qualitative descriptor

Table A2. The mean effect of race order on measurements of hydration status

	Mean Effect (%) for Race 2-1 ± 90% CL[a]	Chances (%) and Qualitative Inference[b]
Body Mass	-0.0 ± 0.6	Unclear
Urine Osmolality	-3.8 ± 20.6	15/52/33 Unclear
Urine Specific Gravity	0.0 ± 0.3	Unclear
Urine Colour	0.4 ± 18.5	21/60/19 Unclear

[a] ±90%CL: Add or subtract this number from the mean effect to obtain the 90% confidence limits
[b] Chances that the true effect of the intervention is positive/trivial/negative with qualitative descriptor

Appendix 11 Sample Size Calculations

Calculation to determine the sample size required to narrow 90% confidence interval sufficiently to produce a certain beneficial effect.

Equation 1:

± 90% Confidence Interval = (1.68 x Standard Error of the Effect (SE))

where 1.68 is the z-score for the 90% confidence interval

Equation 2:

$$SE = \left\{ \frac{SD}{\sqrt{n}} \right\}$$

where n = sample size

Equation 3 can be generated by substituting equation 2 into equation 1:

$$90\% \text{ confidence interval} = 1.68 \times \left\{ \frac{SD}{\sqrt{n}} \right\}$$

With an effect of 1.8%, where the smallest worthwhile effect is considered 1.2% (Paton and Hopkins, 2006) the confidence interval required is ±0.6.

In this study the SD of the effect can be calculated from equation 3, where the 90% CI = 1.8 (see results) and n = 10 (see methods)

$$1.8 = 1.68 \times \left\{ \frac{SD}{\sqrt{10}} \right\}$$

$$\frac{1.8}{1.68} = \frac{SD}{3.16}$$

$$1.07 = \frac{SD}{3.16}$$

$$3.39 = SD$$

The SD value can now be used to work out the required sample size when the CI is 0.6.

$$0.6 = 1.68 \times \left\{ \frac{3.39}{\sqrt{n}} \right\}$$

$$\frac{0.6}{1.68} = \frac{3.39}{\sqrt{n}}$$

$$0.36 = \frac{3.39}{\sqrt{n}}$$

$$0.36 \times \sqrt{n} = 3.39$$

$$\sqrt{n} = 9.49$$

$$n = 9.49^2$$

$$n = 90$$