PRE-EXERCISE CARBOHYDRATE INGESTION:

EFFECT OF THE GLYCAEMIC INDEX ON METABOLISM AND ENDURANCE PERFORMANCE

MATTHEW JAMES SPARKS

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SUMMARY

Carbohydrates are a major substrate contributing to energy transduction during medium to high intensity exercise, and the body's levels of this substrate can be manipulated by dietary and exercise behaviours. Nutritional strategies employed before and during exercise affect endurance exercise performance by altering the metabolism of carbohydrate within the body. Carbohydrate feeding during endurance exercise has repeatedly been demonstrated to be beneficial to the athlete. Studies investigating pre-exercise carbohydrate feeding, particularly in the hour before exercise, have produced conflicting results and justify further investigation. The study reported in this dissertation aimed to further investigate the role of the pre-exercise meal, in particular, examine the effect of differing glycaemic indices of carbohydrate foods on metabolism and exercise performance.

A total of eight, endurance trained subjects participated in this study which involved the ingestion of carbohydrate food with differing glycaemic indices 45 min before cycling at a submaximal workload corresponding to 70% VO_{2max} for 50 min, followed by a self-paced 15 min performance ride. In all trials blood samples were taken from a forearm vein and analysed for metabolites and hormones.

The results from this study demonstrate that the pre- exercise ingestion of carbohydrate foods with different glycaemic indices alter metabolism during rest and subsequent submaximal exercise. The data from this study demonstrated that pre-exercise ingestion of a high glycaemic index (HGI) food resulted in a hyperglycaemic response followed by an insulin-mediated hypoglycaemia at the onset of exercise. In addition, the elevated insulin during the HGI trial resulted in an attenuation in circulating FFA and a higher rate of carbohydrate oxidation compared with the ingestion of a low glycaemic index food (LGI) or placebo (CON). Despite the changes in metabolism associated with pre-exercise CHO feeding, exercise performance following 50 min of submaximal exercise was not affected.

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This dissertation summarises original, previously unpublished work conducted in the Human Performance Laboratory, Department of Physical Education and Recreation at Victoria University of Technology. With the exception of isotope analyses, which were conducted at the University of Melbourne, all analyses were conducted in the Department of Chemistry and Biology, Victoria University of Technology. With the exception of data collection which required collaboration and invasive procedures which were conducted by qualified medical personnel, this dissertation is the result of work performed solely by the author.

Matthew. J. Sparks

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CHAPTER 1

INTRODUCTION

Carbohydrate is a major substrate utilized during prolonged exercise and fatigue often coincides with muscle glycogen depletion and/or hypoglycaemia during exercise of this nature (Bergstrom and Hultman, 1967; Hermansen et al., 1967; Vollestad et al., 1984). Accordingly, increasing body CHO stores by dietary manipulation may result in improved exercise performance (Christensen and Hansen, 1939; Gordon et al., 1925). It is well established that carbohydrate feeding during prolonged exercise enhances endurance performance through the maintenance of blood glucose concentrations at a time when the body's own glycogen reserves may be compromised (Coggan and Coyle, 1989; Coyle et al., 1986). In contrast, the value of pre-exercise carbohydrate ingestion is not clear since this practice either increases (Gleeson et al., 1986; Thomas et al., 1991), decreases (Foster et al., 1979) or does not alter (Chryssanthopoulos et al., 1994; Decombaz et al., 1985) endurance performance. The conflict in the literature may be related to different methodologies which vary in type, timing and quantity of CHO ingested. These studies have yielded equivocal results and, therefore, warrant further investigation into this nutritional practice.

The majority of studies investigating pre-exercise carbohydrate feeding involve subjects ingesting nutrients, such as glucose and fructose, in contrast to the ingestion of "real" foods. These nutrients are absorbed at different rates and can affect circulating levels of hormones and other blood-borne substrates. Pre-exercise glucose (high glycaemic index) ingestion has been observed to result in a state of hyperglycaemia followed by a large rise in plasma insulin concentration (Costill et al., 1977; Guezennec et al., 1989; Koivisto et al., 1985). At the onset of exercise, a state of hyperinsulinaemia results in rebound hypoglycaemia (Costill et al., 1977; Decombaz et al., 1985; Koivisto et al., 1985), a reduction in lipolysis (Gleeson et al., 1986; Koivisto et al., 1985), increased carbohydrate oxidation (Costill et al., 1977) and a decrease in exercise performance (Foster et al., 1979). In contrast, the pre-exercise ingestion of fructose (low glycaemic index) attenuates these disturbances in metabolism during exercise (Decombaz et al., 1985; Guezennec et al., 1989).

Carbohydrate foods are also absorbed and oxidised at different rates (Horowitz and Coyle, 1993) and thus, it is surprising that very little research has examined the influence of pre-exercise ingestion of "real" foods on metabolism during exercise and their effect on exercise performance. Recently, Thomas et al. (1991) reported that pre-exercise ingestion of a low glycaemic index (LGI) food increased endurance performance compared with a high glycaemic index food (HGI). They suggested that the LGI food may confer an advantage over a HGI food by maintaining blood glucose

concentrations at higher levels during prolonged exercise. In contrast to the exercise protocol employed by Thomas et al. (1991), many athletic events involve exercise of a duration of approximately 60 min with a performance criteria as a marker of success. Thus, a study examining the adaptive response of the pre-exercise ingestion of "real" foods on a form of exercise that is of a duration and intensity common to a competitive situation is warranted.

The purpose of the study reported in this dissertation, therefore, was to compare the effects of pre-exercise ingestion of foods with high and low glycaemic indices on metabolism and performance during prolonged submaximal exercise of approximately 60 min duration.

CHAPTER 2

REVIEW OF LITERATURE

2.1 CARBOHYDRATE METABOLISM

Endogenous carbohydrate (CHO) stores, in the form of muscle and liver glycogen, are important substrates for contracting muscles. Muscle and liver glycogen provide ATP for muscle contractile activity during exercise and fatigue has often been demonstrated to coincide with muscle glycogen depletion and/or hypoglycaemia (Bergstrom and Hultman, 1967; Christensen and Hansen, 1939; Hermansen et al., 1967; Vollestad et al., 1984). Furthermore, endurance exercise is enhanced when muscle glycogen levels are elevated prior to exercise (Bergstrom and Hultman, 1967). Researchers have, therefore, focused on the factors affecting CHO metabolism and strategies to maximize CHO levels before, during and after exercise in an attempt to maximize athletic performance.

2.1.1 Glycogen Utilization during Exercise.

The enzymes which regulate glycogen metabolism are glycogen synthetase and phosphorylase, both of which occur in active and inactive forms. Phosphorylase is the enzyme responsible for the breakdown of glycogen, in a process known as glycogenolysis. The *b* form is active in the presence of AMP. In contrast, the *a* form is active in the absence of AMP (Constable et al., 1986). This allosteric control allows the muscle cell to regulate its level of phosphorylase activity and therefore its level of glycogenolysis (Chasiotis, 1988). The degradation of glycogen is largely stimulated by hormonal factors, although its utilization is somewhat

multifactorial. It has been demonstrated that muscle glycogen utilization is enhanced when a greater muscle glycogen concentration is available to be metabolized (Gollnick et al., 1981; Sherman et al., 1981; Richter and Galbo, 1986) and that its degradation is enhanced during the initial stages of exercise (Bergstrom and Hultman, 1967). It is also well documented that the rate of muscle glycogenolysis increases with increasing exercise intensity (Vollestad and Blom, 1985). The cascade of glycogenolysis is largely affected by hormonal factors; adrenaline (Galbo et al., 1975; Gerich et al., 1976) and low blood glucose concentrations stimulating glucagon secretion in the liver (Gerich et al., 1976) thereby stimulating liver glycogenolysis (Richter et al., 1981; Wasserman et al., 1984). Additionally, it has been recently suggested that adrenaline-stimulated liver glycogenolysis during exercise may be subject to a feed forward mechanism in a workload dependent relationship (Kjaer et al., 1986; Kjaer et al., 1987; Sonne and Galbo, 1985). Stressful situations or exercise stimulates the secretion of adrenaline in muscles which subsequently enhances muscle glycogenolysis (Arnall et al., 1986; Cheetham et al., 1986; Jansson et al., 1986; Richter et al., 1980; Richter, 1984).

Glycogenolysis in resting rat muscle is sensitive to changes in adrenaline in a dose-dependent manner and specific fibres (slow twitch red) are more sensitive than others (fast twitch white) (Gorski, 1978). Similarly Chesley et al. (1994) recently reported that high levels of adrenaline were not important in enhancing muscle glycogenolysis in rat muscles comprised predominantly of fast twitch fibres during high intensity stimulation. Furthermore type 1 fibres in electrically stimulated human muscle undergo greater glycogenolysis when adrenaline is infused (Greenhaff et al., 1991). McDermott et al. (1987) reported that levels of muscle glycogenolysis were quite similar in normal contracting rat muscle and non-contracting rat muscle

that had been injected with adrenaline, demonstrating that contractile activity of the muscle is not necessary for glycogen breakdown to occur. This function of adrenaline illustrates its importance in providing substrates such as lactate for hepatic gluconeogenesis from both contracting and non-contracting muscle and has been postulated to be the major function of adrenaline stimulated muscle glycogenolysis (Arnall et al., 1986). When levels of adrenaline are artificially raised during submaximal exercise in man, muscle glycogenolysis is enhanced (Jansson et al., 1986). Conversely, when the source of this hormone is removed in rats via adrenodemedullation, muscle glycogenolysis is impeded (Arnall et al., 1986; McDermott et al., 1987; Richter et al., 1980; Richter et al., 1981). Interestingly, the effect of adrenaline on liver glycogenolysis is somewhat less clear. It has been demonstrated that adrenodemedullated animals have a reduced rate of hepatic glycogenolysis during exercise compared to exercising SHAM controls (Richter, 1980; Richter et al., 1981). In contrast infusion of high physiological concentrations of adrenaline does not enhance hepatic glycogenolysis any greater than infusion of a control (Arnall et al., 1986; Carlson et al., 1985). These discrepancies may be explained by variations in methodologies. Other factors, however, have been reported to affect hepatic glycogenolysis during exercise and among these include the circulating levels of glucagon and stimulation of sympathetic nerves to the liver (Nobin et al., 1977), although one study has demonstrated that surgical removal of nerves of the liver had no effect on hepatic glucose output (Sonne et al., 1985).

In muscle, glycogenolysis can be triggered by other mechanisms that function on more localized level. During muscular contraction, calcium is released from vesicles of the sarcoplasmic reticulum at levels that have been reported to activate phosphorylase *b* kinase (Chasiotis et al., 1982) and has

been suggested to be the primary mechanism for this activation in short-term intense tetanic stimulation in rat hindlimb (Chesley et al., 1994). Phosphorylase *b* is then phosphorylated by active phosphorylase kinase to the active phosphorylase *a* form. It is well documented that glycogenolysis is increased in skeletal muscle under conditions that also increase phosphorylase *a* (Chasiotis et al., 1983; Chasiotis, 1988). Phosphorylase *a* stimulates the breakdown of glycogen to form glucose-1-phosphate. On a cellular level, muscle glycogenolysis can be stimulated by high levels of cyclic AMP which is regulated by adrenaline. Adrenaline induces a transformation of phosphorylase from an inactive to an active form. Adrenaline triggers the enzyme adenyl cyclase, at the cellular membrane, catalyzing the formation of cyclic AMP, which then follows the cascade as mentioned above to activate phosphorylase *a* (Chasiotis et al., 1983; Freedland and Briggs, 1978). Evidence of this has been reported by Chasiotis et al. (1983) who demonstrated that the proportion of phosphorylase in the *a* form in the basal state was estimated to be 22.5% and during adrenaline infusion the proportion of phosphorylase in the *a* form increased to 80-90%. Interestingly, despite nearly all the phosphorylase being in the *a* form during infusion the level of glycogenolysis was extremely low. It was suggested by the authors that the availability of the substrate inorganic phosphate (Pi) from ATP-PCr splitting limits phosphorylase activity in the muscle and thus has a integral role in regulating muscle glycogenolysis (Chasiotis, 1988).

2.1.2 Glucose Metabolism

Skeletal muscle is the primary site for blood glucose utilization during exercise. The utilization of blood glucose is affected by its delivery and its uptake, and both these factors are increased during exercise. It has been

demonstrated that the rate of blood glucose uptake in human skeletal muscle increases in proportion to workload during leg exercise, increasing up to 10-20 times during moderate to heavy exercise (Wahren et al., 1971). More recently, Katz et al. (1986) investigated the effect of skeletal muscle blood glucose uptake during submaximal (50% VO_{2max}) and maximal exercise (97% VO_{2max}). These authors observed that an increased blood glucose uptake by the muscle does not necessarily reflect an increased rate of utilization during maximal exercise and thus at least part of the glucose taken up at this intensity (97% VO_{2max}) is not metabolized. It was suggested that the build up of intracellular glucose 6-phosphate (G-6-P) caused the inhibition of hexokinase and, therefore, prevented further glucose utilization. Under these circumstances glycogenolysis is enhanced and preferentially used as an energy substrate (Katz et al., 1986). Interestingly, these authors suggest that this dissociation between blood glucose uptake and subsequent utilization occurs at about 75% VO_{2max}. During submaximal intensity exercise however, blood glucose demand equals supply and thus blood glucose does not accumulate in the muscle. At these intensities all of the blood glucose taken up by the leg is metabolized (Katz et al., 1986).

To maintain this augmented muscle glucose uptake, liver glycogenolysis is stimulated to meet the demand by increasing glucose output. As exercise continues a larger proportion of glucose output by the liver is met by gluconeogenesis (Wahren et al., 1971). At the tissue level, glucose uptake is regulated by the rate at which glucose can transport across the cell membrane and is mediated through a mobile transporter-protein mechanism that combines with glucose to gain entry to the cell. Recent work has identified two such transporter isoforms: GLUT 1 and the more common skeletal muscle glucose transporter GLUT 4 (Klip and Paquet, 1990). Glucose uptake has been extensively researched and factors regulating

glucose transport have been reported to include an increase in intracellular calcium (Holloszy and Narahara, 1967; Holloszy et al., 1986), tissue hypoxia (Idstrom et al., 1985) and the energy state of the muscle tissue (Walker et al., 1982).

The bulk of literature on glucose transport has focused on the effects of insulin, exercise and their interaction. Early work demonstrated that insulin and muscle contractile activity increased the glucose transport across the cell membrane (Holloszy and Narahara, 1965) however, muscle contractile induced glucose transport was dependent upon the presence of insulin (Berger, 1975). Recent work has shown that the effects of maximal insulin stimulation and exercise are somewhat additive on skeletal muscle permeability to glucose (Zorzano et al., 1986) unless, however, there is prolonged exposure to unphysiologically high insulin concentrations (Constable et al., 1988). Insulin-stimulated glucose uptake has been observed to be related to GLUT-4 protein transport content in rats (Kern et al., 1993) and in humans (Ebeling et al., 1993). Insulin stimulates an increase in the number of glucose transport molecules at the plasma membrane by translocation from an intracellular pool (Fushiki et al., 1989; James et al., 1989; Wardzala and Jeanrenaud, 1981). Fushiki et al. (1989) reported that the addition of insulin resulted in the concentration of plasma membrane transporters increasing from 16.8 to 31.6 $pmol.mg^{-1}$ membrane protein and intracellular glucose transporters decreasing from 29.8 to 17.5 $pmol.mg^{-1}$. Glucose kinetic studies have reported that insulin increases the transport of glucose in skeletal muscles by increasing the maximal rate of transport with little or no change in the apparent affinity of the transport molecule (Sternlicht et al., 1988; Sternlicht et al., 1989) and has been suggested to be the result of an increase in the total number of transport molecules, an increased turnover rate for glucose in the existing molecules,

or both (Sternlicht et al., 1988). In contrast, work by Wallberg-Henriksson and Holloszy, (1984) have reported that large increases in glucose uptake occurred in response to muscle contraction in the absence of insulin in both diabetic and control rats. The mechanism for glucose transport during exercise has been suggested to be the same as that for insulin or use the same transport molecule (Fushiki et al., 1989; Holloszy and Narahara, 1965; Ploug et al. 1987).

It has been reported in studies using rat models that exercise stimulates glucose transporter translocation (Fushiki et al., 1989; Goodyear et al., 1990; King et al., 1989; Sternlicht et al., 1989). Fushiki et al. (1989) reported that one bout of exercise induced translocation of the glucose transporters from the intracellular to the plasma membrane in a similar way that insulin does, although it did not increase the number of transporters. Interestingly, Koivisto et al. (1993) also reported no change in muscle glucose transport protein content (GLUT-4) after one bout of acute exercise in human subjects, although using their technique it was not possible for them to determine if translocation from the intracellular pool to the plasma membrane had occurred. In contrast, cytochalasin binding data reported by Sternlicht et al. (1989) demonstrate that insulin stimulation increased the number of binding sites compared with controls, however exercise had no effect on the number of binding sites. This indicated that exercise and insulin glucose transport work by different mechanisms. Finally, endurance training has been demonstrated to induce elevations in GLUT-4 concentrations in rats (Ploug et al., 1990) and man (Houmard et al., 1991; Koivisto et al., 1992). Short term training cessation does not adversely affect GLUT-4 protein levels in the skeletal muscles of man (Houmard et al., 1993), however more recently McCoy et al. (1994) demonstrated that muscle Glut-4 protein content and oxidative capacity decreases in parallel after detraining, suggesting that the

discrepancies between these studies may be related to the detraining stimulus.

Muscle glucose uptake has also been reported to be affected by the concentration of muscle glycogen (Chesley et al., 1995; Fell et al., 1982; Hargreaves et al., 1995; Hespel and Richter, 1990) and the pattern of glycogen depletion in skeletal muscle (James et al., 1985). The aforementioned authors demonstrated a close relationship between an increase in muscle glucose uptake during exercise in specific fibres and the amount of glycogen degradation that occurred during exercise in those same fibres, namely those that would be more involved in moderate prolonged exercise (red gastrocnemius and soleus)(James et al., 1985). Work by Ploug et al. (1987) would seem to support this finding as they demonstrated that the effects of both contractions and insulin, on increasing glucose transport, are greater in red than white muscle fibres.

The effect of circulating catecholamines have also been reported to influence glucose uptake by the muscle and have been suggested to be under alpha (Richter et al., 1982) or beta-adrenergic control (Young et al., 1985). Young et al. (1985) used beta-blockers to demonstrate beta-adrenergic control. In contrast, others have reported that exercise does not stimulate glucose transport via the beta-adrenergic receptor (Sternlicht et al., 1989). Adrenaline stimulation has been demonstrated to both depress (Jansson et al., 1986; Walaas and Walaas, 1950) and enhance glucose uptake in skeletal muscle (Richter et al., 1982; Young et al., 1985). The inconsistencies in the findings of these studies may be due in part to differences in the methodologies employed by these researchers but on the whole reflect a need to further elucidate the role of adrenaline on glucose transport.

Blood glucose uptake in skeletal muscle is affected by the availability of blood-borne fuel substrates. The concentration of glucose and FFA in the blood affect blood glucose uptake by the muscle and both can be manipulated through diet or infusion. The effects of these manipulations will be discussed in detail subsequently.

2.2 FAT METABOLISM

It is well established that lipids are a major fuel reservoir in the mammalian body (Oscai et al., 1990). Adipose tissue, skeletal muscle and to a lesser extent, plasma triglycerides, are significant fat depots that generate an important energy source for contacting skeletal muscle during prolonged submaximal exercise. Their catabolism via lipolysis (breakdown of triglycerides into glycerol and non-esterified fatty acids) and subsequent beta oxidation in skeletal muscle mitochondria offer an almost endless supply of energy during low intensity exercise (Sahlin, 1992)

It is well known that lipid is mobilized following the hydrolysis of triglycerides in adipose tissue to form a pool of FFA and glycerol in the plasma (Freedland and Briggs, 1977). These albumin-bound FFA mobilized from adipose tissue are readily oxidized by muscle (Havel, 1974; Kiens et al., 1993) and have been reported to constitute approximately 50% of the fat oxidized during exercise (Havel et al., 1964; Issekutz et al., 1968). FFA can also form ketone bodies in the liver and during starvation conditions can account for nearly 80% of the fatty acids catabolised (Krebs et al., 1969).

The remaining fat oxidized during exercise was first suggested by Havel et al, (1967) to come from intramuscular sources, as the uptake of FFA from plasma and plasma triglyceride derived fatty acids could account for little more than half of the carbon dioxide, which was estimated from measurements of respiratory quotient derived from the oxidation of fatty acids in the exercising legs. This was supported by a body of research which demonstrated a significant reduction in the concentration of triglycerides in skeletal muscle during exercise (Carlson et al., 1971; Froberg., 1971; Gollnick and Saltin., 1988; Reitman et al., 1973). In addition, it has also been reported that triglyceride mobilization can be observed in electrically stimulated muscle. These studies demonstrated that when frequency of muscular contraction was increased a greater reduction in triglyceride concentration was observed (Hopp and Palmer., 1990; Spriet et al., 1986).

In contrast, Kiens et al. (1993) recently reported data which suggests that intramuscular triglycerides provide minimal energy during exercise. It was suggested by these authors that the observed increase in lipid oxidation was due to an enhanced FFA uptake by the muscle as a decrease in intracellular triglyceride level was not observed. Interestingly the mobilization of intramuscular triglycerides has been reported to be mainly isolated to red muscle fibres which have a greater oxidative capacity (Baldwin et al., 1973; Froberg., 1971). It is noteworthy also that the activity of lipoprotein lipase (LPL) is higher in muscles containing mainly red fibres as opposed to those containing white (Borensztajn et al., 1975; Tan et al., 1975; Tan et al., 1977). The observation that intramuscular lipolysis does not occur in fast twitch muscle fibres has been suggested to be due to their very low oxidative capacity (Okano and Shimojo et al., 1982) or the ability of the fast twitch fibres to accumulate high levels of lactate (Gorski, 1992). Lactate accumulation increases the re esterification of FFA and reduce lipolysis

(Fredholm, 1969; Issekutz and Miller., 1962; Miller et al., 1964). Other factors which may affect muscle triglyceride metabolism include muscle glycogen concentration (Stankiewicz-Choroszucha and Gorski, 1978b) and the plasma FFA concentration (Carlson et al., 1965; Carlson et al., 1971; Stankiewicz-Choroszucha and Gorski., 1978b).

The third pool of lipids for muscular contraction are plasma triglycerides. Havel et al. (1967) reported that the contribution of plasma triglyceride fatty acids were less than 10% of the fatty acids oxidized in the leg during exercise. Prolonged exercise results in a reduction in plasma triglyceride stores (Holloszy et al., 1964; Oscai et al., 1972) and an increase in the uptake of plasma triglyceride derived fatty acids by the contracting muscle (Kiens et al., 1993; Terjung et al., 1982; Terjung et al., 1983). Despite this, their contribution to energy production is considered small and it has been suggested that plasma triglycerides may be used to replenish the decreased fat stores in muscle during exercise (Oscai et al., 1990).

In mammalian fat cells the hydrolysis of adipose tissue triglyceride is catalyzed by the "hormone-sensitive" lipase, or lipoprotein lipase situated on the endothelial surface of blood capillaries (Fredrickson et al.; Khoo and Steinberg, 1974). Enzyme regulation of muscle triglyceride hydrolysis is not well investigated. Muscle appears to contain 2 different fractions of lipoprotein lipase (Borensztajn, 1979; Robinson, 1970), although more recently it was suggested that 3 different lipoprotein lipase fractions have been found all with different rates of activity at particular pH (Gorski, 1992). It has been hypothesized by Oscai and Palmer, (1988; 1990) that intracellular lipoprotein lipase plays a direct role in muscle triglyceride hydrolysis. This hypothesis was based on their observation that during exercise FFA levels were elevated in the intracellular component of the muscle at the same time

that there was a corresponding increase in intracellular lipoprotein activity. According to Gorski, (1992), however, this hypothesis is somewhat debatable based on the observations that the optimal pH for lipoprotein lipase is 8.5 and the pH inside myocytes is 7.0 at rest, decreasing even further with contraction (Sahlin, 1978). The activity of lipoprotein lipase at a pH of 7.0 is negligible (Strothfeld and Heugel, 1984). In addition, although lipoprotein lipase is manufactured in the myocytes, it is transported to the cell surface in vesicles and therefore is without contact with the triglyceride particles (Gorski, 1992)

Adipose tissue lipolysis is somewhat regulated by hormonal control and the cyclic AMP cascade (Gorski, 1992; Shepherd and Bah, 1987). Catecholamine injection causes a rapid release of FFA from adipose tissue (Shepherd and Bah, 1987), however lipolysis in fat cells is also stimulated by glucagon, growth hormone and thyroid hormones (Fain, 1973). In muscle, epinephrine and glucagon has been reported to stimulate the activity of intracellular LPL in rat skeletal muscle (Miller et al., 1988) and rat heart (Borensztajn et al, 1973) respectively. In addition nor-adrenaline infusion resulted in a significant reduction in intramuscular triglyceride concentration in humans (Froberg et al, 1975). Lipolysis in adipose tissue is under beta-adrenergic control (Lundborg et al., 1981; Smith, 1983) and recent evidence suggests that the hydrolysis of intramuscular triglycerides may also be regulated by beta-adrenoceptors (Cleroux et al., 1987). Intramuscular triglyceride utilization in slow twitch fibres of rats was impaired with non-selective beta-blockade (Stankiewicz-Choroszucha and Gorski, 1978) and more recently in humans complete impairment of intramuscular triglyceride utilization was reported with non-selective beta-blockade (Cleroux et al., 1987).

Lipid metabolism is also influenced by the intensity of exercise undertaken. It was suggested by Jones et al., (1980) that light exercise may favour the release of FFA from adipose tissue and subsequent uptake by the muscle, whereas during heavy exercise may inhibit adipose mobilization and stimulate lipolysis in muscle. Further work is needed to confirm this conclusion. It is well known however, that total fat combustion is affected by exercise intensity. Although the uptake of FFA by the active muscles depends upon the plasma concentration of FFA (Ahlborg et al., 1974; Paul., 1970) respiratory exchange ratio data demonstrate that the importance of FFA as a fuel relative to CHO is greater with increasing duration of exercise and diminishes as the intensity of exercise increases (Christensen and Hansen 1939; Hagenfeldt 1979).

Lipid mobilization may also be affected by the presence of other energy substrates (eg. Glucose) and hormones such as insulin. The mechanism by which they affect fat metabolism will discussed subsequently.

2.3 PROTEIN METABOLISM

The percentage of energy derived from protein catabolism during exercise is equivocal. Both the liver and the skeletal muscles have the ability to metabolize protein to produce energy (Miller, 1962; Odessey and Goldberg, 1972), however early investigations into protein metabolism surmised that energy produced via protein breakdown during exercise was either non existent or minimal. Recent evidence, however, suggests that some energy is produced from protein catabolism during exercise (Booth and Watson, 1985; Lemon and Mullin, 1980; Kasperak et al., 1982; Poortmans, 1984; Rennie et al., 1981). The energy derived from the breakdown of

protein could be considered small but has been estimated to contribute between 3 and 10% of the total energy production during prolonged exercise (Brooks, 1987; Hood and Terjung, 1990; Poortmans, 1984; Rennie et al., 1981).

Historically, many studies investigated protein metabolism by measuring urinary urea as an index of protein breakdown (Dohm et al., 1977; Lemon and Mullin, 1980). This marker however, may not accurately reflect protein degradation or mean that energy was derived from the breakdown of protein (Lemon et al., 1984). For example, sweat loss , water intake and changes in blood flow to the kidneys during exercise may affect urea production and excretion (Lemon and Mullin, 1980; Lemon et al., 1983). Additionally 3-methylhistidine urinary excretion has also been used as a marker of skeletal muscle protein breakdown during exercise (Dohm et al., 1982a; Dohm et al., 1987; Rennie et al., 1981). It has been demonstrated in rats that increases in 3-methylhistidine urinary excretion can be contributed to by protein loss from the gastrointestinal tract (Wassner and Li, 1982) and therefore may also not accurately reflect skeletal muscle protein catabolism (Dohm et al., 1987).

There is considerable evidence that amino acids are catabolised by skeletal muscle during exercise to produce energy (Goldberg and Odessey, 1972; Graham and Maclean, 1990; Hood and Terjung, 1990; Lemon and Mullin, 1980; Maclean et al., 1991; Maclean et al., 1994; Wagenmakers et al., 1990). The availability of amino acids for metabolism is limited by their presence in the tissue free amino acid pool, which is a function of the rate of protein synthesis and protein degradation. During exercise the rate of protein synthesis decreases (Booth and Watson, 1985; Dohm et al., 1980; Rennie et al., 1981) and as mentioned previously, the rate of protein catabolism

increases, thus increasing the tissue free amino acid pool. The main amino acids metabolized by skeletal muscle are alanine, glutamate, aspartate and in particular the branch chain amino acids (BCAA) leucine, isoleucine and valine (Goldberg and Odessey, 1972; Graham and Maclean, 1990). Ahlborg et al. (1974) demonstrated that during prolonged light exercise BCAA were released from the splanchnic bed and were taken up by the working muscle. Additionally amino acid infusion has demonstrated a significant increase (70%) in skeletal muscle BCAA uptake (Gelfand et al., 1986) and more recently BCAA supplementation has been observed to result in a significant uptake of BCAA, as well as a suppression of endogenous protein catabolism during exercise (Maclean et al., 1994).

Although the BCAA are important to skeletal muscle metabolism the other amino acids are also functionally significant in potential energy production, that being the provision of substrate (carbon skeletons) to the liver for gluconeogenesis. Ahlborg et al. (1974) reported that most of the other amino acids (other than BCAA) were released from the muscle and taken up by the liver during light exercise. Alanine is one amino acid in particular that has been reported to have increased efflux from skeletal muscle and uptake by the liver during exercise (Felig and Wahren, 1971). Additionally alanine and glutamine, the latter which can be converted to alanine in the gut (Hood and Terjung, 1990) account for about 50% of the amino acid efflux from the muscle during exercise (Ahlborg et al, 1974; Felig and Wahren, 1971).

Ammonia is produced in skeletal muscle during exercise and thus may also serve as a marker of protein degradation (Maclean et al., 1991). Additionally, plasma ammonia concentrations correlate with both muscle cell ammonia production and muscle ammonia release (Graham and Maclean,

1990). The two main sources of ammonia production during exercise in skeletal muscle evolve from amino acid catabolism and the purine nucleotide cycle (PNC) (Graham and Maclean, 1990; Wagenmakers et al., 1990). The PNC is a series of reactions involved in the deamination of AMP to IMP and ammonia (Graham and Maclean, 1990). During high intensity, short-term exercise, ammonia is produced in significant proportions in skeletal muscles (Babij et al., 1983; Katz et al., 1986). Under these exercise conditions the increase in ammonia accumulation is in a 1:1 stoichiometric relationship with the decrease in total adenine nucleotides (TAN) and increase in IMP, demonstrating that all of the ammonia produced during high intensity exercise comes from AMP deamination (Katz et al., 1986; Meyer and Terjung, 1980; Stathis et al., 1994).

In contrast, plasma ammonia levels rise during prolonged exercise (Graham et al., 1987; Maclean et al., 1991; Maclean et al., 1994; Snow et al., 1995; Wagenmakers, 1990) despite no change in PNC intermediates (Maclean et al., 1991; Maclean et al., 1994; Norman et al., 1987; Snow et al., 1995). In addition administration of BCAA in the pre-exercise period results in an enhanced plasma ammonia concentration (Maclean et al., 1994; Wagenmakers et al., 1990). These findings suggest that much of the ammonia produced during endurance exercise comes from the metabolism of amino acids and during short-term high intensity exercise from the deamination of AMP.

Other factors that appear to affect amino acid metabolism include the dietary status of the subject and carbohydrate availability. Protein catabolism is increased during fasting (Fryburg et al., 1990) and increases in plasma ammonia are enhanced following a low carbohydrate diet and low muscle glycogen concentrations (Maclean et al., 1989). Recently Snow et al. (1995)

demonstrated that plasma ammonia concentrations during exercise were lower when a carbohydrate supplement was ingested during exercise, suggesting that the availability of carbohydrate influences amino acid metabolism.

2.4 CARBOHYDRATE FEEDING AND EXERCISE.

2.4.1 CHO Ingestion during Exercise.

It is well established that CHO feeding during prolonged exercise can increase endurance performance (Askew et al., 1986; Coggan and Coyle , 1987; Coggan and Coyle, 1988; Coggan and Coyle, 1989; Coyle et al., 1983; Coyle and Coggan, 1984; Coyle and Coggan, 1986; Hargreaves et al., 1984; Ivy et al., 1983; Millard-Stafford et al., 1992; Mitchell et al., 1988; Murray et al., 1989). Prior to investigations which utilized the needle biopsy technique it was assumed that the improved performance was due to a sparing of muscle glycogen (Coyle et al., 1993). Subsequent studies have demonstrated that muscle glycogen is not spared when CHO is fed during prolonged steady state exercise (Coyle et al., 1986; Fielding et al., 1985; Flynn et al., 1987; Hargreaves and Briggs, 1988; Mitchell et al., 1989). Although ingestion of glucose during prolonged exercise at 30% VO_{2max} results in a significant enhancement of muscle glucose uptake (Ahlborg and Felig, 1976), during exercise of a higher intensity (70% VO_{2max}) the increase in musle glucose uptake is relatively small and does not contribute significantly to substrate

utilization (McConell et al., 1994). It appears, therefore, that during moderate to high intensity endurance exercise glycogen utilization and muscle glucose uptake are not affected to a great extent when intramuscular glycogen is not depleted. Accordingly, when muscle glycogen stores are adequate to sustain 2 hr of exercise CHO feeding has no effect on subsequent exercise performance (Widrick et al., 1993). The likely mechanism for the increased performance as a result of CHO feeding during exercise is an increase in glucose delivery and extraction late in exercise when muscle glycogen stores are low (Coggan and Coyle, 1987; Coggan et al., 1991). It is probable that the increase in muscle glucose uptake results from an increase in glucose transport since the skeletal muscle glucose transporter (GLUT 4) is activated when glycogen stores are depleted (Hespel and Ricter, 1990).

2.4.2. Pre-exercise Feeding of Carbohydrate.

Unlike the effect of CHO feeding during exercise, the benefits of pre-exercise CHO feeding are equivocal. Early investigations indicated that pre-exercise feeding may be detrimental to metabolism and/or endurance performance (Ahlborg and Felig., 1977; Costill et al., 1977; Foster et al., 1979; Keller and Schwarzkopf, 1984). As a result, the American dietetic Association cautioned athletes against this practice in 1987 (Alberici et al., 1993). Subsequent investigations have questioned these initial findings suggesting that this practice may either increase (Alberici et al., 1993; Gleeson et al., 1986; MacLaren et al., 1994; Neuffer et al., 1987; Okano et

al., 1988, Peden et al., 1989; Sherman et al., 1989; Sherman et al., 1991; Thomas et al., 1991; Wright et al., 1991; Ventura et al., 1994), or have no effect (Chryssanthopoulos et al., 1994; Decombaz et al., 1985; Devlin et al., 1986; Hargreaves et al., 1987; Snyder et al., 1993; Sharp et al., 1993) on exercise performance. The differences in metabolism and performance reported by many studies have made the effect of pre-exercise CHO ingestion unclear.

Since fatigue during endurance exercise often coincides with glycogen depletion and/or hypoglycaemia (Coggan and Coyle, 1987; Coyle et al., 1986) one would expect that increasing CHO availability would be of subsequent benefit. Unlike CHO feeding during exercise which blunts the secretion of insulin (Coyle et al., 1983; Ivy et al., 1979), pre-exercise glucose ingestion results in hyperinsulinaemia (Ahlborg and Felig, 1977; Bonen et al., 1981; Costill et al., 1977; Devlin et al., 1986; Gleeson et al., 1986; Guezennec et al., 1989; Hargreaves et al., 1987; Koivisto et al., 1982; Koivisto et al., 1985; Sherman et al., 1991; Thomas et al., 1991; Wright et al., 1991). Elevated insulin subsequently effects metabolism during exercise such that blood glucose (Ahlborg and Felig, 1977; Costill et al., 1977; Decombaz et al., 1985; Devlin et al., 1986), free fatty acids (Ahlborg and Felig, 1977; Gleeson et al., 1986; Koivisto et al., 1985), muscle (Costill et al., 1977; Hargreaves et al., 1985) and hepatic glycogenolysis (Felig and Warren, 1979) are altered. When CHO is administered at rest insulin will be elevated for several hours (Heath et al., 1983; Montain et al., 1991) before returning to

basal levels. The time at which the pre-exercise CHO meal is ingested is, therefore, a critical factor when assessing the potential beneficial or detrimental effects of such a practice.

2.4.2.1 Glucose Ingestion 30 min -2 hr before Exercise

If glucose is ingested 1-2 hours before exercise, a state of hyperinsulinaemia at the onset of exercise results in a rapid decline in blood glucose concentration (Ahlborg and Felig, 1977; Bonen et al., 1981; Chryssanthopoulos et al., 1994; Costill et al., 1977; Decombaz et al., 1985; Devlin et al., 1986; Guezennec et al., 1989; Hargreaves et al., 1987; Koivisto et al., 1982; Koivisto et al., 1985; Montain et al., 1991; Sherman et al., 1991; Thomas et al., 1991). This decline in blood glucose concentration generally occurs during the first 10 to 20 minutes of exercise and falls below the concentration defined as being hypoglycaemic (Costill et al., 1977; Montain et al., 1991).

Blood glucose concentration reflects the balance between glucose uptake by tissues and glucose output by the liver (Coyle et al., 1985; Montain et al., 1991). Blood glucose is determined by the synergistic effect of insulin and muscular contraction on muscle glucose uptake (Defronzo et al., 1981; Ploug et al., 1987) and the reduction in hepatic glycogenolysis mediated by hyperinsulinaemia (Felig and Wahren, 1979), hypergluconaemia (Ahlborg et al., 1974; Felig et al., 1972; Felig and Wahren, 1979) and an increase in

sympathoadrenal response (Galbo et al., 1975). A suppression in glucagon and increase in insulin concentration has been observed during exercise with CHO ingestion/infusion prior to and during exercise, which may result in a reduction in liver glycogenolysis (Alborg and Felig, 1977; Felig and Wahren, 1979; Luyckx et al. 1978; Mitchell et al., 1990). The increased peripheral uptake of glucose during exercise (Ahlborg and Felig, 1977) mediated by the action of insulin may, therefore, be the mechanism which causes this rapid decline in blood glucose following pre-exercise CHO ingestion (Ahlborg and Felig, 1977). Of note, however, this study examined the effect of pre-exercise feeding on glucose kinetics during low intensity exercise. Further investigations examining glucose kinetics and pre-exercise CHO ingestion during exercise of a higher intensity are warranted. Although muscle glucose uptake and subsequent glucose oxidation may be elevated during the decline in blood glucose, an enhanced muscle glycogen utilization may occur subsequent to this period (Costill et al., 1977; Hargreaves et al., 1985).

Costill et al. (1977) and Hargreaves et al. (1985) observed an increase in muscle glycogen utilization during 30 min of exercise at 70-75% VO_{2max} when glucose was ingested in the hour before exercise. Of note, blood glucose concentrations declined to values lower than 3.5 $mmol.l^{-1}$ in the glucose ingestion trials. In contrast, this has not been reported in other studies during short term (30-60min) (Decombaz et al., 1985; Devlin et al., 1986; Fielding et al., 1987; Hargreaves et al., 1987; Levine et al., 1983) or prolonged exercise (Koivisto et al., 1985). Despite increased plasma insulin

concentrations resulting from prior CHO ingestion, blood glucose concentrations did not decline to values lower than 3.9 $mmol.l^{-1}$ in these latter studies. Furthermore, a subsequent study by Hargreaves et al. (1987) did not observe an increase in muscle glycogen utilization following pre-exercise glucose ingestion when blood glucose concentrations declined to 4.02 $mmol.l^{-1}$ at 15 min, compared with their previous study (Hargreaves et al., 1985) in which concentrations declined to an average of 3.18 $mmol.l^{-1}$. It was suggested by these authors that if the blood glucose concentrations do not decline to very low concentrations muscle glycogenolysis may not be enhanced. Results from these studies demonstrate that if blood glucose is reduced to very low levels then the contribution of this substrate to total CHO oxidation may be compromized resulting in a greater reliance upon endogenous glycogen stores.

Most studies investigating the effect of pre-exercise CHO ingestion in the hour before exercise have not observed an effect on performance, despite changes in metabolism as a result of the ingested meal (Alberici et al., 1993; Chryssanthopoulos et al., 1994; Decombaz et al., 1985; Devlin et al., 1986; Hargreaves et al., 1987; McMurray et al., 1983). Improvement in exercise time to exhaustion in fasted subjects has been demonstrated, however, when glucose has been ingested 45 min before exercise (Gleeson et al., 1986). These authors observed a state of hyperinsulinaemia before exercise, however a rapid decline in blood glucose level was not reported after the commencement of exercise. It is possible that the subjects

(untrained individuals) found the workload (70% VO_{2max}) physiologically stressful, resulting in the absence of an exercise induced rapid decline in blood glucose concentration. The stress of the exercise may augment hepatic glycogenolysis, as suggested by Montain et al., (1991). It is likely that this phenomenon was mediated by a feed-forward control of adrenaline as discussed in Chapter 2.1

2.4.2.2 Pre-exercise CHO Ingestion 3-4 hours before Exercise.

Ingestion of relatively large CHO meals 3-4 hours before exercise results in improvements in exercise performance (Neuffer et al., 1987; Sherman et al., 1989; Wright et al., 1991), likely to result from a considerable portion of the pre-event meal being disposed of as muscle or liver glycogen (Coyle et al., 1985; Neuffer et al., 1987). Coyle et al. (1985) observed a 42% increase in muscle glycogen concentration compared with a 12 hr fast when a large CHO meal was ingested 4 hr pre-exercise. Since endurance performance is related to pre-exercise muscle glycogen concentration (Bergstrom and Hultman, 1967; Sherman et al., 1981), increases in muscle glycogen concentration due to pre-exercise CHO ingestion would be a significant factor in performance in prolonged exercise (Coyle et al., 1985; Neuffer et al., 1987).

CHO ingestion several hours before exercise may nevertheless result in a sudden decline in blood glucose at the onset of (Coyle et al., 1985;

concentrations immediately prior to exercise (Coyle et al., 1985; Montain et al., 1991). A fast of at least 6 hours may be necessary before a normalization of these responses is observed and highlights the persistent action of insulin (Montain et al., 1991).

The increase in CHO availability following a large CHO meal several hours before exercise appears to offset any disadvantage caused by an insulin mediated reduction in blood glucose, plasma FFA and concomitant increase in CHO oxidation (Coyle et al., 1985; Sherman et al., 1989; Wright et al., 1991). Muscle glycogen utilization is increased when concentrations of this metabolite within the muscle are high (Chesley et al., 1995; Gollnick et al., 1981; Hargreaves et al., 1995; Sherman et al., 1981) since glycogen binds to phosphorylase to increase its activity (Hespel and Richter, 1992; Johnson, 1992). Pre-exercise CHO feeding several hours before exercise may, therefore, result in increased CHO oxidation via enhanced muscle glycogen utilization (Coyle et al., 1985). Alternatively, the improved performance may be caused by increased blood glucose availability (Montain et al., 1991; Sherman et al., 1989; Wright et al., 1991).

since muscle samples were not obtained, feeding rats with fructose during exercise results in liver glycogen synthesis, and increased liver glucose output during exercise (Sonne and Galbo., 1986).

In contrast to the study by Okano et al. (1988), most of the literature indicates that the pre-exercise ingestion of fructose offers no ergogenic benefit, despite reducing the disturbance of some metabolic parameters (Decombaz et al., 1985; Hargreaves et al., 1987; McMurray et al., 1983; Ventura et al., 1994). Of note, in the aforementioned studies, subjects exercised in a fasted state, in contrast with the study of Okano et al. (1988).

2.5 GASTRIC EMPTYING AND CHO OXIDATION.

2.5.1 Gastric Emptying

The rate at which fluids and nutrients are made available to the intestine can place restraints on their absorption and subsequent incorporation into the blood stream (Rehrer, 1994). Hence, the rate of gastric emptying is significant when CHO is administered orally before and during exercise. Gastric emptying is influenced by the effects of exercise (Costill and Saltin, 1974; Neuffer et al., 1986), CHO concentration/osmolality (Costill and Saltin, 1974; Foster et al., 1980; Hunt and Pathak, 1960; Mitchell et al., 1989; Naveri et al., 1989) and volume of the meal/fluid (Costill and Saltin, 1974;

Hunt et al., 1985; Mitchell and Voss, 1991; Noakes et al., 1991b; Ryan et al., 1989).

The effect of exercise on the rate of gastric emptying is equivocal since it either stimulates (Neuffer et al., 1986), impairs (Mitchell et al., 1989) and does not alter (Costill and Saltin, 1974; Feldman and Nixon, 1982) gastric emptying. Gastic emptying may be stimulated at lower exercise intensities (Neuffer et al., 1986) and reduced during intermittent (Mitchell et al., 1989) or higher exercise intensities (>70% VO_{2max}) (Cole et al., 1993; Neuffer et al., 1986; Sole and Noakes, 1989). Increasing the CHO concentration of a beverage above 5-8% CHO reduces the the rate of gastric emptying (Foster et al., 1980; Mitchell et al., 1989) but enhances the the delivery of CHO (Mitchell et al.1989). Gastric emptying can further be enhanced by ingesting CHO polymers compared with the ingestion of simple sugars (Coyle et al., 1978; Foster et al., 1980; Nueffer et al., 1986; Sole and Noakes, 1989). This may be due, in part, to polymerised glucose having a molecular weight five times that of free glucose and only one fifth the osmolality, thereby reducing the effect on the osmoreceptor vesicles as postulated by Hunt and Knox, (1968). Despite a greater rate of gastric emptying with the ingestion of polymers, there would appear to be no benefit with respect to the oxidation rate of glucose in contrast with glucose polymers during exercise (Hawley et al., 1991; Massicotte et al., 1989). Gastric volume may be a more important factor than CHO content or osmolality in affecting gastric emptying rate (Noakes et al., 1991b). Several studies employing

repetitive feedings during exercise with differing CHO concentrations have demonstrated the rates of gastric emptying to be quite similar during exercise (Rehrer et al., 1990; Ryan et al., 1989; Sole and Noakes, 1989). When high volumes are regularly administered during exercise, compared with low volumes, gastric emptying rate is enhanced (Mitchell and Voss, 1991). The mechanism for improved gastric emptying may be the influence of larger volumes affecting intragastric pressure (Ryan et al., 1989). At rest, solids empty from the stomach more slowly than liquids (Fink et al., 1983; Malagelada, 1977). and would appear to be related to particle size (Meyer et al., 1981).

2.5.2 CHO Oxidation

CHO ingestion in the hours before, can be readily oxidized during, exercise (Decombaz et al., 1985; Guezennec et al., 1989; Jandrain et al., 1984). Decombaz et al. (1985) reported that the ingestion of 70g of glucose or fructose 60 min before exercise at 61% VO_{2max} for 60 min resulted in similar rates of oxidation for the respective nutrients. In contrast, Massicotte et al. (1986) reported a significantly lower oxidation of fructose compared with glucose during exercise. Subsequent investigations have confirmed this (Guezennec et al., 1989; Jandrain et al., 1993; Massicotte et al., 1989) which may be due to a slow conversion of fructose into glucose by the liver and subsequent oxidation by the muscles (Chen and Whistler, 1977). If the fructose is administered before exercise then it is most likely to be stored as

liver glycogen and used preferentially when exercise begins (Massicotte et al., 1992).

2.6 THE GLYCAEMIC INDEX.

Ingested CHO foods elicit different glycaemic responses and different rates of absorption with or without stimulating insulin secretion (Behall et al., 1988; Crapo et al., 1976; Crapo et al., 1977; Horowitz and Coyle., 1993; O'Dea et al., 1980; Thomas et al., 1991). The glycaemic index (GI) is a method of quantifying the glycaemic response of CHO foods ingested in human subjects (Brand and Collier, 1991; Jenkins et al., 1981). The GI is a ratio of the blood glucose area of 50g of a test food (CHO) to the blood glucose area of 50g of a reference food (bread or glucose) multiplied by 100 (Jenkins et al., 1983; Jenkins et al., 1984; Trout et al., 1993). The glycaemic response to ingested complex carbohydrates can differ (Crapo et al., 1976; Crapo et al., 1977; Horowitz and Coyle, 1993; Jenkins et al., 1981; Jenkins et al., 1983; Jenkins et al., 1984) hence, the GI is a more accurate way of describing the blood glucose response to a CHO than from the chemical composition (Jenkins et al., 1984). For example, potato and sugars such as glucose and sucrose elicit a similar glycaemic response following ingestion (Crapo et al., 1977; Horowitz and Coyle, 1993). Several food factors may be responsible for differences in the glycaemic response. These factors may be chemical or physical, affecting the access of intestinal hydrolytic enzymes to the ingested CHO, therefore, affecting the rate of digestion and subsequent

absorption (O'Dea et al., 1980). Such factors include; the disruption of the botanical structure (Golay et al., 1986; Haber et al., 1977; O'Dea et al., 1980), particle size (Jenkins et al., 1986), gelatinisation (Holm et al., 1988), amylose-amylopectin ratio (Behall et al., 1988; Goddard et al., 1984; Granfeldt and Bjorck, 1991), differences in preparation (Jenkins et al., 1981; O'Dea et al., 1980; Reaven et al., 1979; Wolever, 1990), the degree of ripeness (Wolever, 1990) and the presence of anti-nutrients (Brand and Collier, 1991; Yoon et al., 1983). The glycaemic response of starchy foods is negatively correlated with the rate of gastric emptying (Mourot et al., 1988) and can also be affected by the ingestion of other nutrients (Burke et al., 1995; Horowitz and Coyle, 1993). The addition of fat to a potato and rice meal results in a lower glycaemic response than when compared with a potato and rice meal (Horowitz and Coyle, 1993). This may be related to the effect that fat has on delaying gastric emptying (Thomas, 1957) and interfering with the enzymes responsible for CHO digestion (Collier and O'Dea, 1983).

The glycaemic response of CHO foods are generally classed as having a low, moderate or high glycaemic index. HGI foods (>70) may include potato, glucose, bread, rice. In contrast, LGI foods (<50) include legumes such as beans, peas and lentils (Jenkins et al., 1981). LGI foods stimulate a lower insulin secretion following ingestion (Jenkins et al., 1983; Thomas et al., 1991), a more stable blood glucose response (Jenkins et al., 1981; Jenkins et al., 1983; Thomas et al., 1991) and increased plasma FFA

concentrations during subsequent exercise (Thomas et al., 1991). In contrast, pre-exercise ingestion of HGI foods result in increased insulin secretion, decreased FFA concentrations and an augmented rate of CHO oxidation (Horowitz and Coyle, 1993).

2.6.1 Glycaemic Index and Exercise

A paucity of research has investigated the effect of the glycaemic index of "real life" foods on exercise metabolism and performance. Exercise time to exhaustion is increased with the pre-exercise ingestion of a LGI food (lentils) compared with a HGI food (potato) (Thomas et al., 1991). The performance enhancement reported in this study was suggested by the authors to be due to the LGI food slowly releasing glucose from the gut into the blood over an extended period of time, hence, providing a continuous source of glucose in a similiar way that CHO feeding during exercise is reported to do. In this study, glucose and potato (both HGI foods), lentils and a placebo were ingested 60 min before exercise to exhaustion at 65-70% VO_{2max}. Plasma glucose and insulin concentrations were higher in the hour after ingestion in the HGI trials compared with the LGI and control trials. Furthermore, the LGI trial resulted in a lower insulin response, decreased CHO oxidation and maintained blood glucose and FFA at higher concentrations later in exercise compared with the HGI meals. The LGI meal resulted in improved exercise time to exhaustion compared with the potato trial. The authors, however, did not address the finding that the glucose,

having an identical glycaemic response to the potato, did not also result in decreased performance. The mechanism for this latter observation is unclear. Hence, further investigations which examine the effect of ingesting CHO food with different glycaemic responses on exercise metabolism and performance is required.

No studies have examined the effect of pre-exercise ingestion of "real life" CHO meals with differing glycaemic responses on metabolism and performance during exercise lasting approximately 1 hr. Many athletic events are of this duration and it is more practical for athletes to ingest food rather than nutrients as a pre-event meal. Hence, the study reported in this dissertation aimed to examine this phenomenon.

CHAPTER 3

METHODS

3.1 SUBJECTS

Eight males (22.7 ± 1.4 yrs; 180.6 ± 1.5 cm; 73.7 ± 0.9 kg; VO_{2max} = 67.9 ± 2.8 $ml.kg^{-1}.min^{-1}$, mean ± SD) were recruited for this study, having satisfied the criterion as endurance trained (VO_{2max} > 60 $ml.kg^{-1}.min^{-1}$). All subjects were fully informed of the experimental procedures and signed a letter of informed consent prior to commencement (see Appendix A). This experiment was approved by the Human Experimentation Ethics Committee of Victoria University Of Technology.

3.1.1 MEASUREMENT OF MAXIMAL OXYGEN CONSUMPTION (VO_2max)

Prior to commencing the experimental trials, each subject underwent a maximal exercise test to calculate their maximal oxygen consumption (VO_{2max}) Maximal oxygen uptake (VO_{2max}) was conducted on a friction braked bicycle ergometer (Monark Ergomedic 814E). An electrocardiograph was used to record heart rate. VO_{2max} was determined using open circuit spirometry. Expired air was directed by a Hans-Rudolf valve and plastic tubing, into a mixing chamber and through a ventilometer (Pneumoscan 830). Aliquots of the expired air were directed from the mixing chamber through an oxygen

(Applied Electrochemistry S-3A) and carbon dioxide (Applied Electrochemistry CD-3A) analyser. VO_2max and the respiratory exchange ratio (RER) was calculated using standard equations (Consolazio et al., 1963). During all VO_{2max} tests the subject had their nasal airway occluded by a nose clip and wore running shoes, socks and shorts.

The exercise protocol consisted of an incremental test to volitional exhaustion. Subjects began cycling at 100 watts (W), with the resistance increased by 50 W every two min until volitional exhaustion. The VO_{2max} was the maximal value achieved during exercise. The criteria that was used to justify the success of a VO_{2max} test was as follows; 1) a plateau in oxygen consumption with an increase in work, 2) an RER greater than 1.1, 3) volitional exhaustion. All subjects satisfied these criteria. The steady state submaximal VO_2 ($ml.kg^{-1}.min^{-1}$) values and the corresponding workloads were plotted against each other using a linear regression equation. Using these equations a workload which will elicit 70% VO_{2max} was calculated. The criterion for a successful correlation was set at $R > 0.98$ (workload vs. VO_2).

3.1.2 SUBJECT INSTRUCTIONS

During the two days preceding the trials, the subjects were instructed to refrain from strenuous physical activity and consume their normal diet. Subjects reported to the laboratory having refrained from tobacco, alcohol and caffeine for the 24 hours prior to the tests. Since the trials would be

taking place over a period of three weeks the subjects would attempt to maintain their training program with due consideration to the two days prior to experimental trials. Subjects and the experimenter kept a written record to assist replication of similar dietary and exercise behaviours for all trials.

3.2 EXERCISE TESTING PROTOCOL

Each subject performed three trials in random order with at least 7 days separating trials in which one of the test foods was ingested before exercise. Each subject arrived at the laboratory 4 hours post- prandial after ingesting a standardised breakfast. After resting for 30 min a catheter was introduced into the cephalic vein for blood sampling. After resting for 15 min the subject had 10 min to ingest the test meal. The subject then remained seated for 45 min when exercise on the bicycle ergometer commenced.

Exercise on the cycling ergometer was conducted at approximately 70% VO_{2max} for 50 min. This period of cycling was performed on a Monark friction braked cycling ergometer (Monark Ergomedic 814E). Following the submaximal cycle the subject had 60-s to switch to an air braked cycle ergometer (series A Repco) integrated into a Daos Data Acquisition System, where they completed a 15 min performance cycle. The subject was instructed to perform as much work as possible during this time to simulate the higher intensity demands characteristically reflected in some endurance sports towards the completion of the event.

3.2.1 FAMILIARISATION TRIAL

Prior to the experimental trials a familiarisation trial was undertaken in order to minimise any learning effects. It also enabled confirmation of the subject's submaximal workload and ability to maintain the submaximal workload for 50 min. All subjects were able to tolerate the required workload for 50 min. Blood sampling did not occur during this trial.

3.2.2 MEASUREMENT OF PHYSIOLOGICAL PARAMETERS DURING SUBMAXIMAL AND PERFORMANCE RIDE.

Heart rate was recorded at 15 min intervals during exercise and at the completion of the performance ride using a heart rate monitor (Sports Tester PE3000). During rest and exercise, timed (1 min) expired air collections were made to monitor VO_2 and calculate substrate oxidation. Expired air samples were collected into Douglas bags and the O_2 and CO_2 contents of these expired air samples were measured on the previously mentioned analysers and the volumes determined using a gas meter (Parkison-Cowan), calibrated against a Tissot spirometer. CHO oxidation was calculated based on VO_2 and respiratory exchange ratio (RER) measurements (Frayn, 1983). Thermal stress was minimised by oral administration of 400 ml of cool water at 15 min

intervals during exercise. The laboratory was maintained at 21° C and air was circulated with the use of an electric fan. Body weight was determined pre and post experimental trials. To safeguard against the possibility of thermal stress, core temperature was monitored through the use of rectal thermometry. A thermistor probe (YSI 401) was inserted 10cm beyond the anal sphincter. The output of the probe was monitored by a temperature unit (YSI 46TUC). Ratings of perceived exertion were obtained at 15 min intervals during exercise (Borg, 1973).

3.2.3 FOODS

The three test meals were lentils (glycaemic index :29) (Brand and Collier.1991) soaked overnight then boiled in excess water for 30 min, instant mashed potato (glycaemic index :80) (Jenkins et al.,1981) and a sweetened placebo (non-carbonated Schweppes Diet Solo). The amount of CHO ingested was equal to 1.0 g per kilogram of each subjects body mass and each test meal provided 1486 kJ, 1575 kJ and 0 kJ respectively (based on 70 kg body mass). Each test meal consisted of a volume of 600 ml. Water was added to the lentils and potato to make a volume of 600 ml and the placebo trial consisted of 600 ml of the non-carbonated diet soft drink. The standardised meal on the morning of the trials consisted of 100 g of CHO in the form of 430 ml (25% CHO, 20 mmol. l^{-1} Na^{+}) of a commercially available glucose polymer (Exceed-Ross Laboratories) and was consumed 4 hours before attending the laboratory. This standardised meal contained 1694 kJ of

energy. Forty-five min after attending the laboratory the subjects had 10 min in which to ingest the test meal. Following the ingestion of the test meal the subjects rested quietly before commencing exercise.

3.3 BLOOD SAMPLING, TREATMENT AND ANALYSIS.

Blood samples were obtained from a 20 gauge indwelling teflon catheter (Jelco) kept patent by flushing with a small amount of saline containing heparin (10 $IU.ml^{-1}$) following each sample collection. Blood sampling occurred at rest, before the test meal was ingested and at 15 min intervals until exercise commenced. During exercise, blood was sampled every 10 min for the 50 min submaximal ride and at the conclusion of the performance ride.

Ten ml of blood was collected at each sampling time. Two ml of this blood was placed in a tube containing fluoride heparin and immediately stored on ice before being spun in a centrifuge. The plasma was then transferred and stored at -80°C until analysis for glucose. Blood glucose was measured using an automated glucose analyser (YSI model 23AM). A preservative was prepared by dissolving 2.25g of ethyleneglycol-bis-(betaaminoethylether)N, N'- tetraacetic acid (EGTA) and 1.5g reduced glutathione (GSH) in 25ml of normal saline (0.9% sodium chloride w/v) and adjusted to 6-7.4 pH with 5-10 M NaoH. 30μl of this preservative were placed into tubes and stored on ice. A small amount of blood (approximately 1ml)

was transferred into these tubes containing the preservative (EGTA / GSH) and left on ice. This was later gently spun at 1500rpm for 15 min at 4° C. The supernatant was transferred to another tube and stored at -80° C until analysis of free fatty acids (FFA). Plasma FFA concentration was analysed by a modification of the enzymatic colormetric method for determination of non-esterified fatty acids (NEFAC) of Miles et al.(1983) as described in the Wako NEFAC kit (code no.279-75409). The remaining 6-8 ml of sampled blood was mixed in a tube lined with Lithium Heparin. A 500 µl aliquot of this blood was transferred into a tube containing 1ml of ice cold 3M perchloric acid (PCA) and spun in a centrifuge for 2 min at 2000 rpm. The supernatant was transferred to another tube and stored at -80° C for plasma lactate analysis at a later date. Plasma lactate was analysed in duplicate on a spectrophotometer (Shimadzu UV-120) using an enzymatic spectrophotometric technique (Lowry and Passonneau, 1972). The remaining blood was spun in a centrifuge for 2 min at 2000 rpm and the plasma transferred to tubes, stored in liquid nitrogen or at -80°C until analysis for ammonia and insulin. Analysis of ammonia was performed using flow injection analysis (FIA) (Tecator FIAstar 5020 analyser) connected to a spectrophotometer (Tecator FIAstar 5023) according to the method of Cardwell et al.(1987).

Plasma insulin concentration was determined by radioimmunoassay (RIA) as described in the Incstar Insulin I^{125} RIA kit (Cat. No.06130). Each sample was counted for one min on a gamma counter (LKB Wallac 1277

Gamma Master). All blood metabolites and hormone samples were assayed in duplicate. Chemical assays were rejected and thus repeated if standard concentrations were ± 5% of expected standard concentrations.

3.4 STATISTICAL ANALYSIS

The resting and submaximal data was analysed by a two way (time vs treatment) analysis of variance with repeated measures (ANOVA). The performance trial data was analysed using a one way analysis of variance with repeated measures (ANOVA). When ANOVA revealed a significant interaction, simple main effects and Newman-Keuls post-hoc tests were used to locate differences. An alpha level of 0.05 was used to reject the null hypothesis. A biomedical data processing computer software program was used to complete these analyses. All data are reported as means ± the standard error of the mean (SE).

CHAPTER 4

RESULTS

4.1 REST AND SUBMAXIMAL EXERCISE

4.1.1 Blood and Plasma Metabolites

Blood glucose concentrations were not different prior to ingestion of the meals. Concentrations of this metabolite were higher ($P<0.01$) in HGI compared with both LGI and CON 30 min and 15 min prior to exercise. In addition, blood glucose concentrations were higher ($P<0.01$) at these time points in LGI compared with CON. Blood glucose levels were not different when comparing the three trials at the onset of exercise. In contrast, concentrations of this metabolite were lower in HGI at 10 min ($P<0.05$) and 20 min ($P<0.01$) of exercise compared with LGI and CON. Blood glucose concentrations were not different during exercise when comparing LGI and CON. Furthermore, there were no differences in blood glucose when comparing HGI with other trials subsequent to 20 min during submaximal exercise (Fig.4.1).

Plasma insulin concentrations were not different between trials prior to the ingestion of the test meals. Concentrations of plasma insulin were higher ($P<0.01$) in HGI compared with LGI and CON at 30 min and 15 min prior to exercise and at the onset of exercise. In contrast, insulin concentrations were not different when comparing LGI and CON during rest. Insulin concentrations during exercise were lower ($P<0.01$) than at rest. During exercise plasma insulin concentrations were higher ($P<0.01$) in HGI compared with CON and higher in HGI at 10 min ($P<0.01$) and 20 min ($P<0.05$) compared with LGI. Plasma insulin concentrations were not different during exercise when comparing LGI and CON with the exception of 30 min which was higher ($P<0.05$) in LGI compared with CON (Fig.4.2).

Plasma FFA concentrations were not different prior to ingestion between the three trials and were higher ($P<0.01$) than plasma FFA levels at the onset of and during exercise. At the commencement of exercise plasma FFA concentrations in both HGI and LGI were lower ($P<0.01$) compared with CON. During exercise, concentrations of this metabolite were higher in CON compared with HGI and LGI at 10 min ($P<0.01$) and 20 min ($P<0.05$). Furthermore, plasma FFA concentrations were higher in CON compared with HGI ($P<0.01$) and LGI ($P<0.05$) for the remainder of exercise with the exception of 40 min. Of note, plasma FFA were higher ($P<0.05$) in LGI compared with HGI at 20 min and 50 min (Fig.4.2).

There were no differences between any of the three trials for plasma lactate or plasma NH_3 concentrations before ingestion of the test meals, during rest and exercise. There was an increase ($P<0.01$) in plasma lactate levels during exercise compared with rest. Plasma NH_3 levels were higher ($P<0.01$) during exercise compared with rest. Additionally, plasma NH_3 concentrations were higher ($P<0.01$) at 40 min and 50 min compared with 10 min during exercise (Fig.4.3).

4.1.2 Physiological and Ventilatory data

There were no differences in relative work intensity between the trials during exercise (66.65 ± 8.1% of VO_{2max}, mean ± S.D). There were no differences in oxygen consumption (VO_2) or heart rate between any of the three trials during rest or exercise. There was an increase ($P<0.01$) in mean VO_2 during exercise compared with rest (Table 4.1). A main treatment effect ($P<0.01$) was observed for both RER (Table 4.1) and rate of CHO oxidation (Fig 1). Post hoc analyses revealed that values for these parameters were higher ($P<0.01$) in HGI compared with the other trials. Core temperature increased ($P<0.01$) with time during exercise. Furthermore, ratings of perceived exertion increased ($P<0.01$) with time. There were no differences between any of the three trials with respect to these two parameters. (Table 4.1). There were no differences in the change in body weight between any of the trials at the conclusion of exercise

4.2 PERFORMANCE TRIAL

4.2.1 Blood and Plasma Metabolites

At the conclusion of the performance trial, plasma lactate and NH_3 levels were higher ($P<0.01$) and plasma FFA lower ($P<0.01$) than concentrations at the end of submaximal exercise. There was no change in concentrations for blood glucose and plasma insulin during this time. In addition, there were no differences between any of the three food trials with respect to these metabolites (Table 4.2).

4.2.2 Physiological and Ventilatory data

There was no difference in total work produced during the performance ride in the three trials (Fig. 4.4).

Mean V_{O2}, CHO oxidation, RER, heart rate and core temperature were higher ($P<0.01$), as was the rate of perceived exertion ($P<0.05$) during the performance trial compared with submaximal exercise. During the performance trial there was an increase in RER and CHO oxidation ($P<0.05$) and in heart rate ($P<0.01$), however, there was no differences between trials with respect to these parameters at any time (Table 4.1).

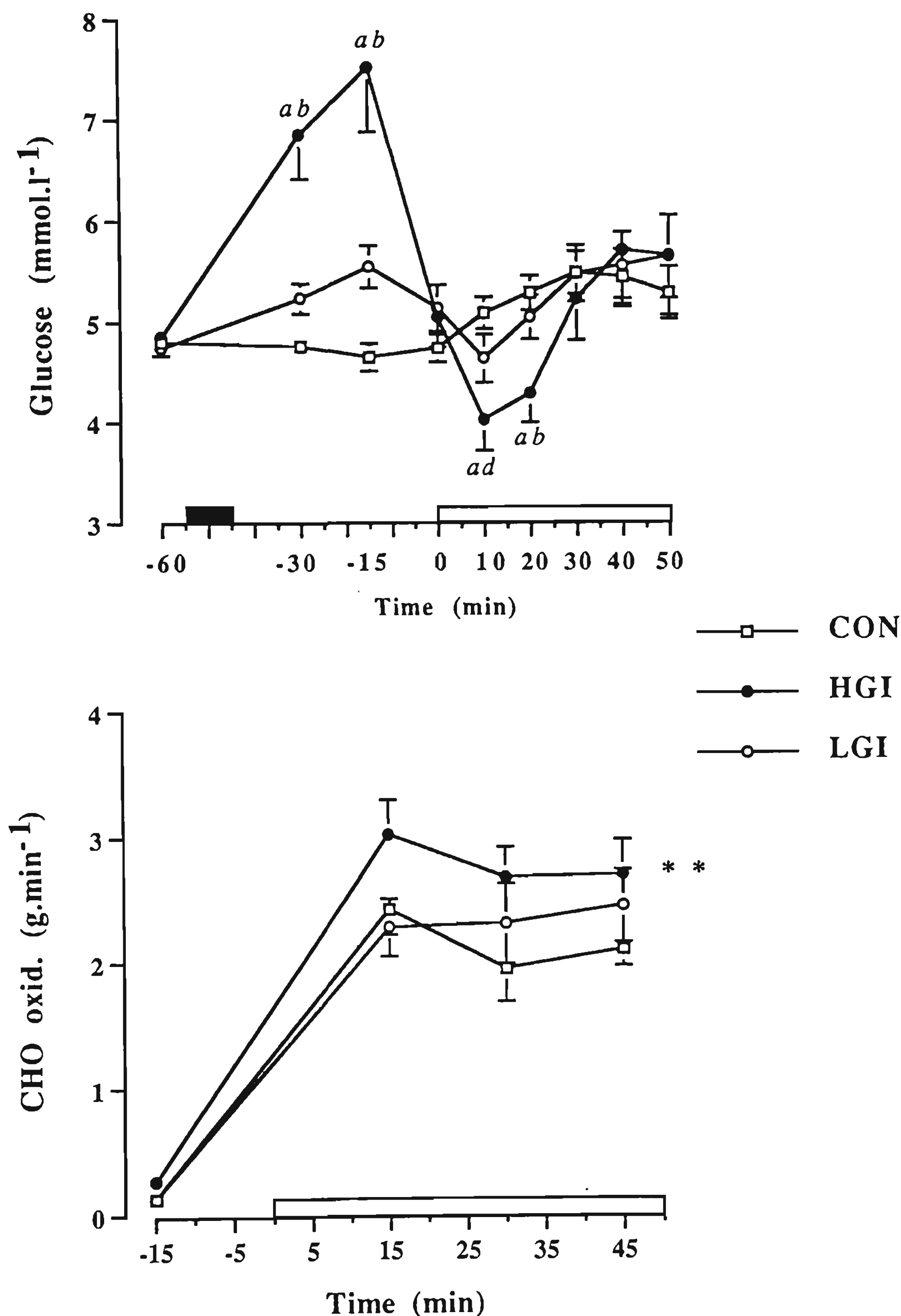

Fig. 4.1 Plasma glucose concentration and estimated carbohydrate oxidation during rest and submaximal exercise in CON, HGI and LGI. ■ denotes feeding, ▯ denotes exercise. Values are means ± SE (n=8). *a* indicates difference (P<0.01) from CON, *b* indicated difference (P<0.01) from LGI, *d* indicated difference (P<0.05) from LGI. ** indicates main treatment effect (P<0.01) compared with LGI & CON.

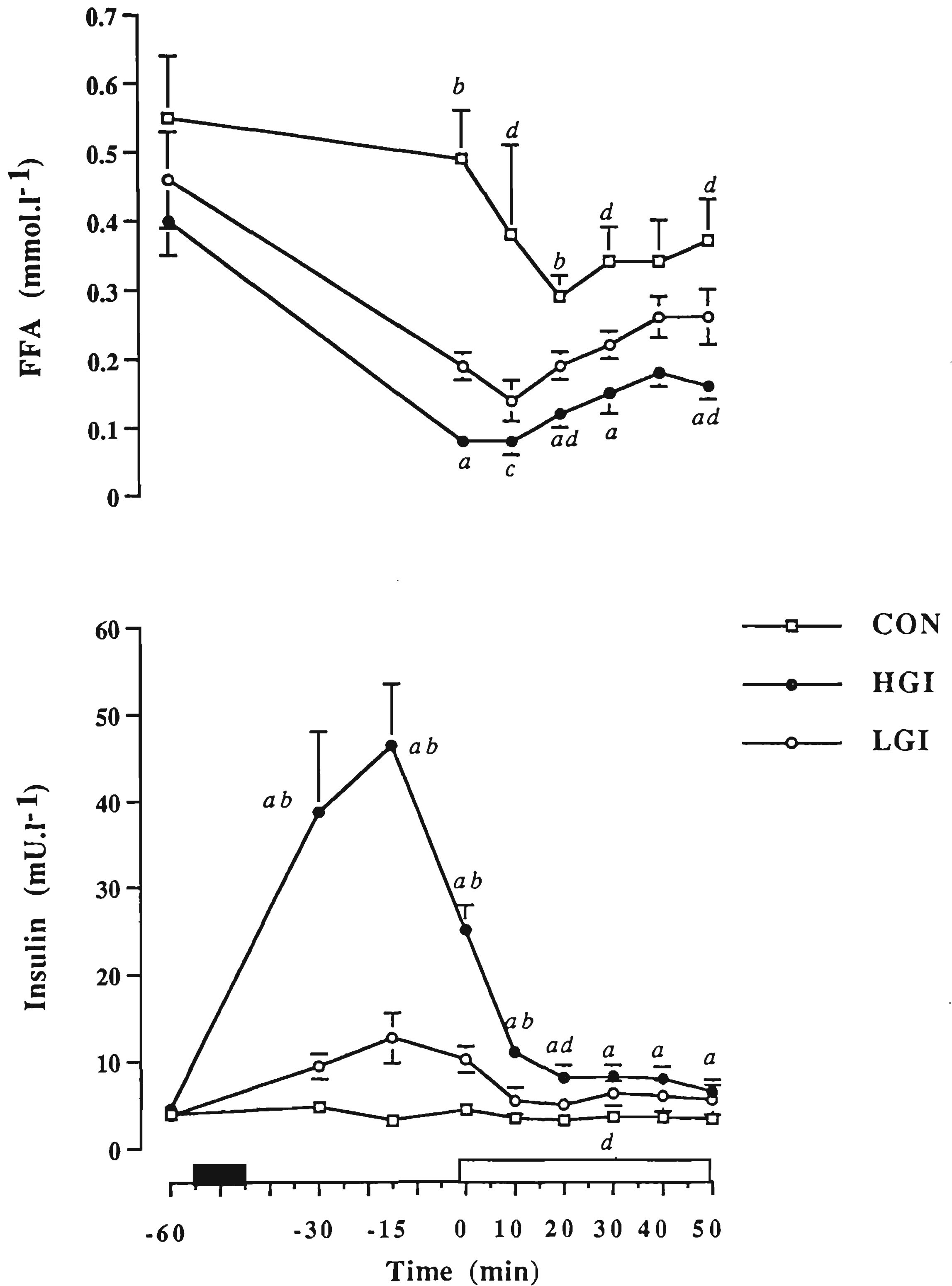

Fig. 4.2 Plasma FFA and Insulin concentrations during rest and submaximal exercise in CON, HGI and LGI. ■ denotes feeding, □ denotes exercise. Values are means ± SE (n=7). *a* indicates difference (P<0.01) from CON, *b* indicated difference (P<0.01) from LGI, *c* indicates difference (P<0.05) from CON, *d* indicated difference (P<0.05) from LGI.

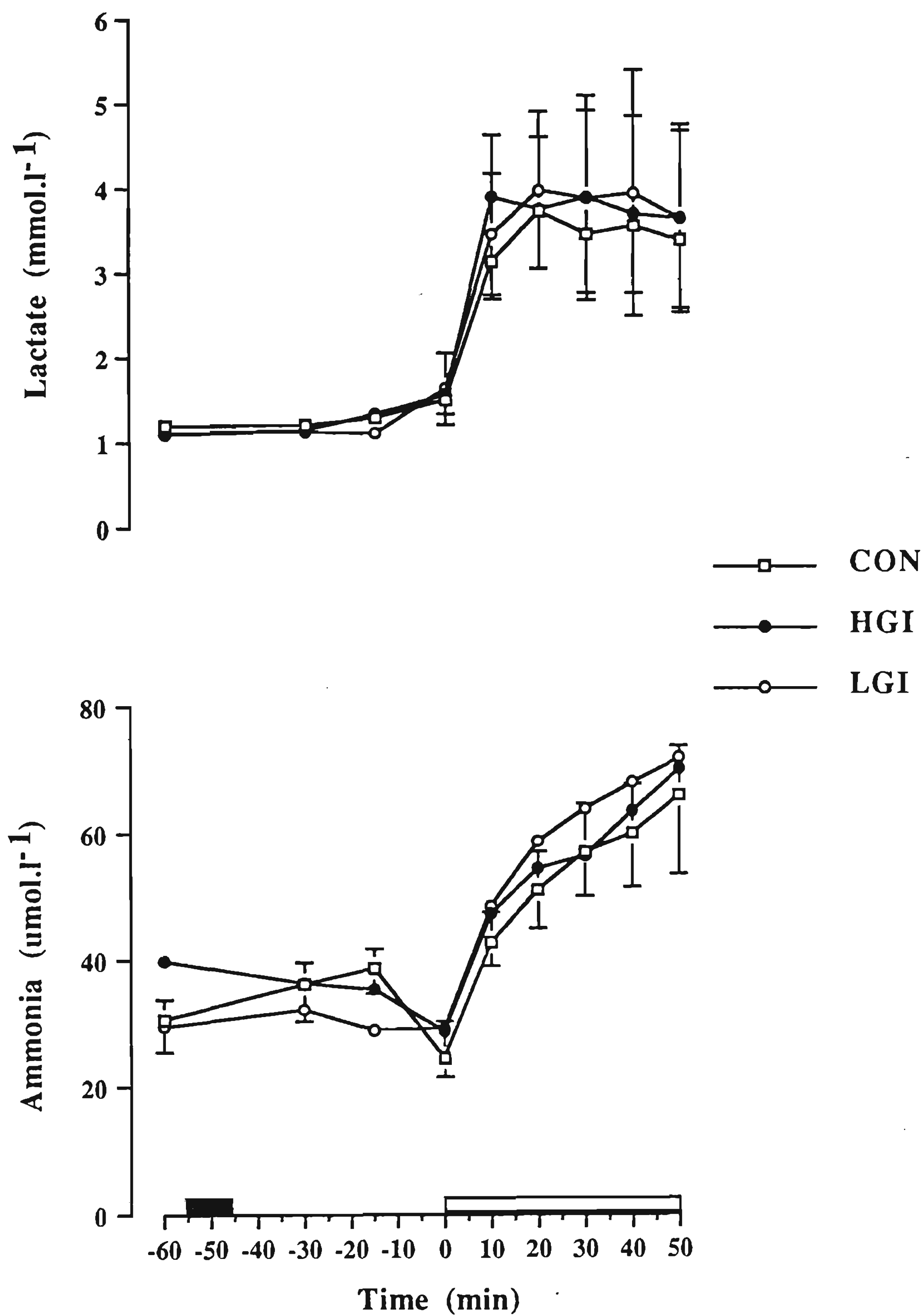

Fig. 4.3 Plasma Lactate and NH_3 concentrations during rest and submaximal exercise in CON, HGI, and LGI. ■ denotes feeding, ▯ denotes exercise. Values are means ± SE (n=8).

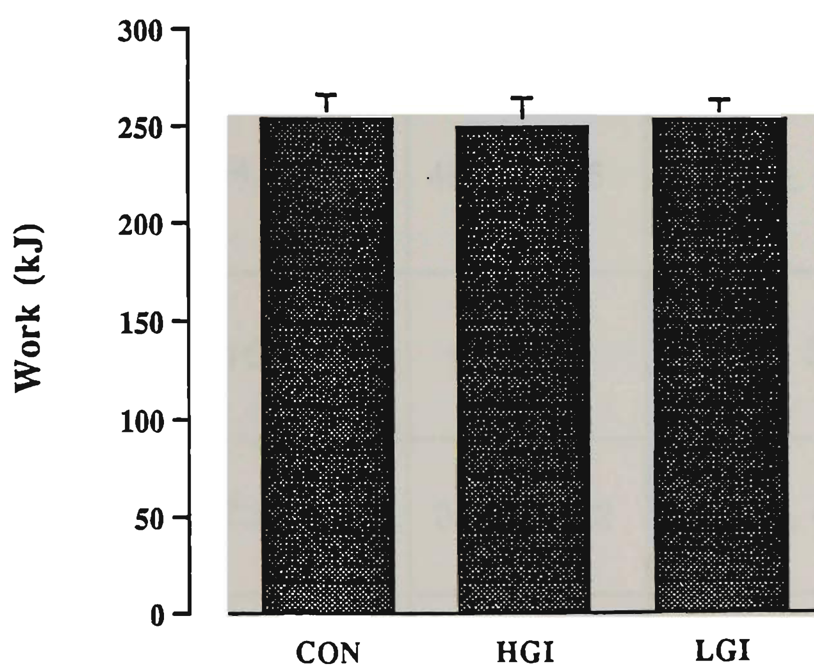

Fig. 4.4 Work output during the performance trial (last 15 min of exercise) in CON, HGI, and LGI. Values are means SE (n=8).

	SUBMAXIMAL			PERFORMANCE		
	CON	HGI	LGI	CON	HGI	LGI
RER	0.85 ± 0.01	0.90 ± 0.01**	0.86 ± 0.01	0.93 ± 0.01	0.94 ± 0.02	0.92 ± 0.01
VO_2 (ml.kg^{-1}.min^{-1})	45.5 ± 2.2	44.5 ± 2.7	45.8 ± 2.5	57.8 ± 2.0	57.6 ± 2.8	56.1 ± 2.8
HR (b.min^{-1})	150 ± 5	152 ± 7	149 ± 5	175 ± 3	173 ± 4	175 ± 4
T_{rec} (°C)	37.6 ± 0.3	37.5 ± 0.3	37.6 ± 0.2	38.3 ± 0.2	38.3 ± 0.2	38.4 ± 0.2
PE	13.5 ± 0.8	12.8 ± 0.6	12.6 ± 0.8	17.8 ± 0.7	17.4 ± 0.5	17.0 ± 0.8

Table 4.1 Physiological and Ventilatory data during rest, submaximal exercise (15, 30, 45 min) and performance trial (5, 10 min) in CON, HGI, and LGI. Values are means ± SE (n=8).

** indicates main treatment effect (P<0.01) compared with LGI & CON.

PERFORMANCE			
	CON	HGI	LGI
Glu (mmol.l^{-1})	5.8 ± 0.3	5.3 ± 0.5	6.3 ± 0.4
La (mmol.l^{-1})	9.3 ± 1.0	9.3 ±1.1	9.2 ± 1.1
FFA (mmol.l^{-1})	0.22 ± 0.02	0.17 ± 0.03	0.17 ± 0.03
NH_3 (μmol.l^{-1})	126.5 ±19.5	124.0 ±16.7	122.2 ±18.5
Ins (mU.ml^{-1})	3.0 ± 0.8	5.6 ± 1.9	5.0 ± 1.3

Table 4.2 Blood glucose, plasma NH_3, plasma lactate (n=8), plasma insulin, plasma FFA concentrations (n=7) at the end of the performance trial. Values are means ± S.E.

CHAPTER 5

5.1 DISCUSSION

The results from this study demonstrated that pre-exercise ingestion of a high glycaemic index food resulted in a hyperglycaemic response prior to exercise followed by an insulin-mediated hypoglycaemia at the onset of exercise (Fig.4.1). In addition, the elevated insulin during HGI resulted in an attenuation in circulating FFA (Fig.4.2) and a higher rate of CHO oxidation (Fig.4.1). Despite these metabolic changes, performance during the all-out cycle was unaffected.

Previous investigations have demonstrated that "complex" carbohydrates differing in chemical structure can have very different glycaemic responses (Crapo et al., 1977; Horowitz and Coyle, 1993; Jenkins et al., 1984). Data from this study support these earlier findings. Plasma glucose and insulin concentrations were higher in HGI compared with LGI and CON following ingestion of the meal. During exercise plasma glucose in HGI dropped rapidly during the first 10 min of submaximal exercise and was lower compared with both LGI and CON during this time. Furthermore, plasma glucose was lower throughout almost half of the submaximal period in HGI compared with LGI and CON. The rapid decline in blood glucose following ingestion of the HGI meal is in support of previous findings (Bonen

et al., 1981; Chryssanthopoulos et al., 1994; Decombaz et al., 1985; Foster et al., 1979; Hargreaves et al., 1985; Hargreaves et al., 1987; Koivisto et al., 1981; Seifert et al., 1994; Thomas et al., 1991). The functional significance of this decline has not, however, been well investigated. It has been suggested (Costill et al., 1977; Hargreaves et al., 1987) that this decline may result in an enhanced rate of muscle glycogenolysis via a reduced glucose supply to the active muscle. Insulin facilitates the cellular uptake of glucose (Fushiki et al., 1989; Holloszy and Narahara, 1965), reduces hepatic glucose output (Felig and Warren, 1979; Galbo, 1983) and thus may account for the rapid decline in blood glucose widely observed in similar studies (Chryssanthopoulos et al., 1994; Decombaz et al., 1985; Hargreaves et al., 1987; Koivisto et al., 1981; Seifert et al., 1994).

Costill et al. (1977) and Hargreaves et al. (1985) observed an enhanced rate of glycogen utilization following glucose ingestion before exercise compared with a control when blood glucose concentrations declined early in exercise to values lower than 3.5 $mmol.l^{-1}$. In contrast, during exercise where blood glucose has not fallen to concentrations below 3.9 $mmol.l^{-1}$, no differences were observed in rates of muscle glycogen utilization (Decombaz et al., 1985; Devlin et al., 1986; Koivisto et al., 1985; Levine et al., 1983).

Despite blood glucose concentrations during exercise in HGI falling to only 4.03 $mmol.l^{-1}$, a higher RER and carbohydrate oxidation was observed

during this trial, confirming earlier results (Costill et al., 1977; Coyle et al., 1985; Horowitz and Coyle, 1993; Thomas et al., 1991). Data from this study may indicate that muscle glycogen utilization was augmented during exercise in this trial although muscle samples were not collected to confirm this. Although muscle glucose uptake is increased when plasma glucose is elevated by carbohydrate feeding, RER and carbohydrate oxidation are unchanged (McConell et al., 1994). These data indicate that the increase in muscle glucose uptake is relatively small and does not influence substrate utilization to a significant extent.

As previously discussed , muscle glucose uptake is stimulated by elevations in insulin, but may be inhibited by high muscle glycogen concentration (Hargreaves et al., 1995). Immediately prior to and during the first 10 min of exercise insulin was elevated and blood glucose concentration declined rapidly, suggesting that muscle glucose uptake was augmented in HGI. Subsequent to the first 10 min of submaximal exercise, however, blood glucose concentration was depressed in HGI. In contrast, the main treatment effect for CHO oxidation (Fig 4.1) suggests that the contribution to energy demand from this substrate remained elevated. It is possible that the increase in CHO oxidation in HGI may have been due, in part, to an increase in the utilization of blood borne glucose during the initial period of exercise. Paradoxically, the higher rate of CHO oxidation in this trial may be a result of increased muscle glycogen utilization subsequent to the first 15 min. Further

research examining glucose kinetics and muscle glycogenolysis during exercise preceded by CHO ingestion warrants further investigation.

Of note, subjects in the present study had a higher VO_{2max} compared with those studies which report no difference in muscle glycogenolysis following pre-exercise carbohydrate feeding (Decombaz et al., 1985; Devlin et al., 1986; Koivisto et al., 1985; Levine et al., 1983). Since insulin sensitivity is increased with aerobic training (Heath et al., 1983) it is possible that the effect of elevated insulin had a greater inhibitory effect on lipolysis in the present study compared with those previously reported.

Insulin reduces the lipolytic rate and thus limits the availability of FFA in the circulation (Gleeson et al., 1986; Wolfe et al., 1986). A reduced FFA concentration during exercise enhances muscle glycogenolysis (Bergstrom et al., 1969). Conversely, elevated FFA concentrations spares muscle glycogen (Costill et al., 1977; Hickson et al., 1977; Stankiewicz-Choroszuchna and Gorski, 1978). Plasma insulin was higher in HGI during the first 20 min of exercise compared with LGI and was higher compared with CON during the entire submaximal period. FFA concentrations were lower in HGI compared with CON during exercise and lower compared with LGI at the start and end of submaximal exercise, suggesting a decreased availability of this substrate.

The present results confirm earlier findings (Horowitz and Coyle, 1993; Thomas et al., 1991) which observed a decrease in plasma FFA during

exercise after the ingestion of a high or moderate glycaemic index meal. In contrast, plasma insulin concentrations were far more stable following the ingestion of the LGI meal and were not different from CON, with the exception of 30 min which was unexpectedly higher. Insulin has a persistent effect on adipocyte hydrolysis (Solomon and Duckworth, 1976), even after concentrations return to basal levels (Coyle et al., 1985). Despite there being no significant difference in insulin concentrations when comparing LGI with CON, the mean concentrations were approximately 2-fold higher in LGI immediately prior to exercise (Fig.4.2). This may have been responsible for lower FFA values in LGI compared with CON. The large increase in plasma insulin concentration associated with HGI may be functionally more important during exercise of a longer duration, than that employed in the present study, since it has lasting effects on adipocytes.

Although possible, it is unlikely that the increase in carbohydrate oxidation observed in HGI was related to an elevated muscle glycogen concentration prior to exercise. Coyle et al. (1985) observed an increase in muscle glycogen storage prior to, and carbohydrate oxidation during, exercise following a carbohydrate meal 4 hours before exercise. It has been well documented (Chesley et al., 1995; Gollnick et al., 1981; Hargreaves et al., 1995; Sherman et al., 1981) that elevated glycogen concentrations prior to exercise results in enhanced glycogenolysis during exercise. This is likely to occur since glycogen can bind to phosphorylase to increase its activity (Hespel and Richter, 1992; Johnson, 1992). The ingestion of CHO, in the

present study took place 45 min prior to exercise. Since the maximal rate of glycogen resynthesis is 5-6 $mmol.kg^{-1}.h^{-1}$ wet weight. (Blom et al., 1987; Reed et al., 1989) the amount of glycogen which may have been stored during the period between ingestion and exercise is unlikely to affect rates of glycogen utilization. In addition, since subjects were fed CHO 6 hours before exercise and were well rested, it is likely that the glycogen stores within skeletal muscle were high prior to ingestion of the test meals. Hence, the ingestion of CHO prior to exercise would have less of an influence on muscle glycogen storage.

Plasma lactate concentrations were not different when comparing the trials suggesting that the energy contribution from anaerobic metabolism was similar (Fig.4.3). Likewise, there were no differences in plasma NH_3 concentration in the three trials during exercise (Fig 4.3) which may suggest a similar degree of protein catabolism. No previous studies have examined the effect of pre-exercise carbohydrate feeding on plasma NH_3 concentration, however, when carbohydrate is fed during exercise plasma NH_3 concentration is attenuated during exercise (Febbraio et al., 1994; Snow et al., 1995). Of note, however, concentrations of this metabolite were only affected by carbohydrate feeding subsequent to the first hour of exercise. Further research examining the effect of pre-exercise carbohydrate feeding and NH_3 metabolism during exercise of a longer duration is warranted.

There was an increase in NH_3 production in all trials during the performance ride which is in agreement with other studies incorporating high intensity work (Babij et al., 1983; Katz et al., 1986). There was an observed increase in mean exercise intensity during the performance trial compared with the submaximal period. It is likely that the majority of the NH_3 produced during the performance ride originated from AMP deamination (Katz et al., 1986; Meyer and Terjung, 1980). The decrease in plasma FFA concentrations during the performance trial (Table 4.2) compared with those in the submaximal cycle is likely to be the result of an increase in anaerobic glycolysis reflected in the higher plasma lactate concentrations observed during this period (Table 4.2). Lactate has been reported to increase the re-esterification of FFA and reduce lipolysis (Issekutz and Miller, 1962).

Despite an increase in CHO oxidation during submaximal exercise in HGI compared with CON and LGI, no differences in performance were observed (Fig.4.4). In addition, no differences in the rate of perceived exertion was observed during this period (Table 4.1). The similar exercise performance reported in this study are in support of previous observations (Chryssanthopoulos et al., 1994; Decombaz et al., 1985; Devlin et al., 1986; Hargreaves et al., 1987; McMurray et al., 1983; Sharp et al., 1993). These previous studies did not observe an effect of pre-exercise carbohydrate ingestion in the hour before exercise on performance, despite alterations in metabolism as a result of the ingested carbohydrate. The results from this study demonstrate that carbohydrate supplementation before exercise of this

nature is neither beneficial nor detrimental to exercise performance compared with placebo ingestion. There are two possibilities which may explain such a phenomenon. Firstly, whilst the rate of glycogenolysis was potentially elevated in HGI compared with LGI and CON, muscle glycogen at the onset of the performance cycle was likely to be of sufficient concentration in all trials to have no effect on performance. Power output during high intensity exercise is unaffected by pre-exercise dietary manipulation which results in different pre-exercise muscle glycogen concentrations (Hargreaves et al., 1994). These authors postulated that this was due to the glycogen being of significant concentration in the low glycogen state to have no influence on performance. Secondly, it is possible that the standardized meal ingested 6 hours prior to exercise may have lessened any likely effect of the pre-exercise carbohydrate ingestion.

Several studies (Gleeson et al., 1986; Neuffer et al., 1987; MacLaren et al., 1994; Okano et al., 1988; Sherman et al., 1991; Snyder et al., 1983; Thomas et al., 1991) have reported an improvement in exercise performance following carbohydrate ingestion in the hour before exercise. The discrepancies between these results and those of the present study may be explained, in part, by the differences in experimental methodologies, the fitness level of the subjects recruited to participate, and/or the foods chosen to ingest before exercise. As previously discussed, if the carbohydrate is ingested just prior to exercise (Neuffer et al., 1987; MacLaren et al., 1994; Snyder et al., 1983) in contrast to ingestion 30-60 min before exercise

(Gleeson et al., 1986) the alterations in metabolism, similar to those observed in this study, are attenuated. Only Gleeson et al. (1986) have observed an improvement in endurance exercise following the ingestion of a HGI food (glucose) compared with the ingestion of a placebo 45 min before exercise. These authors observed blood glucose to decline to not less than 4 mmol.l^{-1} in any subject in spite of hyperinsulinaemia in the pre-exercise period. Montain et al. (1991) hypothesized that if the exercise intensity was "metabolically stressful", hepatic glucose production will match glucose utilization, preventing, therefore, the rapid decline in blood glucose during the initial stages of exercise. Feed-forward control of hepatic glucose production has been suggested to be activated by higher motor centre activity-stimulated neuroendocrine activity, particularly during exercise at higher workloads (Kjaer et al., 1986; Kjaer et al., 1987; Sonne and Galbo, 1985). Kjaer et al. (1987) reported that a greater voluntary effort was necessary to produce a certain work output during neuromuscular blockade compared with a control. The circulating catecholamines were higher with neuromuscular blockade which acted to enhance hepatic glucose output. It is likely that the subjects in the study of Gleeson et al. (1986) (VO_{2max} = 46.7 + 2.7 ml.kg^{-1}.min^{-1}) found the workload (73% VO_{2max}) stressful, as indicated by the relatively high RER (0.96) observed in the absence of pre-exercise carbohydrate ingestion. This may have inhibited the hypoglycaemia observed in other studies, by a similar feed-forward mechanism to that suggested by Kjaer et al. (1987). In the present study, the fitness levels of the subjects were much higher (67.9 + 2.8 ml.kg^{-1}.min^{-1}) and the intensity of exercise was lower (67% VO_{2max}). The RER

during the control trial (0.87) indicates that exercise may not have been stressful enough to prevent a rapid decline in blood glucose. It is possible that this phenomenon coupled with other differences in methodology may account for the discrepancy in the results.

In a recent study by Thomas et al. (1991) an increase in exercise time to exhaustion following the pre-exercise ingestion of LGI food (lentils) compared with ingestion of a HGI food (potato) was observed. Of note, however, no difference was observed in exercise performance when the LGI food was compared with ingestion of a HGI food (glucose) administered in liquid form, a finding the authors failed to address. Perhaps the alterations in metabolism associated with the ingestion of LGI foods, observed by Thomas et al. (1991) and in this study, are of greater significance during exercise of a longer duration. Thomas et al. (1991) suggested that the ingestion of a LGI may provide a sustained release of glucose into the blood stream and thus aid performance in a way that carbohydrate feeding during exercise has been reported to do. As previously discussed, the maintenance of blood glucose by carbohydrate feeding during endurance exercise prevents hypoglycaemia and increases endurance capacity.

In conclusion, the data from this study indicate that pre-exercise ingestion of a HGI food results in hyperglycaemia and subsequent hyperinsulinaemia prior to exercise. As a consequence, an insulin mediated hypoglycaemia and reduced FFA availability was accompanied by an

increased CHO oxidation in this trial. In contrast, ingestion of a LGI food resulted in a lower glycaemic response, reduced insulin secretion, an attenuated fall in FFA and subsequently, a lower rate of CHO oxidation, such that the rate of CHO oxidation was not different compared with CON. Despite these changes work output during a 15 min performance trial was not different when comparing the three trials. Further research examining the effect of pre-exercise ingestion of CHO with differing glycaemic indices during prolonged exercise of greater than 60 min duration may shed further light on the relationship between CHO oxidation and exercise performance.

REFERENCES

Ahlborg, G .and P.Felig. Substrate utilization during prolonged exercise preceded by ingestion of glucose Am. J. Physiol. 2(3): E188-E194, 1977.

Ahlborg, G. and P.Felig. Influence of glucose ingestion on fuel-hormone response during prolonged exercise. J. Appl.Physiol. 41: 683-688, 1976.

Ahlborg, G., P.Felig, L.Hagenfeldt, R.Hendler, and J.Wahren. Substrate turnover during prolonged exercise in man: splanchnic and leg metabolism of glucose, free fatty acids and amino acids. J. Clin.Invest. 53: 1080-1090, 1974.

Ahlborg, G., and O.Bjorkman. Splanchnic and muscle fructose metabolism during and after exercise. J. Appl.Physiol. 69: 1244-1251, 1990.

Alberici, J.C., P.A. Farrell, P.M. Kris-Etherton, and C.A. Shively. Effects of preexercise candy bar ingestion on glycemic response, substrate utilisation, and performance. International Journal Of Sports Nutrition 3: 323-333, 1993.

Allen, D.O., and E.A. Gardner. Antilipolytic action of insulin in the perifused fat cell system. Biochem. Pharmacol. 29: 617-621, 1980.

Arnall, D.A., J.C. Marker, R.K. Conlee, And W.W. Winder. Effect of infusing epinephrine on liver and muscle glycogenolysis during exercise in rats. Am. J. Physiol. E641-E649, 1986.

Askew, E.W., J.R. Claybaugh, G.M. Hashiro, W.S. Stokes, and S.A.Cucinell. Carbohydrate supplementation during exercise at high altitude. Abstract 4758 Federation Proceedings 45: 972, 1986.

Baldwin, K.M., Reitman,J.S., R.L. Terjung, W.W. Winder, and J.O. Holloszy. Substrate depletion in different types of muscle and in liver during prolonged running. Am.J.Phsiol. 225: 1045-1050, 1973.

Behall, K.M., D.J. Scholfield, and J. Canary. Effect of starch structure on glucose and insulin responses in adults. American Journal Of Clinical Nutrition 47: 428-432, 1988.

Berger, M., S. Hagg, and N.B.Ruderman. Glucose metabolism in perfused skeletal muscle: interaction of insulin and exercise on glucose uptake. Biochemical Journal 146: 231-238, 1975.

Bergstrom, J. and E. Hultman. A study of the glycogen metabolism during exercise in man. Scandinavian Journal Of Clinical And Laboratory Investigation 19: 218-228, 1967.

Bergstrom, J., L. Hermansen, E. Hultman, And B. SALTIN. Diet, muscle glycogen, and physical performance. Acta Physiol. Scand. 71: 140-150, 1967.

Bergstrom, J., E. Hultman, L. Jorfeldt, B. Pernow, and J. Wahren. Effect of nicotinic acid on physical working capacity and on metabolism of muscle glycogen in man. J. Appl. Physiol. 26(2): 170-176, 1969.

Blom, P.C.S., A.T. Hostmark, O. Vaage, K.R. Kardel, and S. Maehlum. Effect of different post-exercise sugar diets on the rate of muscle

glycogen synthesis. Med. Sci. Sports Exercise 19: 491-496, 1987.

Bonen, A., S.A. Malcolm, R.D. Kilgour, K.P. Macintyre, and A.N. Belcastro. Glucose ingestion before and during intense exercise. J. Appl. Physiol. 50(4): 766-771, 1981.

Booth, F.W. and P.A. Watson. Control of adaptations in protein levels in response to exercise. Fed. Proc. 44: 2293-2300, 1985.

Borensztajn, J., M.S. Rone, S.P. Babirak, J.A. Mcgarr, and L.B. Oscai. Effect of exercise on lipoprotein lipase activity in rat heart and skeletal muscle. Am.J.Phsiol. 229: 394-397, 1975.

Borensztajn, J., P.Keig, And A.H.Rubenstein. The role of glucagon in the regulation of myocardial lipoprotein lipase activity. Biochemical and Biophysical Research Communications 53: 603-608, 1973.

Borensztajn, J. Lipoprotein lipase. In Scanu, A.M., R.W. Wissler, and G.S.Getz (Eds). The biochemistry of Atherolsclerosis. New York: Marcek Dekker, Inc, 1979.

Borg, G. Perceived exertion: a note on "history" and methods. Med. Sci. Sports Exerc. 5(2): 90-93, 1973.

Brand, J. and G. Collier. A tool for diabetes: New directions Clinical Update 2, 1991.

Brouns, F., N.J. Rehrer, W.H.M. Saris, E. Beckers, P. Menheere, and F.Ten Hoor. Effect of CHO intake during warming up on the regulation of blood glucose during exercise. International Journal Of Sports Medicine 10: S68-S75, 1989.

Burke, L., G.R. Collier, S.K. Beasley, P.G. Davis, P.A. Fricker, P. Heeley, and M. Hargreaves. Effect of coingestion of fat and protein with carbohydrate feedings on muscle glycogen storage. J. Appl. Physiol. (In Press), 1995.

Cardwell, T.J., M.F. Carey, R.W. Cattrall, I.C. Hamilton, P.J. Iles, W.R. Payne, and R.J. Snow. The determination of total ammonia/ammonium in human plasma by flow injection analysis. Proc. 9th Australian Symposium on Analytical Chemistry. 774-777, 1987.

Carlson, L.A., S.O. Liljedahl, and C. Wirsen. Blood and tissue changes in the dog during and after excessive free fatty acid mobilization. A biochemical and morphological study. Acta Med. Scand.178: 81-102, 1965.

Carlson, L.A., L.G. Ekelund, and S.O. Froberg. Concentration of triglycerides, phospholipds and glycogen in skeletal muscle and of free fatty acids and y-hydroxybutyric acid in blood in man in response to exercise. European Journal Clinical Investigation 1: 248-254, 1971.

Carlson, K.I., J.C.Marker, D.A. Arnall, M.L. Terry, H.T. Yang, L.G. Lindsay, M.E. Bracken, and W.W. Winder. Epinephrine is unessential for stimulation of liver glycogenolysis during exercise. J. Appl. Physiol. 58: 544-548, 1985.

Chasiotis, D., K.Sahlin, and E. Hultman. Regulation of glycogenolysis in human muscle at rest and during exercise. J. Appl. Physiol. 53(3): 708-715, 1982.

Chasiotis, D., K.Sahlin, and E.Hultman. Regulation of glycogenolysis in human muscle in response to epinephrine infusion. J. Appl. Physiol. 54(1): 45-50, 1983.

Chasiotis, D. Role of cyclic AMP and inorganic phosphate in the regulation of muscle glycogenolysis during exercise. Med. Sci. Sports Exerc. 20(6): 545-550, 1988.

Cheetham, M.E., L.H. Boobis, S. Brooks, and C. Williams. Human muscle metabolism during sprint running. J. Appl. physiol. 61(1): 54-60, 1986.

Chen, M. and R.L. Whistler. Metabolism of D-Fructose. Advances In Carbohydrate Chemistry And Biochemistry 34: 265-343, 1977.

Chesley, A., D.J. Dyck, and L.L. Spriet. High physiological levels of epinephrine do not enhance muscle glycogenolysis during tetanic stimulation. J. Appl. Physiol. 77(2): 956-962, 1994.

Chesley, A., E. Hultman, and L.L. Spriet. Effects of epinephrine infusion on muscle glycogenolysis during intense aerobic exercise. Am. J. Physiol. E127-E134, 1995.

Christensen, E.H. and O. Hansen. Arbeitsfahigkeit und Ernahrung. Skand. Arch. Phsyiol. 81: 160-171, 1939a.

Chryssanthopoulos, C., L.C.M. Hennessy, and C. Williams. The influence of pre-exercise glucose ingestion on endurance running capacity. Br.J. Sports Med. 28(2): 105-109, 1994.

Collier, G. and K. O'dea. The effect of coingestion of fat on the glucose, insulin, and gastric inhibitory polypeptide responses to

carbohydrate and protein. American Journal Of Clinical Nutrition 37: 941-944, 1983.

Coggan, A.R. and E.F. Coyle. Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. J. Appl. Physiol. 63: 2388-2395, 1987.

Coggan, A.R. and E.F. Coyle. Effect of carbohydrate feedings during high-intensity exercise. J. Appl.Physiol. 65: 1703-1709, 1988.

Coggan, A.R. and E.F. Coyle. Metabolism and performance following carbohydrate ingestion late in exercise. Med. Sci. Sports Exerc. 21: 59-65, 1989.

Coggan, A.R., R.J. Spina, W.M. Kohrt, D.M. Bier, And J.O. Holloszy. Plasma glucose kinetics in a well-trained cyclist fed glucose throughout exercise. Int.J.Sports Nutr. 1: 279-288,1991.

Cole, K.J., P.W. Grandjean, R.J. Sobszak, and J.B. Mitchell. Effect of carbohydrate composition on fluid balance, gastric emptying, and exercise performance. International Journal Of Sports Nutrition. 3: 408-417, 1993.

Consolazio, C.G., R.E. Johnson, and L.J. Pecora. Physiological measurements of metabolic functions in man. New York, McGraw-Hill, 1963.

Constable, S.H., R.J. Favier, and J.O. Holloszy. Exercise and glycogen depletion: effects on ability to activate muscle phosphorylase. J. Appl. Physiol. 60: 518-1523, 1986.

Constable, S.H., R.J. Favier, G.D. Cartee, D.A. Young, and J.O. Holloszy. Muscle glucose transport: interactions of in vitro contractions, insulin, and exercise. J.. Appl. Physiol. 64(6): 2329-2332, 1988.

Costill, D.L. and B. Saltin. Factors limiting gastric emptying during rest and exercise. J. App. Physiol. 37(5): 679-683, 1974.

Costill, D.L., E. Coyle, G. Dalsky, W. Evans, W. Fink, and D. Hoopes. Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. J. Appl. Physiol. 43(4): 695-699, 1977.

Coyle, E.F., D.L.Costill, W.J.Fink, and D.G.Hoopes. Gastric emptying rates for selected athletic drinks. Research Quarterly 49(2): 119-124, 1978.

Coyle, E. Carbohydrate feeding during prolonged strenuous exercise can delay fatigue J. Appl. Physiol. 55: 230-235, 1983.

Coyle, E.F. and A.R. Coggan. Effectiveness of carbohydrate feeding in delaying fatigue during prolonged exercise. Sports Med. 1: 446-458, 1984.

Coyle, E., A.R. Coggan, M.K. Hemmert, R.C. Lowe, and T.J. Walters. Substrate usage during prolonged exercise following a preexercise meal. J. Appl. Physiol. 59(2): 429-433, 1985.

Coyle, E., A.R. Coggan, M.K. Hemmert, and J.L. Ivy. Muscle glycogen utilization during prolonged strenuous exercise when fed CHO.J. .Appl. Physiol. 61: 165-172, 1986.

Craig, B.W. The influence of fructose feeding on physical performance. American Journal Of Clinical Nutrition 58(Supp): 815S-819S, 1993.

Crapo, P.A., G. Reaven, and J. Olefsky. Plasma glucose and insulin responses to orally administered simple and complex CHOs Diabetes 25: 741-747, 1976.

Crapo, P.A., G. Reaven, and J. Olefsky. Postprandial plasma glucose and insulin responses to different complex CHOs. Diabetes 26(12): 1178-1183, 1977.

Decombaz, J.,D.Sartori, M.J.Arnaud, A.L.Thelin, P.Schurch, and H.Howald. Oxidation and metabolic effects of fructose or glucose ingested before exercise. Int. J. Sports Med. 6:282-286, 1985.

Defronzo, R.A., E. Ferrannini, Y. Sarto, P. Felig, and J. Wahren. Synergistic interaction between exercise and insulin on peripheral glucose uptake. J. Clin. Investi. 68: 1468-1474, 1981.

Devlin, J.T.,J. Calles-Escandon, and E.S. Horton. Effects of preexercise snack feeding on endurance cycling exercise. J. Appl. Physiol. 60(3): 980-985, 1986.

Dohm, G.L., A.L. Hecker, W.E. Brown, G.J. Klain, F.R. Puente, E.W. Askew, and G.R. Beecher. Adaptation of protein metabolism to endurance training. Biochem. J. 164: 705-708, 1977.

Dohm, G.L., R.T. Williams, G.L. Kasperek, and A.M. Van Rij. Increased excretion of N-methylhistidine and urea by rats and humans after a bout of exercise. J. Appl. physiol. 52: 27-33, 1982a.

Dohm, G.L., E.B.Tapscott, and G.L. Kasperek. Protein degradation during exercise and recovery. Med. Sci. Sports. Ex. 19(5): Suppl S166-S171, 1987.

Dohm, G.L., G.L.Kasperek, E.B.Tapscott, and G.R.Beecher. Effect of exercise on synthesis and degradation of muscle protein. Biochem. J. 188: 255-262, 1980 .

Ebeling, P., R. Bourey, L. Koranyi, J.A. Tuominen, L.C. Groop, J. Henriksson, M. Mueckler, A. Sovijarvi, and V.A. Koivisto. Mechanism of enhanced insulin sensitivity in athletes: increased blood flow, muscle glucose transport protein (GLUT-4) concentration, and glycogen synthase activity. J. Clin. Invest.92: 623-1631, 1993.

Fain, J.N. Biochemical aspects of drug and hormone action on adipose tissue. Pharmacology Review 25: 67-118, 1973.

Febbraio, M.A., S.E.Selig, S.A. Clarke, R. Walsh, D. Ruter, and W. Portier. The effect of ingesting carbohydrate beverages during prolonged exhaustive exercise in hot and cool conditions. Proc. Aust. Physiol. Pharmacol. Soc. 25: 132P, 1994.

Feldman, M., and J.V. Nixon. Effect of exercise on postprandial gastric secretion and emptying in humans. J. Appl. Physiol. 53(4): 851-854, 1982.

Felig, P., J. Wahren, R. Hendler, and G. Ahlborg. Plasma glucogon levels in exercising man. New England Journal Of Medicine 287: 184-185, 1972.

Felig, P., and J. Wahren. Role of insulin and glucagon in regulation of hepatic glucose production during exercise. Diabetes 28 (Supp 1) :71-75, 1979.

Fell , R.D.,S.E. Terblanche, J.L. Ivy, J.C. Young, and J.O. Holloszy. Effect of muscle glycogen content on glucose uptake following exercise. J. Appl. Physiol. 52: 434-437, 1982.

Fielding, A., D.L. Costill, W.J. Fink, D.S. King, and M. Hargreaves. Effect of carbohydrate feeding frequencies and dosage on muscle glycogen use during exercise. Med. Sci. Sports Exerc. 17: 472-476, 1985.

Fielding, A., D.L. Costill, W.J. Fink, D.S. King, J.E. Kovaleski, and J.P. Kirwan. Effects of preexercise CHO feedings on muscle glycogen use during exercise in well trained runners Eur. J. Appl. Physiol. 56: 225-229, 1987.

Fink, W.J., D.L. Costill, and C.F. Stevens. Gastric emptying characteristics of complete nutritional liquids. In: Nutrient utilisation during exercise. Ohio, Ross Laboratories 112-115, 1983.

Flynn, M.G, D.L. Costill, J.A. Hawley, W.J.Fink, P.D. Neuffer, R.A. Fielding, and M.D. Sleeper. Influence of selected carbohydrate drinks on cycling performance and glycogen use. Med. Sci. Sports. Ex. 19(1): 37-40, 1987.

Foster, C., D.L. Costill, and W.J. Fink. Effects of preexercise feedings on endurance performance Med. Sci. Sports Exerc. 11: 1-5, 1979.

Foster, C., D.L. Costill, and W.J. Fink. Gastric emptying characteristics of glucose and glucose polymer solutions. Research Quarterly For Exercise And Sport 51(2): 299-305, 1980.

Frayn, K., Calculation of substrate oxidation rates in vivo from gas exchange. J. Appl. Physiol. 55(2): 628-634, 1983.

Fredholm, B.B. The effect of lactate in canine subcutaneous adipose tissue in situ. Acta. Physiol. Scand. 82: 110-123, 1971.

Fredrickson, G., P. Stralfors, N.O. Nilsson, and P. Belfrage. Hormone-sensitive lipase of rat adipose tissue. Purification and some properties. J. Biol. Chem. 256: 6311-6320, 1981.

Freedland, R.A. and S. Briggs. A biochemical approach to nutrition. Chapman and Hall, London 1978.

Froberg, S.O. Effect of acute exercise on tissue lipids in rats. Metabolism 20: 714-720, 1971.

Froberg, S.O., E. Hultman, and L.H. Nilsson. Effect of noradrenaline on triglyceride and glycogen concentrations in liver and muscle from man. Metab. Cli. Exp. 23: 119-126, 1975.

Fryburg, D,A., E.J. Barrett, R.J. Louard, and R.A Gelfand. Effect of starvation on human muscle protein metabolism and its response to insulin. Am. J. Physiol. E477-E482, 1990.

Fushiki, T., J.A. Wells, E.B. Tapscott, and G. Lynis Dohm. Changes in glucose transporters in muscle in response to exercise. Am. J. Physiol. 256(19) E580-E587, 1989.

Galbo, H., J.J. Holst, and N.J. Christensen. Glucogon and plasma catecholamine responses to graded and prolonged exercise in man. J. Appl. Physiol. 38(1): 70-76, 1975.

Gelfand, R.A., M.G. Glickman, R. Jacob, R.S. Sherwin, and R.A. Defranzo. Removal of infused amino acids by splanchnic and leg tissues in humans. Am. J. Physiol. 250: E407- E413, 1986.

Gerich, J.E., M.A. Charles, and G.M. Grodsky. Regulation of pancreatic insulin and glucogon secretion. Annu. Rev. Physiol. 38: 353-388, 1976.

Gerich, J.E., J.H. Karam, and.P.H. Forsham. Stimulation of glucagon secretion by epinephrine in man. J. Clin. Endocrin. Metab. 37: 479-481, 1973.

Gleeson, M., R.J. Maughan, and P.L. Greenhaff. Comparison of the effects of preexercise feeding of glucose, glycerol and placebo on endurance and fuel homeostasis in man Europ. J. Appl. Physiol. 55: 645-653, 1986.

Goddard, M.S., G. Young, and R. Marcus. The effect of amylose content on insulin and glucose responses to ingested rice. Am. J. Clin. Nut. 39: 388-392, 1984.

Golay, A., A.M. Coulston, and C.B Hollenbeck. Comparison of metabolic effects of white beans processed into two different physical forms. Diabetes care 9: 260-266, 1986.

Gollnick, P.D., B. Pernow, B. Essen, E. Jansson, and B. Saltin. Availabilty of glycogen and plasma FFA for substrate utilization in leg muscle of man during exercise. Clin. Phys. 1: 27-42, 1981.

Gollnick, P.D. and B. Saltin. Fuel for muscular exercise: role of fat. In Horton, E.S., and R.L.Terjung (Eds). Exercise, nutrition and energy metabolism. Macmillan Publishing Co.N.Y., p72-88, 1988.

Goodyear, L.J., M.F. Hirshman, P.A. King, E.D. Horton, C.M.Thompson, and E.S. Horton. Skeletal muscle plasma membrane glucose transport and glucose transporters after exercise. J. Appl. Physiol. 68(1): 193-198, 1990.

Gordon, B., L.A. Kohn, S.A Levine, M. Matton, W. Scriver, and W.B. Whiting. Sugar content of the blood in runners following a marathon race. With special reference to the prevention of hypoglycemia: further observations. J.A.M.A. 85: 508-509, 1925.

Gorski, J. Exercise induced changes of activity of different types of muscle on glycogenolytic effect of adrenative. Pflugers Arch. 373: 1-7, 1978.

Gorski, J. Muscle triglyceride metabolism during exercise. Canad. J. Physiol. Pharmacol. 70: 123-131, 1992.

Graham, T.E., and D.A. Maclean. Ammonia and amino acid metabolism in human skeletal muscle during exercise. Canad. J. Physiol. Pharmacol. 70: 132-141, 1990.

Granfeldt, Y. and I. Bjorck. Glycemic response to starch in pasta: a study of mechanisms of limited enzyme availability. Journal Of Cereal science 14: 47-61, 1991.

Greenhaff, P.L., J. Ren, K. Soderland, and E. Hultman. Energy metabolism in single human muscle fibres during contraction with and without epinephrine infusion. Am. J. Physiol. 260: E713-E718, 1991.

Guezennec, C.Y., P. Satabin, F. Duforez, D. Merino, F. Peronnet, and J. Koziet. Oxidation of corn starch, glucose, and fructose ingested before exercise. Med. Sci. Sports Exerc. 21(1): 45-50, 1989.

Haber, G.B., K.W. Heaton, D. Murphy, and L.F. Burroughs. Depletion and disruption of dietary fibre. Effects on satiety, plasma glucose, and serum insulin. Lancet 2: 679-682, 1977.

Hagenfeldt, L. Turnover of individual fatty acids in man. Fed. Proc. Fed. Am. Soc. Exp. 34: 2236-2240, 1975.

Hargreaves, M., D.L. Costill, A. Coggan, W.J. Fink, and I. Nishibita. Effect of CHO feedings on muscle glycogen utilization and exercise performance. Med. Sci. Sports Exerc. 16(3): 219-222, 1984.

Hargreaves, M., D.L. Costill, A. Katz, and W.J. Fink. Effect of fructose ingestion on muscle glycogen usage during exercise. Med. Sci. Sports. Ex. 17(3): 360-363, 1985.

Hargreaves, M., D.L. Costill, W.J. Fink, D.S. King, and R.A. Fielding. Effect of preexercise CHO feedings on endurance cycling performance Med. Sci. Sports. Ex. 19 (1): 33-36, 1987.

Hargreaves, M. and C.A. Briggs. Effect of carbohydrate ingestion on exercise metabolism. J. Appl. Physiol. 65(4):1553-1555, 1988.

Hargreaves, M., B. Kiens, and E.A. Richter. Effect of increased plasma free fatty acid concentrations on muscle metabolism in exercising men. J. Applied Physiol. 70 (1): 194-201, 1991.

Hargreaves, M., J.P. Finn, R.T. Withers, G.C. Scroop, J. Halbert, M. Mackay, and M.F. Carey. Influence of muscle glycogen on maximal exercise performance. Proceedings of the Australian Physiological and Pharmacollogical Society 25(2): 129P 1994.

Hargreaves, M., G. McConell, and J. Proietto. Influence of muscle glycogen on glycogenolysis and glucose uptake during exercise in humans. J. Applied Physiol. 78: 288-292, 1995.

Havel, R.J., B. Pernow, and N.L. Jones. Uptake and release of free fatty acids and other metabolites in the legs of exercising men. J. Appl. Physiol. 23: 90-99, 1967.

Havel, R.J. The fuels for muscular exercise. In Science and Medicine of Exercise and Sport. W.R.Johnson and E.R.Buskirk, editors. Harper and Row, New York. 137-152.

Havel, R.J., L.A. Carlson, L.G. Ekelund, and A. Holmgren. Turnover rate and oxidation of different free fatty acids in man during exercise. J. Appl. Physiol. 19: 613-618, 1964.

Heath, G.W., J.R. Gavin Iii, J.M. Hinerliter, J.M. Hagberg, S.A. Bloomfield, and J.O. Holloszy. Effects of exercise and lack of exercise on glucose tolerance and insulin sensitivity. J.Appl. Physiol. 55(2): 512-517, 1983.

Hermansen, L., E. Hultman, and B. Saltin. Muscle glycogen during prolonged severe exercise. Acta Physiol Scand 71: 129-139, 1967.

Hespel, P., and E.A. Richter. Mechanism linking glycogen concentration and glycogenolytic rate in perfused contracting rat skeletal muscle. J. Biochem. 284: 777-780, 1992.

Hickson, R.C., M.J. Rennie, R.K. Conlee, W.W. Winder, and J.O. Holloszy. The effects of increased plasma free fatty acids on glycogen utilization and endurance. J. Appl. Physiol. 43(5): 829-833, 1977.

Holloszy, J.O., J.S. Skinner, G. Toro, and T.K. Cureton. Effects of a six month program of endurance exercise on the serum lipids of middle aged men. Am. J. Cardiol. 14: 753-760, 1964.

Holloszy, J. and H.T. Narahara. Studies on tissue permeability. X. Changes in tissue permeability to 3-methylglucose associated with contraction of isolated frog muscle. J. Biol. Chem. 240: 3493-3500, 1965.

Holloszy, J. and H.T. Narahara. Enhanced permeability to sugar associated with muscle contraction; Studies of the role of CA+2. J. Gen.Physiol. 50: 551-562 1967.

Holloszy, J.O., S.H. Constable, and D.A. Young. Activation of glucose transport in muscle by exercise. Diabetes Metabolism reviews 1: 409-423 1968.

Holm, J., I. Lundquist, and I. Bjorck. Degree of starch gelatinization, digestion rate of starch in vitro and metabolic response of rats. Am. J. Clin. Nut. 47: 1010-1016, 1988.

Hopp, J.F., and W.K. Palmer. Effect of electrical stimulation on intracellular triacylglycerol in isolated skeletal muscle. J. Appl. Physiol. 68: 348-354, 1990.

Hood, D.A., and R.J. Terjung. Amino acid metabolism during exercise. Sports Med. 9(1): 23-35, 1990.

Horowitz, J.F. and E.F. Coyle. Metabolic responses to preexercise meals containing various carbohydrates and fat. Am. J. Clin. Nut. 58: 235-241, 1993.

Houmard, J.A., P.C. Egan, P.D. Neuffer, J.E. Friedman, W.S. Wheeler, R.G. Israel, and G.L. Dohm. Elevated skeletal muscle glucose transporter levels in exercise-trained middle aged men. Am. J. Physiol. 261(24): E437-E443, 1991.

Houmard, J.A., T. Hortobagyi, P.D. Neuffer, R. Anderson Johns, D.D. Fraser, R.G. Israel, and G.L. Dohm. Training cessation does not alter GLUT-4 protein levels in human skeletal muscle. J. Appl. Physiol. 74(2): 776-781, 1993.

Hunt, J.N. and J.D. Pathak. The osmotic effect of some simple molecules and ions on gastric emptying. J. Physiol. 154: 254-269, 1960.

Hunt, J.N. and M.T. Knox. Regulation of gastric emptying. In: Handbook of Physiology IV, Alimentary Canal, C.F. Code (Ed.) Washington D.C: American Physiological Society, 1917-1935, 1969.

Hunt, J.N., J.L. Smith, and C.L. Jiang. Effect of meal volume and energy density on the gastric emptying of carbohydrates. Gastro. 89: 1326-1330, 1985.

Idstrom, J.P., M.J. Rennie, T. Schersten, and A.C. Bylund-Fellenius. Membrane transport in relation to net uptake of glucose in the perfused rat hindlimb: stimulatory effect of insulin, hypoxia, and contractile activity. Biochem. J. 233: 131-137, 1985.

Issekutz, B., and H. Miller. Plasma free fatty acids during exercise and the effect of lactic acid. Proc. Soc. Biol. Med. 110: 237-239, 1962.

Issekutz, B., and P. Paul. Intramuscular energy sources in exercising normal and pancreatectomized dogs. Am. J. Physiol. 215: 197-204, 1968.

Ivy, J.L., D.L. Costill, W.J. Fink, and R.W. Lower. Influence of caffeine and carbohydrate feedings on endurance performance. Med. Sci. Sports. 11: 6-11, 1979.

Ivy, J.L., W. Miller, V. Dover, L.G. Goodyear, W.M. Sherman, S. Farrell, and H. Williams. Endurance improved by ingestion of a glucose polymer supplement. Med. Sci. Sports 15(6): 466-471, 1983.

James, D.E., E.W. Kraegen, and D.J. Chisholm. Muscle glucose metabolism in exercising rats: comparison with insulin stimulation. Am. J. Physiol. 248(11): E575-E580, 1985.

James, D.E., M. Strube, And M. Mueckler. Molecular cloning and characterisation of an insulin-regulatable glucose transporter. Nature Lond. 338: 83-87, 1989.

Jandrain, B., G. Krzentowski, F. Pirnay, F. Mosora, M. Lacroix, A. Luyckx, and P. Lefebvre. Metabolic availability of glucose ingested 3 h before prolonged exercise in humans. J. Appl. Physiol. 56(5): 1314-1319, 1984.

Jandrain, B., N. Pallikarakis, S. Normand, F. Pirnay, M. Lacroix, F. Mosora, C. Pachiaudi, J.F.Gauthier, A.J. Scheen, J.P. Riou, and P. Lefebvre. Fructose utilisation during exercise in men: rapid conversion of ingested fructose to circulating glucose. J. Appl. Physiol. 74: 2146-2154, 1993.

Jansson, E., P. Hjemdahl, and L. Kaijser. Epinephrine-induced changes in muscle carbohydrate metabolism during exercise in male subjects. J. Appl. Physiol. 60(5): 1466-1470. 1986.

Jenkins, D.J.A., T.M.S. Wolever, R.H. Taylor, H. Barker, H. Fielden, J.M. Baldwin, A.C. Bowling, H.C. Newman, A.L. Jenkins, and D.V .Goff. Glycemic index of foods: a physiological basis for CHO exchange. Am. J. Clin. Nutr. March: 362-366, 1981.

Jenkins, D.J.A., T.M.S. Wolever, A.L. Jenkins, M.J. Thorne, R. Lee, J.Kalmusky, R .Reichert, and G.S. Wong. The glycaemic index of foods tested indiabetic patients: A new basis for carbohydrate exchange favouring the use of legumes. Diabetologia 24: 257-264, 1983.

Jenkins, D.J.A., T.M.S. Wolever, A.L. Jenkins, R.G Josse, and G.S. Wong . The glycaemic response to CHO foods. Lancet 388-391, Aug 1984.

Jenkins, D.J.A., T.M.S. Wolever, and A.L. Jenkins. Low glycemic response to traditionally processed wheat and rye products: bulgar and pumpernickel bread. Am. J. Clin. Nut. 43: 516-520, 1986.

Johnson, L.N. Glycogen phosphorylase: control by phosphorylation and allosteric effectors. FASEB J. 2274-2282 1992.

Jones, N.L., J.F. Heigenhauser, A. Kuksis, C.G. Matsos, J.R. Sutton, and C.J. Toews. Fat metabolism in heavy exercise. Clin. Sci. 59: 469-478, 1980.

Kasperek, G.J., G.L. Dohm, H.A. Barakat, P.H. Strausbauch, D.W. Barnes, and R.D. Snider. The role of of lyosomes in exercise-induced hepatic protein loss. Biochem. J. 202: 281-288, 1982.

Katz, A., S. Bromberg, K. Sahlin, and J. Wahren. Leg glucose uptake during maximal dynamic exercise in humans. Am. J. Physiol. 251: E65-E70, 1986.

Keller, K., and R. Schwarzkopf. Preexercise snacks may decrease exercise performance Physician And Sports Medicine 12(4): 89-91, 1984.

Kern, M., J.A. Wells, J.M. Stehens, C.W. Elton, J.E. Friedman, E.B. Tapscott, P.H. Pekala, and G.L. Dohm. Insulin responsiveness in skeletal muscle is determined by glucose transporter (Glut-4) protein level. J. Biochem. 270: 397-400, 1990.

Khoo, J.C. and D. Steinberg. Reversible protein kinase activation of hormone sensitve lipase from chicken adipose tissue. J. Lipid Res. 15: 602-610, 1974.

Kiens, B., B. Essen-Gustavsson, N.J Christensen, and B. Saltin. Skeletal muscle substrate utilisation during submaximal exercise in man: Effect of endurance training. J. Physiol.469: 459-478, 1993.

King, P.A., M. Hirshman, E.D. Horton, and E.S. Horton. Glucose transport in skeletal muscle membrane vesicles from control and exercised rats. Am. J. Physiol. 257(26): C1128-C1134, 1989.

Kjaer, M., P.A. Farrell, N.J. Christensen, and H. Galbo. Increased epinephrine response and inaccurate glucose regulation in exercising athletes. J. Appl. Physiol. 61: 1693-1700, 1986.

Kjaer, M., N.H. Secher, F.W. Bach, and H. Galbo. Role of motor center activity for hormonal changes and substrate mobilization in humans. American J. Physiol. 253: R687-R695, 1987.

Klip, A. and M.R. Paquet. Glucose transport and glucose transporters in muscle and their metabolic regulation. Diabetes Care 13: 228-243, 1990.

Koivisto, V., S.L. Karonen, and E.A. Nikkila. Carbohydrate ingestion before exercise: comparison of glucose, fructose and sweet placebo. J. Appl. Physiol. 51(4): 783-787. 1981.

Koivisto, V., M. Harkonen, S.L. Karonnen, P.H. Groop, R. Elovaino, E.Ferrannini, L. Sacca, and R. Defronzo. Glycogen depletion during prolonged exercise: influence of glucose, fructose, or placebo. J. Appl. Physiol. 58(3): 731-737, 1985.

Koivisto, V.A., P. Ebeling, A. Sovijarvi, and R.E. Bourey. Enhanced insulin sensitivity in the athletes: increased muscle blood flow and GLUT-4 protein content. (Abstract) Diabetologia 35: Suppl 1 A53, 1992.

Koivisto, V.A., R.E. Bourey. H. Vourinen-Markkola, and L. Koranyi. Exercise reduces muscle glucose transport protein (GLUT-4) mRNA in

type 1 diabetic patients. J. Appl. Physiol. 74(4): 1755-1760, 1993.

Krebs, H.A., R.A. Freedland, R. Hems, and M. Stubbs. J. Biochem. 112 p117

Lemon, P.W.R., and J.P. Mullin. Effect of initial muscle glycogen levels on protein catabolism during exercise. J. Appl. Physiol. 48: 624-629, 1980.

Lemon, P.W.R., K.E. Yarasheski, and D.G. Dolny. The importance of protein for athletes. Sports Med. 1: 474-484, 1984.

Lemon ,P.W.R., D.G. Dolny, and B.A. Sherman. Effect of intense prolonged running on protein catabolism. In Knuttgen et al.(Eds). Biochemistry of Exercise 362-372, Human kinetics, Champaign 1983.

Levine, L., W.J. Evans, B.S. Cadarette, E.C. Fisher, and B.A. Bullen. Fructose and glucose ingestion and muscle glycogen use during submaximal exercise J. Appl. Physiol. 55(6): 1767-1771, 1983.

Lowry, O.H., and J.V. Passonneau. Flexible System of Enzymatic Analysis. New York: Academic Press, 1972.

Luyckx, A.S.,F. Pirnay, and P.J. Lefebvre. Effect of glucose and plasma glucagon and free fatty acids during prolonged exercise. Europ. J. Appl. Physiol. 39: 53-61 1978.

Maclaren, D.P.M., T. Reilly, I.T. Campbell, and K.N. Frayn. Hormonal and metabolite responses to glucose and maltodextrin ingestion with

or without the addition of guar gum. Int. J. Sports Med. 15(8): 466-471 1994.

Maclean, D.A., L.L. Spriet, E. Hultman, and T.E. Graham. Plasma and muscle amino acid and ammonia responses during prolonged exercise in humans. Journal Of Applied Physiology 70(5): 2095-2103, 1991.

Maclean, D.A., T.E. Graham, and B. Saltin. Branched- chain amino acids augment ammonia metabolism while attenuating protein breakdown during exercise. Am. J. Physiol. 267: E1010- E1022, 1994.

Massicotte, D., F. Peronnet, C. Allah, C. Hillaire-Marcel, M. Ledoux, and G. Brisson. Metabolic response to [13C] glucose and [13C] fructose ingestion during exercise. J. Appl. Physiol. 61: 1180-1184, 1986.

Massicotte, D., F.Peronnet, G.R.Brisson, K.Bakkouch, and C.Hillaire-Marcel. Oxidation of a glucose polymer during exercise: comparison with glucose and fructose. J. Appl. Physiol. 66(1): 179-183, 1989.

McConell, G., S. Fabris, J. Proietto, and M. Hargreaves. Effect of carbohydrate ingestion on glucose kinetics during exercise. J. Appl. Physiol. 77(3): 1537-1541, 1994.

McCoy, M., J. Proietto, and M. Hargreaves. Effect of detraining on GLUT-4 protein in human skeletal muscle. J. Appl. Physiol. 77(3): 1532 1536, 1994.

McDermott, J.C., G.C.B. Elder, and A. Bonen. Adrenal hormones enhance glycogenolysis in non exercising muscle during exercise. J. Appl. Physiol. 63(3): 1275-1283, 1987.

McMurray, R., J.R. Wilson, and B.S. Kitchell. The effects of fructose and glucose on high intensity endurance performance Res. Quart. Exer. Sports Sci. 54(2): 156-162, 1983.

Meyer, J.H., H. Ohashi, D. Jehn, and J.B. Thomson. Size of liver particle emptied from the human stomach. Gastro. 80: 1489-1496, 1981.

Miles, J., R. Glasscock, J. Aikens, J. Gerich, and M. Haymond. A microfluometric method for determination of free fatty acids in plasma. J. Lipid Res. 24: 96-99,1983.

Millard-Stafford, M.L.,P.B. Sparling, L.B. Roskopf, and L.J. Dicarlo. Carbohydrate-electrolyte replacement improves distance running performance in the heat. Med. Sci. Sports Exerc. 24(8): 934-940, 1992.

Miller, H.I., Issekutz,B, P. Paul, and K. Rodahl. Effect of lactic acid on plasma free fatty acids in pancreatectomized dogs. Am. J. Physiol. 207: 1226-1230, 1964.

Miller, L.L The role of the liver and non-hepatic tissues in the regulation of free amino acid levels in the blood; In Holden (Ed). Amino acid pools, 708-721, Elsevier, Amsterdam, 1962.

Miller, W.C., R.C. Hickson, and N.M. Bass. Fatty acid binding proteins in the three types of rat skeketal muscle. Proc. Soc. Exp. Biol. 189: 183-188, 1988.

Mitchell, J.B., D.L. Costill, J.A. Houmard, M.G. Flynn, W.J. Fink, and J.D. Beltz. Effects of Carbohydrate ingestion on gastric emptying and exercise performance. Med Sci Sports Exer. 20(2): 110-115, 1988.

Mitchell, J.B., D.L. Costill, J.A. Houmard, W.J. Fink, D.D. Pascoe, and D.R. Pearson. Influence of carbohydrate dosage on exercise performance and glycogen metabolism. J. Appl. Physiol. 67(5): 1843-1849 1989.

Mitchell, J.B., D.L. Costill, W.J. Fink, R.A. Robergs, and J.A. Davis. Gastric emptying: influence of prolonged exercise and carbohydrate concentration. Med. Sci. Sports Exer. 21(3): 269-274, 1989.

Mitchell, J.B., D.L. Costill, J.A. Houmard, M.G. Flynn, W.J. Fink, and J.D. Beltz.. Influence of CHO ingestion on counterregulatory hormones during prolonged exercise. Int. J. Sports Med. 11: 33-36, 1990.

Mitchell, J.B. and Voss.K.W. The influence of volume on gastric emptying and fluid balance during prolonged exercise. Med. Sci. Sports Exerc. 23(3): 314-319, 1991.

Montain, S., M.K .Hopper, A.R. Coggan, and E.F. Coyle. Exercise metabolism at different time intervals after a meal J. Appl. Physiol. 70(2): 882-888, 1991.

Mourot, J., P. Thouvenot, C. Couet, J.M. Antione, A. Krobicka, and G. Debry. Relationship between the rate of gastric emptying and glucose and insulin responses to starchy foods in young healthy adults American J. Clin. Nut. 48: 1035-1040, 1988.

Murray, R., G.L. Paul, J.G. Seifert, D.E. Eddy, and G.A. Halaby. The effects of glucose, fructose, and sucrose ingestion during exercise. Med. Sci. Sports Exerc. 21(3): 275-282, 1989.

Naveri, H., H. Tikkanen, A.L. Kairento, and M. Harkonen. Gastric emptying and serum insulin levels after intake of glucose polymer solutions. Europ. J. Appl. Physiol. 58: 661-665, 1989.

Neufer, P.D., D.L. Costill, W.J. Fink, J.P .Kirwan, R.A. Fielding, and M.G. Flynn. Effects of exercise and CHO composition on gastric emptying. Med.and Sci. Sports Exer. 18(6): 658-662, 1986.

Neufer, P.D., D.L. Costill, M.G. Flynn,J.P. Kirwan, J.B. Mitchell, and J. Houmard. Improvements in exercise performance: effects of CHO feedings and diet. J. Appl. Physiol. 62(3): 983-988, 1987.

Nilsson, L.H., and E.H ultman. Liver and muscle glycogen in man after glucose and fructose infusion. Scand. J. Clin. Lab. Invest. 33: 5-10, 1974.

Noakes, T.D., N.J. Rehrer, and R.J. Maughan. The importance in volume in regulating gastric emptying. Med. Sci. Sports Exerc. 23(3): 307-313, 1991.

Nobin, A., B. Falck, S.Ingemansson, J. Jarhult, and B. Rosengren. Organization and function of the sympathetic innervation of human liver. Acta Physiol. Scand. Suppl 452: 103-106, 1977.

Odessey, R., and A.L. Goldberg. Oxidation of leucine by rat skeletal muscle. Am. J. Physiol. 223: 1376-1383, 1972

O'dea, K,, P.J. Nestel and L. Antonoff. Physical factors influencing postprandial glucose and insulin responses to starch. Am. Journal Clin. Nut. 33: 760-765, 1980.

Okano, G. and T. Shimojo. Utilization of long-chain free fatty acids in white and red muscle of rats. Biochim. Biophys. Acta 710: 122-127, 1982.

Okano, G., H. Takeda, I. Morita, M. Katoh, Z. Mu, and S. Miyake. Effect of preexercise fructose ingestion on endurance performance in fed men. Med. Sci.Sports Exer. 20(2): 105-109, 1988.

Oscai, L.B., J.A. Patterson, D.L. Bogard, R.J. Beck, and B.L. Rothermel. Normalization of serum triglycerides and lipoprotein electrophoretic patterns by exercise. Am. Cardiol. 30: 775-780, 1972.

Oscai, L.B. and W.K. Palmer. Muscle Lipolysis during exercise; An update. Sports Med. 6: 23-28, 1988.

Oscai, L.B., D.A. Essig, and W.K. Palmer. Lipase regulation of muscle triglceride hydrolysis. Journal Of Applied Physiology 69(5): 1571-1577, 1990.

Paul, P. FFA metabolism of normal dogs during steady state exercise at different work loads. Journal Of Applied Physiology 28: 127-132, 1970.

Peden, C., and W.M. Sherman. 1 H prexercise CHO meals enhance performance Medicine And Science In Sports And Exercise 21(2): supp S59, 1989.

Ploug, T., H. Galbo, J. Vinten, M. Jorgensen, and E.A. Richter. Kinetics of glucose transport in rat muscle: effects of insulin and contractions. Am.J. Physiol. 253(16): E12-E20, 1987.

Ploug, T., B.M Stallknecht, O. Pedersen, B.B. Kahn, T. Ohkuwa, J. Vinten, and H. Galbo. Effect of endurance training on glucose transport capacity and glucose transporter expression in rat skeletal muscle. Am. J. Physiol. 259(22): E778-E786, 1990.

Porte, D. and R.H. Williams. Inhibition of insulin release by norepinephrine in man. Science 152: 1248-1250, 1966.

Reaven, G.M. Effects of differences in amount and kind of dietary carbohydrate on plasma glucose and insulin responses in man. Am.J. Of Clin.Nut. 32: 2568-2578, 1979.

Reed, M.J., J.T. Brozinick, Jr., M.C. Lee, and J.L Ivy. Muscle glycogen storage postexercise: effect of mode of carbohydrate administration. J. Appl. Physiol. 66(2): 720-726, 1989.

Rehrer, N,J. The maintenance of fluid balance during exercise. J. Appl. Physiol. 15(3): 122-125, 1994.

Reitman, J.K., M. Baldwin, and J.O. Holloszy. Intramuscular triglyceride utilization by red, white and intermediate skeletal muscle and heart during exhaustive exercise. Proc.Soc.Exp.Biol.Med 142: 628-631, 1973.

Rennie, M.J., R.H.T. Edwards, S. Krywawych, C.T.M. Davies, D. Halliday, and D.J. Millward. Effects of exercise on protein turnover in man. Clin.Sci. 61: 627-639, 1981.

Riby, J.E., T. Fujisawa, and N. Kretchmer. Fructose absorption. Am. J.Of Clin. Nut. 58: 746S-751S, 1993.

Richter, E.A., H. Galbo, B. Sonne, J.J. Holst, and N. J. Christensen. Adrenal medullary control of muscular and hepatic glycogenolysis and of pancreatic hormonal secretion in exercising rats. Acta. Physiol. Scand. 108: 235-242, 1980.

Richter, E.A., H. Galbo, and N. J. Christensen. Control of exercise-induced muscular glycogenolysis by adrenal medullary hormones in rats. Journal Of Applied Physiology 50(1): 21-26, 1981.

Richter, E.A., N.B. Ruderman, and H. Galbo. Alpha and beta adrenergic effects on metabolism in contracting perfused muscle. Acta Physiol. Scand. 116: 215-222, 1982.

Richter, E.A. and H. Galbo. High glycogen levels enhance glycogen breakdown in isolated contracting skeletal muscle. J. Appl. Physiol. 61: 827-831, 1986.

Richter, E.A. Influence of the sympathoadrenal system on some metabolic and hormonal responses to exercise in the rat. Acta. Physiol. Scand. Suppl 528: 1-42, 1984.

Robinson, D.S. The function of plasma triglycerides in fatty acid transport. In Florkin & Stotz (Ed). Comprehensive biochemistry. 18: 51-116, Elsevier Publishing Co. Amsterdam, 1970.

Romijn, J.A., E.F. Coyle, L.S. Sidossis, A. Gastaldelli, J.F. Horowitz, E. Endert, and R.R. Wolfe. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. Am. J. Physiol. 265(28): E380-E391, 1993.

Ryan, A.J., T.L. Bleiler, J.E. Carter, and C.V. Gisolfi. Gastric emptying during prolonged cycling exercise in the heat. Med. Sci. Sports Exerc. 21(1): 51-58, 1989.

Sahlin, K. Intracellur pH and energy metabolism in skeletal muscle of man. With special reference to exercise. Acta. Physiol. Scand. Suppl. 455: 1-56, 1978.

Sharp, R.L., C.J. Lockard, D.S. King, and J.F. Robyt. Effect of pre-exercise modified starch feedings on carbohydrate metabolism during endurance exercise. Med. Sci. Sports Exerc. 25(5): Supp (abstract) S99, 1993.

Shepherd, R.E., and M.D. Bah. Cyclic AMP regulation of fuel metabolism during exercise: regulation of adipose tissue lipolysis during exercise. Med. and Sci. Sports Exer. 20(6): 531-538, 1988.

Sherman, W.M., D.L. Costill, and W.J. Fink. Effects of exercise-diet manipulation on muscle glycogen and its subsequent utilisation during performance. Int. J. Sports Med. 2(2): 114-118, 1981.

Sherman, W.M., G. Brodowicz, D.A. Wright, W.K Allen, J. Simonsen, and A. Dernbach. Effects of 4hr preexercise CHO feedings on cycling performance. Med. Sci. Sports Exerc. 21(5): 598-604, 1989.

Sherman, W.M., C.Peden, and D.A.Wright. Carbohydrate feedings 1h before exercise improves cycling performance. Am. J. Clin. Nutr. 54: 866-70, 1991.

Snow, R.J., C.G. Stathis, M.A. Febbraio, M.F. Carey, and M. Hargreaves. Carbohydrate ingestion attenuates muscle and plasma

ammonia accumulation during prolonged exercise. (manuscript in preparation), 1995.

Snyder, A.C., D.R. Lamb, T. Baur, D. Connors, and G. Brodowicz. Maltodextrin feeding immediately before prolonged cycling at 62% vo2max increases time to exhaustion Med. Sci. Sports Exer. 15(Supp): 126, 1983.

Snyder, A.C., K .Moorhead, J. Luedtke, and M. Small. Carbohydrate consumption prior to repeated bouts of high intensity exercise European Journal Of Applied Physiology 66: 141-145, 1993.

Sole, C.C., and T.D. Noakes. Faster gastric emptying rates for glucose-polymer and fructose solutions than for glucose in humans. Europ. J. Appl. Physiol.58: 605-612, 1989.

Solomon, S.S. and W.C. Duckworth. Effect of antecedent hormone administration on lipolysis in the perifused isolated fat cell. J. Lab. Clin.Med. 88: 984-994, 1976.

Sonne, B., K.J. Mikines, E.A. Richter, N.J. Christensen, and H. Galbo. Role of liver nerves and adrenal medulla in glucose turnover of running rats. Journal Of Applied Physiology 59: 1640-1646, 1985.

Sonne, B,. and H. Galbo. Carbohydrate metabolism during and after exercise in rats: studies with radio glucose. J. Appl. Physiol. 59: 1627-1639, 1985.

Sonne, B., and H. Galbo. Carbohydrate metabolism in fructose fed and food restricted running rats. Journal Of Applied Physiology 6: 1457-1466, 1986.

Spriet, L.L., G.J.F. Heigenhauser, and N.L. Jones. Endogenous triacylglycerol utilization by rat skeletal muscle during tetanic stimulation. Journal Of Applied Physiology 60: 410-415, 1986.

Stathis, C.G., M.A. Febbraio, M.F. Carey, and R.J. Snow. Influence of sprint training on human skeletal muscle purine nucleotide metabolism. Journal Of Applied Physiology 76(4): 1802-1809, 1994.

Stankiewicz-Choroszucha, B., and J. Gorski. Effect of decreased availability of substrates on intramuscular triglyceride utilisation during exercise. Europ. J. Appl. Physiol. 40: 27-35, 1978.

Sternlicht, E., R. James Barnard, and G.L. Grimditch. Mechanism of insulin action on glucose transport in rat skeletal muscle. Am. J. Physiol. 254(17): E633-E638, 1988.

Sternlicht, E., R. James Barnard, and G.L. Grimditch. Beta-adrenergic receptors are not responsible for exercise stimulation of glucose transport. Journal Of Applied Physiology 66(5): 2419-2422, 1989.

Sternlicht, E., R. James Barnard, and G.L. Grimditch. Exercise and insulin stimulate skeletal muscle glucose transport through different mechanisms. Am. J. Physiol.) 256(19): E227-E230, 1989.

Strohfeld, P., and C. Heugel. Characterization of triglyceride lipase activities in rat skeletal muscle. Biochem. Biophs. Res. Commun. 121: 87-94, 1984.

Tan, M.H., T. Sata, and R.J. Havel. The significance of lipoprotein lipase in rat skeletal muscles. J.Lipid Res. 18: 363-370, 1977.

Tan, M.H. and R.J. Havel. Lipoprotein lipase in rat skeletal muscles of different fiber types. Diabetes 24(Suppl 2): 417, 1975.

Terjung, R.L., L. Budohoski, K. Nazar, A. Kobryn, and H. Kaciuba-Uscilko. Chylomicron triglyceride metabolism in resting and exercising fed dogs. Journal Of Applied Physiology. 52: 815- 820, 1982.

Terjung, R.L., B.G. Mackie, G.A Dudley, and H. Kaciuba-Uscilko. Influence of exercise on Chylomicron triaclglycerol metabolism: plasma turnover and muscle uptake. Med. Sci. Sport Exer. 15: 340-347, 1983.

Thomas, E.J. Mechanics and regulation of gastric emptying. Physiol.rev.37: 453-474, 1953.

Thomas, D.E., J.R. Brotherhood, and J.C. Brand. Carbohydrate feeding before exercise: Effect of glycemic index. Int. J. .Sports Med. 12: 180-186, 1991.

Tounian, P., P. Schneiter, S. Henry, E. Jequier, and L. Tappy. Effects of infused fructose on endogenous glucose production, gluconeogenesis, and glycogen metabolism. Am. J. Physiol. 267: E710-E717, 1994.

Trout, D.L., K.M. Behall, and O. Osilesi. Prediction of glycemic index for starchy foods. Am. J. Clin. Nut. 58: 873-878, 1993.

Ventura, J.L., A. Estruch, G. Rodas, and R. Segura. Effect of prior ingestion of glucose or fructose on the performance of exercise of intermediate duration. European J. Appl. Physiol. 68: 345-349, 1994.

Vollestad, N.K, O. Vaage, and L. Hermansen. Muscle glycogen depletion patterns in type 1 and subgroups of type 11 fibres during prolonged severe exercise in man. Acta Physiologica Scandinavia 122: 433-441, 1984.

Vollestad, N.K., and P.C.S. Blom. Effect of varying exercise intensity on glycogen depletion in human muscle fibres. Acta Physiologica Scandinavia 125: 395-405, 1985.

Wagenmakers, A.J.M., J.H. Coakley, and R.H.T. Edwards. Metabolism of Branched-chain amino acids and ammonia during exercise: Clues from McArdle's disease. Int. J. Sports Med. 11 (Supp 2): S101-S113, 1990.

Wahren, J., P. Felig, G. Ahlborg, and L. Jorfeldt. Glucose metabolism during leg exercise in man. J. Clin. Invest. 50: 2715-2725, 1971.

Walaas, O. and E. Walaas. Effect of epinephrine on rat diaphragm. J. Biol.l Chem.187: 769-776, 1950.

Walker, P.M., J.P. Idstrom, T. Schersten, and A.C. Bylund-Fellenius. Glucose uptake in relation to metabolic state in perfused rat hind limb at rest and during exercise. European J. Appl. Physiol. 48: 163-176, 1982.

Wallberg-Henriksson, H. and J.O. Holloszy. Contractile activity increases glucose uptake by muscle in severely diabetic rats. J. Appl. Physiol. 57(4): 1045-1049, 1984.

Wardzala, L.J., and B. Jeanrenaud. Potential mechanism of insulin action on glucose transport in the isolated rat diaphragm. Apparent

translocation of intracellular transport units to the plasma membrane. J. Biol. Chem. 256: 7090-7093, 1981.

Wasserman, D.H., H. Lavina, A. Lickley, and M. Vranic. Interactions between glucagon and other counterregulatory hormones during normoglycemic and hypoglycemic exercise in dogs. J. Clin. Invest. 74: 1404-1413, 1984.

Wassner, S.J., and J.B. Li. N-methylhistidine release: Contributions of rat skeletal muscle, GI tract and skin. Am. J. Physiol. 243: E293-E297, 1982.

Widrick, J,J., D.L. Costill, W.J. Fink, M.S. Hickey, G.K. Mcconell, and H. Tanaka. Carbohydrate feedings and exercise performance: effect of initial muscle glycogen concentration. J. Appl. Physiol. 74(6): 2998-3005, 1993.

Wolever, T.M.S.The glycemic index. In: Bourne G.H, ed. Aspects of some vitamins, minerals and enzymes in health and disease. World Rev Nutr Diet 62: 120-185, 1990.

Wolfe, R.R., E.R. Nadel, J.H.F. Shaw, L.A. Stephenson, and M.H. Wolfe. Role of changes in insulin and glucagon in glucose homeostasis in exercise. Metabolism 77: 900-907, 1986.

Wright, D.A, W.M. Sherman, and A.R. Dernbach. Carbohydrate feedings before, during, or in combination impove cycling performance J. Appl. Physiol. 71(3): 1082-1088, 1991.

Yoon, J.H., L.U. Thompson, and D.J.A. Jenkins. The effect of phytic acid on in vitro rate of starch digestibility and blood glucose response. Am.J. Clin. Nut. 38: 835-842, 1983.

Young, D.A., H. Wallberg-Henriksson, J. Cranshaw, M. Chen, and J.O Holloszy. Effect of catecholamines on glucose uptake and glycogenolysis in rat skeletal muscle. Am. J. Physiol. 248 (17): C406-C409, 1985.

Zorzano, A., T.W. Balon, M.N. Goodman, and N.B. Rudderman. Additive effects of prior exercise and insulin on glucose and AIB uptake by muscle. American Journal Of Physiology 251 (14): E21-E26,1986.

Appendix A

Subject Correspondence

INFORMATION FOR SUBJECTS

This study aims to examine the effect of eating particular types of food containing carbohydrate before exercise, on metabolism and exercise performance. You will be asked to attend the laboratory on five separate occasions to participate in exercise trials on a cycling ergometer. The tests will include an incremental exercise test, a familiarisation trial and three experimental trials. These tests are described below. It is essential that you arrive at the laboratory well rested and in a fasted state. In order to ensure this you will need to refrain from strenuous exercise for 24 hours prior to each trial and should not smoke on the day of the trial nor consume alcohol or caffeine.

In the **incremental** test trial you will exercise on a cycling ergometer. During this test you will wear a mouthpiece with a valve attached (similar to a snorkel) to collect the air you have expired. This will enable us to measure your oxygen consumption during exercise. Your heart rate will also be monitored using a device strapped to your chest. The exercise intensity will be increased at regular intervals until you can no longer maintain the required workload.

The_**familiarisation** trial will attempt to duplicate the experimental trials to make you aware of the experimental protocol, with the exception that blood sampling will not take place. You will be required to cycle for 50 minutes at a set workload with expired gas collection occurring at regular intervals and therefore the mouthpiece will not be used for the majority of the exercise trial. For your well being, core temperature will be monitored through rectal thermometry. This is a very simple procedure and requires the insertion of a very small probe in the anus. After 50 minutes you will swap to another cycling ergometer and cycle for a further 15 minutes. During this ride you will be asked to produce as much work as possible.

The next three trials are the experimental trials. They will take place over three consecutive weeks. You therefore will be tested on the same day of the week and at the same time of the day. It is very important that a similar diet and exercise pattern are followed before these trials. Therefore subjects must refrain from strenuous exercise for the two (2) days prior to each trial and maintain their normal diet. You will be asked to report to the laboratory having refrained from tobacco, alcohol and caffeine for the 24 hours prior to the tests. Each subject upon coming to the laboratory for the first experimental trial will be asked to present a documented diary of the preceding 72 hours in which all exercise and dietary patterns will be recorded. This will be copied and returned with the instructions to follow the same patterns for subsequent trials. Thus you will be

able to continue normal training up to two days before each experimental trial.

On the night before an experimental trial you will be asked to commence fasting at either 8 or 10 P.M. On the morning of the trial you will ingest a normalised breakfast (provided) at either 6 or 8 A.M (These times depend upon your specific laboratory time). After ingesting the normalised breakfast you will rest until arriving at the laboratory 4 hours later.

During the experimental trials you will be asked to ingest a food substance 45 minutes before exercising. The exercise protocol will be the same as for the familiarisation trial, however blood sampling will take place at set intervals before and during exercise. Small blood samples will be obtained using an indwelling catheter in a forearm vein. The volume of blood collected is approximately one-sixth of that collected during a standard blood donation. The catheter consists of a needle and Teflon tubing. The tubing is fed over the top of the needle (which has punctured the vein) and into the vein. A tap (stopcock) is placed into the tubing so the flow of blood along the tubing can be altered at will. This procedure allows the taking of multiple blood samples without the need for multiple venepunctures (puncturing of the vein). Each time a blood sample is taken a small volume of sterile hepranised saline will be injected to clear the catheter and keep it patent. Catherterisation of subjects is slightly discomforting and can lead to the possibility of bruising and infection. In our experience this occurs very rarely. Catheterisation will be performed by Dr.Steve Selig who is qualified in this technique.

Do not hesitate to ask any questions and remember you are free to withdraw from the study at any time.

VICTORIA UNIVERSITY OF TECHNOLOGY

STANDARD CONSENT FORM FOR SUBJECTS INVOLVED IN EXPERIMENTS

CERTIFICATION BY SUBJECT

I,
of
certify that I have the legal ability to give valid consent and that I am voluntarily giving my consent to participate in the experiment entitled :

Pre-exercise carbohydrate ingestion and exercise performance : effect of glycemic index.

being conducted at Victoria University of Technology by :

Dr . Steve Selig
Dr. John Carlson
Mr. Matthew Sparks
Mr. Mark Febbraio

I certify that the objectives of the experiment, together with any risks to me associated with the procedures listed hereunder to be carried out in the experiment, have been fully explained to me by :

Mr. Matthew Sparks

and that I freely consent to participation involving the use on me of these procedures.

Procedures

1) Catherterisation
2) Incremental VO2 test
3) Submaximal exercise test / performance trial
4) Monitoring of core temperature by rectal thermometry.

I certify that I have had the opportunity to have my questions answered and that I understand that I can withdraw from the experiment at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the confidentiality of the information I provide will be safeguarded.

Signed : ....)
)
Witness other than the experimenter :) Date : ....
)
....)

CATHETERISATION

At specific intervals throughout the trials a small blood sample will be taken via a catheter placed into a forearm vein. The catheter consists of a needle and teflon tubing. The tubing is fed over the top of the needle (which has punctured the vein) and into the vein. The needle is then immediately withdrawn, leaving only the teflon tubing in your vein for the remainder of the experiment. A tap (stopcock) is placed into the tubing so the flow of blood along the tubing can be altered at will. This procedure allows the taking of multiple blood samples without the need for multiple venepunctures (puncturing of the vein). Each time a blood sample is taken, a small volume of sterile heparinised saline will be injected to clear the catheter and keep it patent. Catheterisation of subjects is slightly discomforting and can lead to the possibility of bruising and infection. The use of sterile, disposable catheters, syringes, swabs etc. will markedly reduce the possibility of infection caused by the catheterisation procedure. The use of qualified and experienced staff will reduce the likelihood of bruising as this is primarily caused by poor venepuncture techniques. Although the possibility of infection and significant bruising is quite small, if by chance it does eventuate, inform us immediately and then consult your doctor.

CARDIOVASCULAR RISK FACTOR QUESTIONNAIRE

In order to be eligible to participate in the experiment investigating

"Pre-exercise carbohydrate feeding: effect of Glycaemic Index"

you are required to complete the following questionnaire which is designed to assess the risk of you having a cardiovascular event occurring during an exhaustive exercise bout.

Name: ______________________________ **Date:** __________

Age: __________ years

Weight: ______________ kg **Height:** ______________ cms

Give a brief description of your average activity pattern in the past 2 months:

____

____

____

Circle the appropriate response to the following questions.

1. Are you overweight? Yes No Don't know
2. Do you smoke? Yes No Social
3. Does your family have a history of premature cardiovascular problems (eg. heart attack, stroke)? Yes No Don't know
4. Are you an asthmatic? Yes No Don't know
5. Are you a diabetic? Yes No Don't know
6. Do you have a high blood cholesterol level? Yes No Don't know
7. Do you have high blood pressure? Yes No Don't know
8. Are you on any medication? Yes No
   If so, what is the medication? ______________________________
9. Do you think you have any medical complaint or any other reason which you know of which you think may prevent you from participating in strenuous exercise? No
   Yes, please elaborate ______________________________

I, ______________________________, believe that the answers to these questions are true and correct.

Signed: ______________________

Date: __________

CATHETERISATION QUESTIONNAIRE

NAME: ____________________

AGE: ____________ years

1. Have you or your family suffered from any tendency to excessively bleed? (eg. haemophilia)

   Yes No Don't know

   If yes, please elaborate.... ____________________

   ____

2. Are you allergic to local anaesthetic?

   Yes No Don't know

   If yes, please elaborate.... ____________________

   ____

3. Do you have any skin allergies?

   Yes No Don't know

   If yes, please elaborate.... ____________________

   ____

4. Have you any allergies that should be made known?

   Yes No Don't know

   If yes, please elaborate.... ____________________

   ____

5. Are you currently on any medication?

   Yes No Don't know

   If yes, what is the medication? ____________________

   ____

6. Do you have any other medical problem that should be made known?

   Yes No

   If yes, please elaborate.... ____________________

   ____

7. Have you ever fainted when you have had an injection or blood sample taken?

   Yes No Never had it done

   If yes, please elaborate.... ____________________

To the best of my knowledge, the above questionnaire has been completed accurately and truthfully.

Signature ____________________

Date ____________________

FOOD AND EXERCISE DIARY

NAME:

TRIAL DATE:

2 DAYS BEFORE TRIAL

Day:
Date:

	FOOD/FLUID	EXERCISE
7am		
8am		
9am		
10am		
11am		NO
12noon		
1pm		STRENUOUS
2pm		EXERCISE
3pm		
4pm		
5pm		
6pm		
7pm		
8pm		
9pm		
10pm		
11pm		
midnight		

DAY BEFORE TRIAL

NO CAFFEINE
NO ALCOHOL CONSUMPTION
NO CIGARETTE SMOKING

Day:
Date:

	FOOD/FLUID	EXERCISE
7am		
8am		
9am		
10am		NO
11am		
12noon		
1pm		EXERCISE
2pm		
3pm		
4pm		
5pm		
6pm		
7pm		
8pm		
8.15PM DO NOT EAT AFTER THIS TIME		
9pm		
10pm		
11pm		

DAY OF THE TRIAL

NO CAFFEINE
NO ALCOHOL CONSUMPTION
NO CIGARETTE SMOKING

DRINK YOUR STANDARDISED BREAKFAST AT 6.15AM

REST UNTIL ARRIVING AT LABORATORY AT 10.55AM

You will be required at the laboratory until 1.30pm.

N.B For each experimental trial wear the same shoes and bike pants

Any problems on the day of the trial should be directed to me as early as possible. Before 7.30am for a morning trial or at the laboratory after 8.30am.

sincerely
Matt Sparks

Appendix B

Data from study

TREATMENT 1= CON
TREATMENT 2= HGI
TREATMENT 3= LGI

subj-ject	treat-ment	time	sample	gluc	La	NH3	Ins	FFA	VO2	RER	RPE	core temp	heart rate	CHO OX	work output
1.0	1.0	R1	1.0	4.7	1.5	38.3	5.2	0.6							
1.0	1.0	R2	2.0	4.8	1.3	42.1	4.3								
1.0	1.0	R3	3.0	4.7	1.2	67.8	3.4		2.58	0.78				0.0	
1.0	1.0	R4	4.0	4.9	1.5	23.4	6.9	0.4							
1.0	1.0	10.0	5.0	4.9	3.1	52.8	2.4	0.3							
1.0	1.0	15.0	6.0						52.19	0.86	16.0	37.0	166.0	2.7	
1.0	1.0	20.0	7.0	5.0	3.5	65.9	2.5	0.3							
1.0	1.0	30.0	8.0	5.1	3.3	66.9	2.8	0.5	52.67	0.85	17.0	37.5	171.0	2.5	
1.0	1.0	40.0	9.0	5.0	3.8	65.5	2.9	0.5							
1.0	1.0	45.0	10.0						53.70	0.84	18.0	37.6	170.0	2.4	
1.0	1.0	50.0	11.0	5.1	3.7	70.1	1.9	0.6							
1.0	1.0	55.0	12.0						62.98	0.96				5.3	-
1.0	1.0	60.0	13.0						54.22	0.88	20.0	37.8	180.0	3.1	
1.0	1.0	65.0	14.0	5.5	7.5	127.2	3.1	0.4	66.17	0.88			186.0	3.8	256.9
1.0	2.0	R1	1.0	4.4	1.0	39.3	7.5	0.5							
1.0	2.0	R2	2.0	7.2	1.3	38.3	65.0								
1.0	2.0	R3	3.0	5.3	1.4	32.7	35.1		6.73	0.97				0.6	
1.0	2.0	R4	4.0	3.9	1.4	34.1	21.2	0.1							
1.0	2.0	10.0	5.0	3.5	2.6	67.3	8.4	0.1							
1.0	2.0	15.0	6.0						42.93	0.94	13.0	36.6	169.0	3.3	
1.0	2.0	20.0	7.0	4.3	1.9	78.5	5.6	0.2							
1.0	2.0	30.0	8.0	4.8	2.2	72.0	5.6	0.2	47.53	0.92	15.0	37.6	172.0	3.4	
1.0	2.0	40.0	9.0	5.1	1.6	92.6	7.5	0.3							
1.0	2.0	45.0	10.0						48.43	0.92	14.0	37.9	170.0	3.4	
1.0	2.0	50.0	11.0	4.9	2.1	92.1	5.1	0.3							
1.0	2.0	55.0	12.0						57.63	0.96				4.9	
1.0	2.0	60.0	13.0						57.16	0.94	19.0	38.0	178.0	4.5	
1.0	2.0	65.0	14.0	4.9	5.5	145.9	5.0	0.3	55.44	1.02			186.0	5.8	229.3
1.0	3.0	R1	1.0	4.3	1.3	37.4	5.3	0.6							
1.0	3.0	R2	2.0	4.7	1.5	39.7	12.8								
1.0	3.0	R3	3.0	4.9	1.2	24.3	12.7		6.08	0.77				0.1	
1.0	3.0	R4	4.0	5.2	1.4	15.0	11.8	0.2							
1.0	3.0	10.0	5.0	4.5	3.2	60.3	4.4	0.1							
1.0	3.0	15.0	6.0						44.71	0.89	14.0	36.7	173.0	2.7	
1.0	3.0	20.0	7.0	5.3	3.8	74.8	4.8	0.3							
1.0	3.0	30.0	8.0	5.4	4.2	71.5	5.1	0.3	54.76	0.83	17.0	37.4	177.0	2.3	
1.0	3.0	40.0	9.0	5.7	4.0	82.3	5.4	0.3							
1.0	3.0	45.0	10.0						49.46	0.93	18.0	37.8	180.0	3.6	
1.0	3.0	50.0	11.0	6.1	4.6	87.0	6.4	0.4							
1.0	3.0	55.0	12.0						53.16	0.93				3.9	
1.0	3.0	60.0	13.0						52.04	0.92	20.0	38.1	182.0	3.6	
1.0	3.0	65.0	14.0	7.1	5.8	91.6	8.3	0.3	53.16	0.91			186.0	3.6	257.6
subj-ject	treat-ment	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core temp	Heart rate	CHO OX	work output
2.0	1.0	R1	1.0	4.9	1.2	16.8	1.6	0.4							
2.0	1.0	R2	2.0	5.1	1.3	15.0	2.5								
2.0	1.0	R3	3.0	4.7	1.7	13.1	1.1		5.94	0.68				0.0	
2.0	1.0	R4	4.0	5.1	1.8	14.0	2.7	0.5							
2.0	1.0	10.0	5.0	5.2	2.3			0.3							
2.0	1.0	15.0	6.0						32.84	0.87	10.0	38.1	134.0	1.8	
2.0	1.0	20.0	7.0	5.2	2.7	34.1	0.4	0.3							
2.0	1.0	30.0	8.0	5.2	2.7	50.5	0.4	0.3	36.63	0.76	13.0	38.5	129.0	0.8	
2.0	1.0	40.0	9.0	4.8	2.9	43.0		0.3							

2.0	1.0	45.0	10.0						44.78	0.86	15.0	38.5	140.0	2.3	
2.0	1.0	50.0	11.0	4.7	2.9	51.9	0.5	0.4							
2.0	1.0	55.0	12.0						53.99	0.92				3.7	
2.0	1.0	60.0	13.0						54.40	0.91	18.0	38.5	169.0	3.6	
2.0	1.0	65.0	14.0	6.3	10.5	159.0	0.1	0.3	57.48	0.97			177.0	5.0	240.0
2.0	2.0	R1	1.0	5.3	1.0	56.1	3.3	0.4							
2.0	2.0	R2	2.0	6.4	1.0	50.0	21.8								
2.0	2.0	R3	3.0	8.3	1.1	57.0	57.5		3.45	0.45				0.1	
2.0	2.0	R4	4.0	5.5	1.3	42.5	31.2	0.1							
2.0	2.0	10.0	5.0	3.2	3.0	52.4	9.6	0.0							
2.0	2.0	15.0	6.0						29.73	0.97	12.0	37.8	141.0	2.6	
2.0	2.0	20.0	7.0	3.9	2.8	50.5	2.6	0.1							
2.0	2.0	30.0	8.0	4.9	3.6	49.6	4.1	0.1	30.60	0.90	13.0	38.4	148.0	2.0	
2.0	2.0	40.0	9.0	5.2	2.7	58.4	3.2	0.1							
2.0	2.0	45.0	10.0						36.21	0.90	15.0	38.7	145.0	2.3	
2.0	2.0	50.0	11.0	5.4	2.6	59.8	2.4	0.1							
2.0	2.0	55.0	12.0						54.40	0.91				3.7	
2.0	2.0	60.0	13.0						48.05	0.94	17.0	39.2	168.0	3.7	
2.0	2.0	65.0	14.0	5.1	9.0	145.9	1.5	0.1	48.39	1.00			173.0	4.7	228.5
2.0	3.0	R1	1.0	4.8	1.2	32.3	2.1	0.4							
2.0	3.0	R2	2.0	5.4	1.4	41.1	10.0								
2.0	3.0	R3	3.0	5.9	1.4	36.9	13.8		4.06	0.69				0.0	
2.0	3.0	R4	4.0	5.9	1.4	62.2	12.1	0.2							
2.0	3.0	10.0	5.0	4.3	3.7	58.4	1.6	0.1							
2.0	3.0	15.0	6.0						38.18	0.85	10.0	37.7	130.0	1.8	
2.0	3.0	20.0	7.0	4.8	3.9	54.2	2.9	0.2							
2.0	3.0	30.0	8.0	5.4	3.2	54.2	3.7	0.3	42.38	0.81	12.0	37.5	141.0	1.5	
2.0	3.0	40.0	9.0	5.2	2.7	58.3	2.8	0.4							
2.0	3.0	45.0	10.0						41.21	0.80	12.0	37.3	141.0	1.3	
2.0	3.0	50.0	11.0	4.9	2.5	71.5	0.7	0.4							
2.0	3.0	55.0	12.0						52.66	0.92				3.8	
2.0	3.0	60.0	13.0						53.29	0.89	16.0	37.3	168.0	3.3	
2.0	3.0	65.0	14.0	5.5	9.3	126.2	0.5	0.3	53.17	0.89			173.0	3.3	227.1
subj-	treat-	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core	Heart	CHO	work
ject	ment											temp	rate	OX	output
3.0	1.0	R1	1.0	4.8	0.8	32.7	4.2	0.4							
3.0	1.0	R2	2.0	4.6	1.2	60.8									
3.0	1.0	R3	3.0	4.2	1.2	48.2	1.6		4.53	0.93				0.4	
3.0	1.0	R4	4.0	4.4	2.1	21.5	5.1	0.6							
3.0	1.0	10.0	5.0	5.5	4.9	34.1	5.0	1.2							
3.0	1.0	15.0	6.0						42.17	0.90	12.0	36.0	158.0	2.8	
3.0	1.0	20.0	7.0	5.9	7.5	32.3	6.4	0.4							
3.0	1.0	30.0	8.0	6.6	7.5	35.5	7.9	0.3	44.99	0.90	14.0	36.6	164.0	3.0	
3.0	1.0	40.0	9.0	7.1	8.2	42.5	7.1	0.3							
3.0	1.0	45.0	10.0						45.54	0.88	15.0	37.5	164.0	2.7	
3.0	1.0	50.0	11.0	6.9	8.7	42.6	6.8	0.3							
3.0	1.0	55.0	12.0						51.68	0.93				3.9	
3.0	1.0	60.0	13.0						50.72	0.95	14.0	38.5	162.0	4.1	
3.0	1.0	65.0	14.0	5.6	8.0	51.0	6.2	0.2	52.30	1.19			177.0	8.5	200.5
3.0	2.0	R1	1.0	4.7	0.8	44.4	3.3	0.2							
3.0	2.0	R2	2.0	5.5	0.7	28.1	11.5								
3.0	2.0	R3	3.0	5.6	0.8	23.8	14.9		6.31	0.92				0.5	
3.0	2.0	R4	4.0	5.0	1.1	17.8	14.4	0.1							
3.0	2.0	10.0	5.0	5.6	6.7	36.9	11.4	0.1							
3.0	2.0	15.0	6.0						48.91	0.96	13.0	36.8	161.0	4.3	
3.0	2.0	20.0	7.0	5.7	8.0	43.5	9.4	0.1							

3.0	2.0	30.0	8.0	7.6	9.0	48.2	11.0	0.2	49.07	0.93	15.0	37.7	168.0	3.8	
3.0	2.0	40.0	9.0	9.0	9.3	53.3	11.0	0.2							
3.0	2.0	45.0	10.0						51.85	0.93	16.0	38.5	165.0	3.9	
3.0	2.0	50.0	11.0	9.4	8.0	55.2	14.1	0.2							
3.0	2.0	55.0	12.0						44.79	0.88				2.7	
3.0	2.0	60.0	13.0						49.90	0.93	15.0	38.5	148.0	3.8	
3.0	2.0	65.0	14.0	8.3	7.3	39.7	18.3	0.2	57.05	1.04			177.0	6.5	214.0
3.0	3.0	R1	1.0	4.7	0.9	21.0	3.1	0.2							
3.0	3.0	R2	2.0	4.7	0.9	23.8	3.9								
3.0	3.0	R3	3.0	4.9	0.6	17.8	4.6		4.26	0.94				0.3	
3.0	3.0	R4	4.0	5.5	4.4	11.7	8.7	0.3							
3.0	3.0	10.0	5.0	5.3	8.0	39.7	8.8	0.3							
3.0	3.0	15.0	6.0						48.21	1.01	12.0	36.5	151.0	3.2	
3.0	3.0	20.0	7.0	5.9	10.3	60.8	6.7	0.2							
3.0	3.0	30.0	8.0	7.1	11.8	79.9	8.9	0.3	51.61	0.92	15.0	37.5	164.0	3.8	
3.0	3.0	40.0	9.0	7.5	13.6	83.7	9.5	0.3							
3.0	3.0	45.0	10.0						46.76	0.91	17.0	38.3	160.0	3.3	
3.0	3.0	50.0	11.0	8.2	10.8	85.6	10.9	0.3							
3.0	3.0	55.0	12.0						42.51	0.82				1.7	
3.0	3.0	60.0	13.0						45.98	0.87	15.0	38.5	147.0	2.6	
3.0	3.0	65.0	14.0	6.3	8.8	71.5	10.7	0.2	53.40	0.94			168.0	4.3	211.8
subj-	treat-	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core	Heart	CHO	work
ject	ment											temp	rate	OX	output
4.0	1.0	R1	1.0	5.0	1.4	25.7	4.8	0.7							
4.0	1.0	R2	2.0	4.9	1.7	25.7	6.9								
4.0	1.0	R3	3.0	5.1	1.7	14.0	5.3		4.15	0.79				0.1	
4.0	1.0	R4	4.0	5.0	1.9	26.2	4.9	0.5							
4.0	1.0	10.0	5.0	5.7	1.9	22.9	5.7	0.1							
4.0	1.0	15.0	6.0						38.79	0.81	9.0	36.6	149.0	1.4	
4.0	1.0	20.0	7.0	6.0	2.6	25.7	4.5	0.3							
4.0	1.0	30.0	8.0	6.0	2.3	35.5	5.0	0.3	39.43	0.79	8.0	37.0	135.0	1.2	
4.0	1.0	40.0	9.0	5.9	2.0	28.1	5.5	0.2							
4.0	1.0	45.0	10.0						38.28	0.83	12.0	38.2	145.0	1.7	
4.0	1.0	50.0	11.0	5.8	1.8	33.2	4.5	0.3							
4.0	1.0	55.0	12.0						60.46	0.93				4.7	
4.0	1.0	60.0	13.0						62.30	0.92	17.0	38.3	176.0	4.6	
4.0	1.0	65.0	14.0	6.4	13.3	105.1	3.1	0.1	66.48	1.00			182.0	6.8	308.5
4.0	2.0	R1	1.0	5.6	1.2	59.8	7.2	0.4							
4.0	2.0	R2	2.0	8.8	1.3	68.3	75.0								
4.0	2.0	R3	3.0	9.3	1.7	41.1	64.2		5.21	0.83				0.3	
4.0	2.0	R4	4.0	5.2	1.7	36.5	23.8	0.1							
4.0	2.0	10.0	5.0	4.9	1.5	37.9	11.9	0.1							
4.0	2.0	15.0	6.0						36.71	0.89	8.0	36.5	136.0	2.3	
4.0	2.0	20.0	7.0	5.1	1.7	46.8	14.0	0.2							
4.0	2.0	30.0	8.0	6.1	1.3	34.1	14.0	0.3	37.58	0.87	10.0	37.1	124.0	2.1	
4.0	2.0	40.0	9.0	6.5	1.2	39.7	12.2	0.3							
4.0	2.0	45.0	10.0						36.85	0.82	12.0	37.3	130.0	1.5	
4.0	2.0	50.0	11.0	6.0	1.3	41.6	8.3	0.1							
4.0	2.0	55.0	12.0						65.49	0.96				5.8	
4.0	2.0	60.0	13.0						62.56	0.94	17.0	37.8	178.0	5.1	
4.0	2.0	65.0	14.0	5.5	12.4	124.4	4.2	0.3	61.89	0.95			187.0	5.3	334.8
4.0	3.0	R1	1.0	5.1	1.1	33.2		0.5							
4.0	3.0	R2	2.0	5.8	0.9	40.2									
4.0	3.0	R3	3.0	6.6	1.1	35.1			4.31	0.73				0.0	
4.0	3.0	R4	4.0	6.1	1.3	36.5		0.1							
4.0	3.0	10.0	5.0	5.9	1.4	21.5		0.1							

4.0	3.0	15.0	6.0						34.77	0.82	8.0	36.8	130.0	1.4	
4.0	3.0	20.0	7.0	5.8	2.0	22.9		0.2							
4.0	3.0	30.0	8.0	6.1	1.6	22.4		0.3	36.27	0.79	9.0	37.5	131.0	1.1	
4.0	3.0	40.0	9.0	6.2	1.7	24.8		0.3							
4.0	3.0	45.0	10.0						35.12	0.82	11.0	38.0	134.0	1.4	
4.0	3.0	50.0	11.0	5.9	1.7	23.4		0.2							
4.0	3.0	55.0	12.0						61.24	0.89				3.9	
4.0	3.0	60.0	13.0						62.64	0.94	15.0	38.5	186.0	5.0	
4.0	3.0	65.0	14.0	6.9	13.6	87.0		0.1	63.18	0.96			196.0	5.1	270.0
subj-	treat-	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core	Heart	CHO	work
ject	ment											temp	rate	OX	output
5.0	1.0	R1	1.0	4.7	1.2	40.2	2.5	0.4							
5.0	1.0	R2	2.0	4.6	1.2	53.8	4.8								
5.0	1.0	R3	3.0	4.6	1.2	58.0	3.0		4.79	0.76				0.1	
5.0	1.0	R4	4.0	4.3	1.3	32.7	1.8	0.3							
5.0	1.0	10.0	5.0	4.6	2.0	58.4	1.7	0.2							
5.0	1.0	15.0	6.0						54.17	0.87	12.0		141.0	2.8	
5.0	1.0	20.0	7.0	4.6	1.9	51.0	1.5	0.2							
5.0	1.0	30.0	8.0	4.7	1.8	50.9	0.9	0.3	48.08	0.86	14.0		142.0	2.3	
5.0	1.0	40.0	9.0	4.9	1.4	55.6	1.3	0.3							
5.0	1.0	45.0	10.0						51.26	0.82	15.0		137.0	1.9	
5.0	1.0	50.0	11.0	4.6	1.9	66.4	1.4	0.3							
5.0	1.0	55.0	12.0						66.78	0.89				3.9	
5.0	1.0	60.0	13.0						63.26	0.90	17.0		165.0	3.9	
5.0	1.0	65.0	14.0	4.5	5.4	70.6		0.2	66.54	0.91			174.0	4.3	287.3
5.0	2.0	R1	1.0	5.2	1.2	36.5	3.7	0.3							
5.0	2.0	R2	2.0	8.6	1.4	33.2	48.4								
5.0	2.0	R3	3.0	10.6	1.4	44.4	57.8		6.74	0.73				0.1	
5.0	2.0	R4	4.0	6.7	1.8	23.8	36.9	0.1							
5.0	2.0	10.0	5.0	4.6	2.7	42.5	15.9	0.1							
5.0	2.0	15.0	6.0						56.23	0.80	13.0		145.0	1.7	
5.0	2.0	20.0	7.0	4.4	2.5	36.9	12.2	0.1							
5.0	2.0	30.0	8.0	5.0	2.1	41.6	10.6	0.1	55.20	0.83	14.0		143.0	2.2	
5.0	2.0	40.0	9.0	5.2	1.9	43.0	11.8	0.2							
5.0	2.0	45.0	10.0						51.02	0.82	14.0		144.0	1.9	
5.0	2.0	50.0	11.0	5.0	1.8	49.6	6.0	0.2							
5.0	2.0	55.0	12.0						79.87	0.79				2.2	
5.0	2.0	60.0	13.0						58.53	0.77	17.0		162.0	1.3	
5.0	2.0	65.0	14.0	3.5	5.1	65.9	2.3	0.2	76.75	0.83			171.0	3.1	235.2
5.0	3.0	R1	1.0	4.5	1.4	21.0	1.1	0.3							
5.0	3.0	R2	2.0	5.2	1.4	22.9	10.2								
5.0	3.0	R3	3.0	5.2	1.6	21.0	8.4		5.44	0.66				0.0	
5.0	3.0	R4	4.0	4.4	1.6	17.8	4.6	0.2							
5.0	3.0	10.0	5.0	4.6	2.3	47.2	1.4	0.1							
5.0	3.0	15.0	6.0						56.87	0.82	12.0		142.0	2.1	
5.0	3.0	20.0	7.0	5.2	2.1	47.1	1.8	0.2							
5.0	3.0	30.0	8.0	5.2	1.9	46.8	2.9	0.2	60.43	0.79	13.0		148.0	1.7	
5.0	3.0	40.0	9.0	5.4	2.1	51.4	2.0	0.2							
5.0	3.0	45.0	10.0						55.22	0.85	13.0		143.0	2.6	
5.0	3.0	50.0	11.0	5.2	2.1	57.5	1.3	0.2							
5.0	3.0	55.0	12.0						68.25	0.89				4.1	
5.0	3.0	60.0	13.0						70.69	0.90	15.0		169.0	4.5	
5.0	3.0	65.0	14.0	5.6	6.8	74.8	0.7	0.2	70.67	0.90			182.0	4.4	300.5
subj-	treat-	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core	Heart	CHO	work
ject	ment											temp	rate	OX	output
6.0	1.0	R1	1.0	4.9	1.0	40.2									

6.0	1.0	R2	2.0	4.9	0.9	48.2									
6.0	1.0	R3	3.0	4.8	1.1	58.0			2.37	0.79				0.1	
6.0	1.0	R4	4.0	4.8	1.4	******									
6.0	1.0	10.0	5.0	4.9	5.0	54.7									
6.0	1.0	15.0	6.0						53.33	0.84	12.0	37.7	166.0	2.5	
6.0	1.0	20.0	7.0	5.1	5.6	83.2									
6.0	1.0	30.0	8.0	5.3	5.3	83.7			49.53	0.79	13.0	38.5	165.0	1.5	
6.0	1.0	40.0	9.0	5.2	5.5	99.1									
6.0	1.0	45.0	10.0						49.96	0.80	14.0	39.1	168.0	1.7	
6.0	1.0	50.0	11.0	4.9	4.4	104.3									
6.0	1.0	55.0	12.0						62.64	0.90				4.3	
6.0	1.0	60.0	13.0						54.74	0.89	19.0	39.2	182.0	3.6	
6.0	1.0	65.0	14.0	6.0	12.3	225.3			60.98	0.89			185.0	4.0	276.6
6.0	2.0	R1	1.0	5.0	1.2	29.9	5.9								
6.0	2.0	R2	2.0	6.6	1.3	31.8	53.6								
6.0	2.0	R3	3.0	7.4	1.5	29.9	86.7		4.04	0.81				0.2	
6.0	2.0	R4	4.0	4.7	1.9	25.7	52.9								
6.0	2.0	10.0	5.0	3.6	7.3	56.1	18.8								
6.0	2.0	15.0	6.0						49.09	0.88	11.0	37.6	177.0	3.1	
6.0	2.0	20.0	7.0	4.3	7.1	83.7	10.3								
6.0	2.0	30.0	8.0	5.3	7.9	96.3	9.9		50.70	0.82	13.0	38.6	183.0	2.1	
6.0	2.0	40.0	9.0	6.4	8.6	108.5	8.7								
6.0	2.0	45.0	10.0						53.75	0.86	14.0	39.5	184.0	3.0	
6.0	2.0	50.0	11.0	6.2	8.6	149.6	7.6								
6.0	2.0	55.0	12.0						56.45	0.90				4.0	
6.0	2.0	60.0	13.0						55.88	0.89	17.0	39.2	181.0	3.7	
6.0	2.0	65.0	14.0	6.2	9.7	181.4	6.9		58.56	0.88			195.0	3.7	258.0
6.0	3.0	R1	1.0	5.2	1.0	44.9									
6.0	3.0	R2	2.0	5.5	1.0	42.5									
6.0	3.0	R3	3.0	6.1	1.1	43.5			2.02	1.45*				0.1	
6.0	3.0	R4	4.0	4.8	1.3	38.8									
6.0	3.0	10.0	5.0	3.7	3.7	60.8									
6.0	3.0	15.0	6.0						44.48	0.88	12.0	36.9	154.0	2.7	
6.0	3.0	20.0	7.0	4.3	4.5	73.4									
6.0	3.0	30.0	8.0	5.1	4.1	86.5			47.34	0.90	12.0	36.5	161.0	3.2	
6.0	3.0	40.0	9.0	5.2	4.1	89.8									
6.0	3.0	45.0	10.0						48.35	0.87	13.0	38.6	162.0	2.8	
6.0	3.0	50.0	11.0	5.3	3.9	87.9									
6.0	3.0	55.0	12.0						61.56	0.96				5.5	
6.0	3.0	60.0	13.0						52.17	0.94	20.0	38.8	183.0	4.3	
6.0	3.0	65.0	14.0	7.6	13.4	184.2			55.33	0.95			188.0	4.8	272.1
subj-ject	treat-ment	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core temp	Heart rate	CHO OX	work output
7.0	1.0	R1	1.0	5.1	0.9	19.2	6.3	1.0							
7.0	1.0	R2	2.0	4.9	0.8	18.7	5.3								
7.0	1.0	R3	3.0	5.1	0.7	15.0	4.1		6.67	0.80				0.2	
7.0	1.0	R4	4.0	5.2	0.9	14.0	5.5	0.8							
7.0	1.0	10.0	5.0	5.5	3.5	44.4	5.1	0.4							
7.0	1.0	15.0	6.0						41.50	0.93	14.0	37.2	145.0	3.0	
7.0	1.0	20.0	7.0	5.4	3.6	62.6	3.1	0.4							
7.0	1.0	30.0	8.0	5.7	2.2	65.5	4.1	0.6	43.12	0.85	15.0	37.4	148.0	2.0	
7.0	1.0	40.0	9.0	5.5	2.8	75.3	3.0	0.6							
7.0	1.0	45.0	10.0						42.34	0.86	15.0	37.6	147.0	2.1	
7.0	1.0	50.0	11.0	5.2	1.9	83.7	3.1	0.6							
7.0	1.0	55.0	12.0						48.54	0.89				2.9	
7.0	1.0	60.0	13.0						51.43	0.92	17.0	38.0	165.0	3.6	

7.0	1.0	65.0	14.0	4.9	6.5	113.1	1.9	0.3	55.86	1.01			176.0	5.5	230.1
7.0	2.0	R1	1.0	4.0	0.7	15.0	4.0	0.7							
7.0	2.0	R2	2.0	6.0	0.6	14.0	33.4								
7.0	2.0	R3	3.0	7.3	1.0	24.8	62.0		5.05	0.87				0.3	
7.0	2.0	R4	4.0	5.1	1.1	22.0	28.1	0.1							
7.0	2.0	10.0	5.0	3.6	4.6	43.0	10.9	0.0							
7.0	2.0	15.0	6.0						42.09	0.96	12.0	35.8	146.0	3.5	
7.0	2.0	20.0	7.0	3.3	3.9	49.1	6.6	0.1							
7.0	2.0	30.0	8.0	3.8	3.1	60.3	5.7	0.1	42.02	0.92	13.0	36.4	147.0	2.9	
7.0	2.0	40.0	9.0	4.2	2.6	53.3	4.8	0.1							
7.0	2.0	45.0	10.0						42.07	0.90	14.0	37.0	146.0	2.7	
7.0	2.0	50.0	11.0		3.0	57.5	4.8	0.1							
7.0	2.0	55.0	12.0	4.1					52.42	0.97				4.5	
7.0	2.0	60.0	13.0						55.35	1.00	17.0	37.5	160.0	5.3	
7.0	2.0	65.0	14.0	3.7	14.0	137.0	2.5	0.1	58.11	1.07			168.0	6.9	212.7
7.0	3.0	R1	1.0	4.7	0.3	19.3	4.0	0.5							
7.0	3.0	R2	2.0	4.9	0.6	18.5	6.2								
7.0	3.0	R3	3.0	5.3	0.4	28.0	12.0		6.23	0.88				0.4	
7.0	3.0	R4	4.0	5.2	1.0	26.5	8.2	0.1							
7.0	3.0	10.0	5.0	4.5	3.2	44.6	4.7	0.0							
7.0	3.0	15.0	6.0						41.61	0.91	13.0	37.6	141.0	2.8	
7.0	3.0	20.0	7.0	4.3	3.1	62.9	4.2	0.1							
7.0	3.0	30.0	8.0	4.5	2.4	70.0	5.4	0.1	41.29	0.92	14.0	38.4	144.0	2.8	
7.0	3.0	40.0	9.0	4.4	1.6	70.7	3.6	0.1							
7.0	3.0	45.0	10.0						39.88	0.90	15.0	38.6	142.0	2.5	
7.0	3.0	50.0	11.0	4.3	1.7	80.7	3.1	0.1							
7.0	3.0	55.0	12.0						44.26	0.95				3.5	
7.0	3.0	60.0	13.0						50.81	0.99	17.0	38.7	163.0	4.7	
7.0	3.0	65.0	14.0	4.1	5.4	128.9	2.5	0.1	50.41	1.00			176.0	4.8	232.1

subj-ject	treat-ment	time	sample	gluc	La	NH3	insulin	FFA	VO2	RER	RPE	core temp	Heart rate	CHO OX	work output
8.0	1.0	R1	1.0	4.3	1.0	31.3	3.1	0.4							
8.0	1.0	R2	2.0	4.3	1.0	25.2	5.9								
8.0	1.0	R3	3.0	4.0	1.3	35.1	3.9		4.73	0.82				0.2	
8.0	1.0	R4	4.0	4.2	0.9	22.9	3.7	0.3							
8.0	1.0	10.0	5.0	4.4	2.4	51.9	3.2	0.2							
8.0	1.0	15.0	6.0						43.04	0.88	11.0	37.0	139.0	2.4	
8.0	1.0	20.0	7.0	5.1	2.4	52.8	3.7	0.2							
8.0	1.0	30.0	8.0	5.3	2.4	67.5	3.8	0.2	45.18	0.87	12.0	37.6	145.0	2.3	
8.0	1.0	40.0	9.0	5.2	2.0	70.6	4.1	0.2							
8.0	1.0	45.0	10.0						44.32	0.86	12.0	38.0	142.0	1.2	
8.0	1.0	50.0	11.0	5.1	1.8	75.3	4.8	0.2							
8.0	1.0	55.0	12.0						55.27	0.92				3.8	
8.0	1.0	60.0	13.0						55.29	0.93	20.0	38.0	172.0	3.9	
8.0	1.0	65.0	14.0	7.1	11.6	160.8	4.9	0.1	52.86	0.93			172.0	3.7	231.3
8.0	2.0	R1	1.0	4.6	1.3	37.4	2.8	0.4							
8.0	2.0	R2	2.0	5.8	1.2	26.2	15.9								
8.0	2.0	R3	3.0	6.4	1.4	28.5	33.8		5.12	0.89				0.3	
8.0	2.0	R4	4.0	4.3	1.8	27.1	19.5	0.1							
8.0	2.0	10.0	5.0	3.2	2.7	42.1	8.6	0.1							
8.0	2.0	15.0	6.0						42.31	0.96	11.0	37.1	132.0	3.4	
8.0	2.0	20.0	7.0	3.3	2.0	45.8	5.4	0.1							
8.0	2.0	30.0	8.0	4.3	1.8	49.1	5.8	0.1	42.91	0.92	11.0	37.1	135.0	2.9	
8.0	2.0	40.0	9.0	4.2	1.7	58.9	4.7	0.1							
8.0	2.0	45.0	10.0						42.80	0.89	11.0	38.0	136.0	2.8	
8.0	2.0	50.0	11.0	4.3	1.6	55.2	3.6	0.2							

8.0	2.0	55.0	12.0						55.76	1.00				5.2	
8.0	2.0	60.0	13.0						56.11	0.98	20.0	38.1	167.0	4.9	
8.0	2.0	65.0	14.0	5.4	11.7	152.4	4.4	0.1	56.75	1.00			171.0	5.3	283.5
8.0	3.0	R1	1.0	4.6	1.1	26.6	3.2	0.7							
8.0	3.0	R2	2.0	5.7	0.9	28.1	8.0								
8.0	3.0	R3	3.0	5.5	1.0	24.3	8.8		4.99	0.79				0.2	
8.0	3.0	R4	4.0	4.2	0.5	24.8	8.5	0.2							
8.0	3.0	10.0	5.0	4.3	2.1	54.7	4.2	0.2							
8.0	3.0	15.0	6.0						47.74	0.81	10.0	37.4	138.0	1.6	
8.0	3.0	20.0	7.0	4.8	2.3	72.9	3.0	0.2							
8.0	3.0	30.0	8.0	5.0	1.8	79.0	3.9	0.2	45.87	0.85	11.0	38.0	140.0	2.2	
8.0	3.0	40.0	9.0	4.9	1.5	82.7	3.0	0.2							
8.0	3.0	45.0	10.0						45.25	0.85	10.0	38.5	136.0	2.1	
8.0	3.0	50.0	11.0	5.3	1.6	80.9	3.9	0.2							
8.0	3.0	55.0	12.0						58.67	0.92				4.1	
8.0	3.0	60.0	13.0						59.78	0.93	18.0	38.6	166.0	4.4	
8.0	3.0	65.0	14.0	6.9	10.9	213.2	4.6	0.1	57.53	0.91			172.0	3.8	250.2