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THE INFLUENCE OF DIET ON RECOVERY FROM PROLONGED EXERCISE

by

AMANDA J. PATTON

'A Master's Thesis'

submitted in partial fulfilment of
the requirements for the award of
Master of Philosophy of the Loughborough University of Technology

July 1985

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Endurance capacity has been shown to be related to the magnitude of pre-exercise muscle glycogen stores (Bergstrom, Hermansen, Hultman and Saltin 1967) and, moreover, fatigue during prolonged exercise is associated with a depletion of these stores (Saltin and Hermansen 1967). The rate of repletion of muscle glycogen and, therefore, recovery from prolonged exercise is most rapid when a high carbohydrate (CHO) diet is consumed (Costill and Miller 1980). The purpose of this study was to investigate the effect of a high CHO diet on recovery from exhaustive exercise using a performance test to assess the efficacy of such a diet. The CHO content of the diet was increased by supplementation of the normal diet with confectionery products. Confectionery products were used as these provide CHO in an "easy to eat" form when it is not possible to consume the more conventional high CHO foods.

The 20 individuals (10 males, 10 females) who volunteered to take part in this study performed two runs to exhaustion 3 days apart. Each run was performed on a motor driven treadmill at a speed equivalent to 70% of each individual's VO_{2} max. The subjects weighed and recorded their food intakes for a 7 day period, for 3 days prior to run 1 and for the 3 days of recovery between run 1 and run 2. A prescribed mixed diet similar to the individual's diet in terms of energy and CHO content was consumed for 3 days prior to run 1. After run 1 the subjects were divided into two groups i.e. a control group and a confectionery group. For the 3 days of recovery between run 1 and run 2 those subjects assigned to the control group were required to maintain their CHO intake whereas those assigned to the confectionery group were required to increase their CHO intake by supplementation with confectionery products. During run 2 each individual attempted to match or improve on the running time achieved during run 1.

The confectionery group achieved a 74% increase in CHO intake (265±45g vis 462±81g) whereas the control group showed a slight increase in CHO intake of 13% (295±87g vis 339±92g). The
confectionery group also showed a significant (p < 0.01) improvement in running time of 22.8% (114.5±15.6mins vis 140.6±27mins) compared to the control group which showed a small but non significant increase in running time during run 2 of 2.7% (119.2±19.5mins vis 122.4±22.4mins).

The results suggest, therefore, that recovery from prolonged exercise is facilitated by an increase in CHO intake which can be achieved by supplementation of the normal diet with confectionery products.
I would like to express my sincere thanks to all my colleagues in the Sports Science laboratory at Loughborough, especially my coworker John Brewer, for all their help and encouragement. My thanks also to Dr. Clyde Williams for his guidance and advice throughout the study and to MARS confectionery Ltd for financial support.

Finally I would like to express my gratitude to all the subjects who willing gave their time and energy and without whom the study would not have been possible.
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PUBLICATIONS

Part of the work presented in this thesis has been published in the following journals.


CHAPTER 1

INTRODUCTION

The effect of diet on athletic performance has been a subject of discussion for many years. Interest exists not only in maintenance of an adequate diet during training but also in manipulation of diet to enhance performance. Many dietary supplementations have been advocated to produce improvements in athletic performance and the most common misconception is that athletes require a larger than normal protein intake.

Evidence shows, however, that fat and carbohydrate (CHO) are the main metabolic fuels under normal conditions (Krogh and Lindhard 1920; Fritz, Davis, Holtrop and Dundee 1958; Christensen and Hansen 1939). Although fat stores in the body are greater than CHO stores and while fat provides more energy per unit weight than CHO, CHO is, nevertheless, the major source of energy during high intensity exercise (Ahlborg, Bergstrom, Eklund, Hultman and Maschio 1967; Hermansen, Hultman and Saltin 1967; Saltin and Karlsson 1971). The reason being that during high intensity exercise the rate rather than the capacity for energy production is paramount, and CHO metabolism provides energy at a much faster rate than metabolism of fat (McGilivery 1975). Carbohydrate metabolism also provides energy at a much lower oxygen cost than fat metabolism, which is also of major importance during high intensity prolonged exercise.

Muscle CHO (glycogen) stores have been shown to be depleted during prolonged exercise and, moreover, low glycogen stores have been associated with fatigue during cycling (Bergstrom et al 1967; Hultman 1967; Saltin and Hermansen 1967).

It has also been shown that endurance capacity is related to pre-exercise glycogen stores (Ahlborg et al 1967; Bergstrom and Hultman 1967; Christensen and Hansen 1939) which can be elevated by a regimen of diet and exercise (Bergstrom and Hultman 1967; Hultman
The combination of diet and exercise that results in maximum glycogen resynthesis is as follows: depletion of glycogen stores by exercise, followed by 3 days of a low CHO diet, followed finally by 3 days of a high CHO diet (Ahlborg et al 1967). The low CHO phase has unpleasant side effects, for example fatigue, irritability and nausea and so experiments have been carried out by Sherman and colleagues (1981) to investigate the possibility of glycogen repletion without this low CHO phase. Their results showed that glycogen repletion was as successful when the low CHO phase was omitted as when it was included.

The rate of glycogen repletion following prolonged exercise has been shown to be related to the amount of CHO consumed (Hultman 1967; Costill, Sherman, Fink, Maresh and Witten 1981). If a high CHO diet is consumed following prolonged exercise then glycogen resynthesis proceeds at a much faster rate than if a mixed or low CHO diet is followed (Bergstrom and Hultman 1967; Piehl 1974; Ivy, Sherman, Miller, Farrell and Frishberg 1982) returning to pre-exercise concentrations in between 24-46 hours (Piehl 1974; Piehl, Adolfsson and Nazar 1974). The mechanism for glycogen repletion is still unclear but appears to involve not only an adequate supply of substrate (glucose) but also an activation of the enzyme glycogen synthetase (Danforth 1965), an exercise induced permeability of the muscle to glucose (Young, Garthwaite, Bryan, & Holloszy 1983) and an adequate insulin concentration (Maehlum & Hermansen 1978).

The CHO content of the diet is normally increased prior to competition by consumption of high CHO foods such as pasta, bread, potatoes and rice. Such diets are very bulky and require advanced planning and preparation. The busy lifestyle of the athlete does not allow for such organisation and so supplementation of the diet with an "easy to eat" form of CHO would facilitate the consumption of a high CHO diet. Confectionery products could provide such a convenient supplementation for the occasions when the more traditional CHO meals are not available. Bearing in mind that pre-exercise muscle glycogen concentrations determine endurance capacity, the resynthesis of
glycogen to normal levels, following prolonged exercise is usually measured to determine recovery. For the athlete, recovery is the ability to participate fully in training or even another endurance event.

The aim of this study was to investigate the influence of high CHO diets on recovery from prolonged exercise not by determination of the restoration of energy stores but by the ability to reproduce a previous performance. Therefore, the experimental protocol required that all subjects performed two treadmill runs to exhaustion with only 3 days of recovery between run 1 and run 2. The increased CHO consumption was to be achieved by supplementation of the normal diet with confectionery products to investigate the possibility of using this type of convenience food for such purposes.

A comparison of the responses of a high CHO diet on prolonged exercise and recovery with a control diet requires careful prescription of the latter diet. Supplementation of the normal diet with confectionery products would increase the total energy intake of the subject’s diet as well as increasing the CHO intake. Therefore, the mixed diet consumed by the control group has to contain an equivalent amount of energy but with little increase in CHO content. The aim being to prescribe diets during the recovery period between run 1 and run 2 which are of similar energy content i.e. isoenergetic (isocaloric).

ORGANISATION OF THE THESIS

The thesis is divided into 3 experimental chapters. The first chapter (chapter 4) examines the dietary intakes of the male and female recreational runners who were subjects in this study and compares these with those of a group of male and female marathon runners.

The second chapter presents a system for dietary manipulation and investigates the efficacy of supplementing the diet with
confectionery products in order to achieve an increase in CHO intake.

This thesis is part of a collaborative study and although the overall study was to compare the effect of supplementation of the normal diet with both conventional high CHO foods and confectionery products with a control diet on recovery from prolonged exercise, only the performance results of the confectionery group and control group will be presented in this thesis. The remaining data is to be reported by my coworker.

Chapter 6 then, investigates the effect of exercise on metabolism and the effect of dietary supplementation with confectionery products on metabolism and recovery from prolonged exercise.
CHAPTER 2

REVIEW OF LITERATURE

2.1 ENERGY SOURCES FOR MUSCULAR EXERCISE

Prolonged physical activity results in an increase in the rate of mobilisation of fuels from their storage sites to the exercising muscle and an increase in the flux of metabolites through the energy producing pathways. The two main metabolic fuels are fat and carbohydrate (CHO), and their release and relative contributions to the energy production for muscular contraction are regulated by hormones and metabolites.

Originally it was thought that CHO was the exclusive fuel for exercise (Chaveau 1896). If fat was used at all by the muscle it was thought to be used indirectly by first being converted to ketone bodies in the liver (Gemmill 1942; Stadie 1945).

The first studies to investigate the relative contribution of fat and CHO to muscle metabolism made use of the fact that metabolism of fat and CHO utilises and produces different amounts of oxygen and carbon dioxide. The ratio of carbon dioxide to oxygen is known as the respiratory exchange ratio (R). An R value of 1 denotes exclusive oxidation of CHO whereas an R value of 0.7 denotes that only fat is being oxidised. If a mixture of fat and CHO is being used then an R value between these two figures will be obtained, the exact figure depending on the relative percentages being oxidised. Krogh and Lindhard (1920) obtained R values less than unity during exercise under various dietary conditions suggesting that fat as well as CHO was being used by the exercising muscle as a substrate for energy.

The advent of radioisotope techniques in the early 1950's made it possible to measure directly the utilisation of fat by the exercising muscle. This technique involved the infusion of Palmitate-1-C^{14} and
the determination of the amount of C\textsuperscript{14} in the carbon dioxide in the expired air. Many studies were performed using this technique both on tissue slices (Geyer, Matthews and Stare 1949; Fritz, Holtrop and Dundee 1958) and whole body (Fredrickson and Gordon 1958; Friedberg, Harlan, Trout and Estes 1960; Havel, Naimark and Borchgrevink 1963). In addition the development of the technique of catheterisation of working muscles made it possible to measure substrate uptake and release by tissues using arterio-venous differences (Keul, Doll and Keppler 1967; Jansson 1980) and using these techniques it was concluded that both fat and CHO were used directly by the muscle at rest and during exercise as metabolic fuels.

2.2 CARBOHYDRATE METABOLISM

Carbohydrate is stored in the liver and skeletal muscle as diffuses granules of glycogen, a highly branched glucose polymer. The glucose units of this polymer are connected to each other by glycosidic linkages between carbons 1 and 4. Branch points occur every 8 to 12 glucose residues and are formed by \( \alpha-1-6 \) bonding (Figure 2.1). The transfer of glucose residues onto the branch chains is catalysed by the enzyme glycogen synthetase (EC 2.4.1.11) while 1,4-\( \alpha- \) glucan branching enzyme is responsible for the formation of the branch points (Figure 2.2).

The liver contains about 10\% of its weight as glycogen (approximately 100g) whereas only 1-2\% of skeletal muscle weight is glycogen. It is more usual to express glycogen concentration in millimoles of glucosyl units (gu) per kilogramme of tissue (either wet or dry weight). Using this nomenclature, resting skeletal muscle contains 60-100 mmol gu kg\(^{-1} \) wwt and liver, after an overnight fast, contains 300 mmol gu kg\(^{-1} \) wwt.

The main function of glycogen in the liver is to provide glucose for the brain, which under normal conditions uses glucose exclusively as a metabolic fuel. If blood glucose levels fall below normal values
FIGURE 2.1 THE STRUCTURE OF TWO OUTER BRANCHES OF A GLYCOGEN MOLECULE.
FIGURE 2.2 STEPS IN THE FORMATION OF GLYCOGEN.
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then hypoglycaemic symptoms such as irritability, nausea and mental confusion result. Free glucose is formed in the liver from the dephosphorylation of glucose-6-phosphate (G6P) in the presence of the enzyme glucose-6-phosphatase (EC 3.1.3.9). Skeletal muscle does not contain this enzyme and so cannot form free glucose. Skeletal muscle, however, can contribute indirectly to blood glucose concentrations as several metabolites of glycogenolysis are transported from the exercising muscle to the liver where they are resynthesised to glycogen and thus free glucose if necessary.

In muscle the main responsibility of glycogen is to provide energy for muscle contraction. Glycogen can provide this energy under both aerobic (with oxygen) and anaerobic (without oxygen) conditions. In the absence of oxygen muscle glycogen can generate energy as adenosine triphosphate (ATP) by the process known as glycogenolysis (Figure 2.3). When oxygen is available the pyruvate formed by glycogenolysis can be decarboxylated to form acetyl CoA which can enter the tricarboxylic acid (TCA) cycle to generate additional molecules of ATP (Figure 2.4). The TCA cycle can only operate under aerobic conditions as it requires a supply of the cofactors NAD⁺ and FAD which can only be generated in the presence of oxygen.

2.3
FAT METABOLISM

Fat is stored as globules of triacylglycerols (TG); glycerol molecules esterified at each hydroxyl group with a fatty acid. There are many different types of TG, classified according to the identity and position of the three fatty acid components. Simple TG contain the same fatty acid on all three positions whereas mixed TG contain one or more different types of fatty acid. Natural fats are a mixture of both simple and complex TG and fat deposits within the body reflect, to some extent, the composition of ingested lipids.

Triacylglycerols are stored mainly in adipose tissue but are used
FIGURE 2.3  THEANAEROBIC DEGREDATION OF GLYCOGEN. 
FIGURE 2.4 THE TRICARBOXYLIC ACID (TCA) CYCLE.
by skeletal muscle and, therefore, need to be mobilised and transported in the blood. The hydrolysis of TG to free fatty acids (FFA) and glycerol occurs within the adipose tissue and the enzymes responsible (lipases) are under hormonal control. In this way the mobilisation of TG is regulated to suit the needs of the working muscle. The resulting FFA are insoluble and so are transported to skeletal muscle bound to a hydrophilic protein, albumin. Entry into the muscle cell is by simple diffusion, the rate being proportional to the concentration of FFA in the bloodstream (Armstrong, Steele, Altszuler and Bishop 1961).

Triacylglycerols are also found, to a limited extent, in the sarcoplasm of muscle cells. It has been suggested that these act either as a temporary store of FFA released from adipose tissue (Zierler 1976) or as a separate source of energy to cover the deficit between the total fat oxidised by the muscle and the amount released by the adipose tissue (Wahren 1973).

As an energy store TG have important advantages over CHO. Weight for weight pure TG can yield, on complete oxidation, nearly 2.3 times as much ATP as pure glycogen. The reason for this lies in the fact that TG are highly reduced so produce a greater number of hydrogen atoms per unit weight on oxidation than glycogen. These are used to drive the phosphorylation of ADP to ATP and so it follows that a greater yield of hydrogen atoms will produce more energy. In addition TG are stored in a pure form unlike glycogen which is stored in hydrated form. It has been estimated (Lehninger 1978) that if the 15 kg of TG in a normal 70 kg adult male were replaced by glycogen in a quantity sufficient to yield the same amount of ATP on combustion, his total body weight would have to be increased by 65 kg!

Why, therefore, when there is such a large quantity of TG stored in the body, which have obvious advantages, do working muscles use glycogen at all? The answer lies in the fact that fatty acids can only produce ATP under aerobic conditions. Glycogen on the other hand can produce ATP under both aerobic and anaerobic conditions.
Carbohydrate also provides energy at a much lower cost, 21 KJ (5.0 Kcals) per litre of oxygen per unit weight, whilst FFA provide 19.7 KJ (4.7 Kcals) per litre oxygen per unit weight (Lehninger 1978). Therefore, during high intensity exercise, when oxygen delivery to the muscle is a limiting factor, glycogen can provide the energy required. At low work loads, however, when oxygen delivery to the muscle is less of a problem, FFA play a major role in energy production and may contribute up to 80% of the energy required for contraction (Young, Pelligra and Adachi 1966; Therriault, Beller, Smoake and Hartley 1973).

The oxidation of fat and CHO by the muscle is closely integrated, rarely is one used to the exclusion of the other. If CHO alone is used for energy production high blood lactic acid concentrations result which limit exercise (Newsholme and Start 1973). If fat is used as the sole energy source, then only low intensity exercise can be performed (Hermansen, Hultman and Saltin 1967). At low exercise intensities plasma FFA concentrations are elevated and their rate of oxidation in the muscle increases (Costill, Coyle, Dalsky, Evans, Fink and Hoopes 1977). One of the intermediates of fatty acid oxidation, citrate, is a potent inhibitor of phosphofructokinase (PFK; EC 2.7.1.11), an enzyme of glycolysis (Randle, Garland, Hales and Newsholme 1963). Therefore, it follows that an increased oxidation of FFA causes a decrease in the production of ATP from glycogen thus sparing this energy source (Saltin and Karlsson 1971; Costill, Coyle, Dalsky, Evans, Fink and Hoopes 1977; Galbo, Holst and Christensen 1979). If, however, the exercise intensity increases and FFA are unable to produce ATP at a sufficient rate the ADP/ATP ratio increases and the inhibition of PFK is removed (Newsholme 1977), thus increasing the rate of flux through glycogenolysis. This increase in flux will cause an increase in blood lactate concentrations which is also known to inhibit lipolysis (Isssekutz, Miller and Rodahl 1966; Boyd, Giamber, Mager and Ledovitz 1979).

At the onset of exercise CHO is the obligatory fuel in order to provide the sudden increased requirement of ATP (Chapler and Stainsby
1968; Corsi, Midrio and Granata 1969). As exercise continues the aerobic contribution to the energy requirement increases as a result of an increase in the transport of oxygen to the exercising muscle. During prolonged exercise the concentration of glycogen in the muscle decreases which is accompanied by an increase in the oxidation of FFA to provide the energy required. Labile glucose is reserved mainly for use by extramuscular tissues such as the brain which are unable to use fat as an energy source. Nevertheless, during prolonged exercise at 50-70% VO\textsubscript{2} max blood glucose makes a significant contribution to the energy demands of the muscle. During exercise at this intensity (50-70% VO\textsubscript{2} max) glycogen stores are reduced with the resultant decline in blood glucose concentrations caused by the mismatching between hepatic output and muscle uptake (Felig, Cherif, Minagawa and Wahren 1982; Ahlborg and Felig 1982). At high (> 70% VO\textsubscript{2} max) exercise intensities, however, levels of insulin and the enzyme hexokinase (EC 2.7.1.1) decrease and so glucose uptake by the muscle is limited (Galbo, Richter, Hilsted, Holst, Christensen and Henriksson 1977).

2.4
LOW LEVELS OF GLYCOGEN LIMIT EXERCISE

Fatigue, the inability to maintain a given pace, is associated with a mismatching between the rate of ATP production and utilisation and is a result of various factors of which exercise intensity is one. During high intensity exercise fatigue is associated with an increase in hydrogen ions from lactic acid within the muscle which inhibit PFK and thus glycolysis (Sahlin 1978) whilst at work loads less than 65% VO\textsubscript{2} max, hypoglycaemia may be the cause of fatigue in subjects sensitive to a decrease in blood sugar levels (Pruett 1970a; Felig et al 1982). At exercise intensities greater than 65% VO\textsubscript{2} max several workers, using muscle biopsy techniques, have been able to show that there is a direct relationship between muscle glycogen content and exhaustion (Bergstrom and Hultman 1967; Hermansen, Hultman and Saltin 1967). The ability to exercise on a cycle ergometer is limited when glycogen levels in vastus lateralis are
very low. Indeed the rate of glycogen breakdown has been shown to be proportional to the relative exercise intensity (Hermansen 1977; Gollnick, Piehl and Saltin 1974) (Figure 2.5). At high work loads FFA alone are unable to produce ATP at a fast enough rate to maintain the pace as they require more oxygen than glycogen for a given ATP production. Therefore, when glycogen levels are low the runner can only carry on if he slows down to a pace where FFA can produce energy at a rate sufficient for contractile activity to be sustained (Pernow and Saltin 1971). Bergstrom and Hultman (1967) have shown that exercise intensitieis of 70-75% VO_2 max cannot be maintained when muscle glycogen levels are reduced to less than 25 mmoles gu kg^{-1}wwt. Such low levels have not been observed in running studies (Costill et al 1977) probably due to utilisation of a larger muscle mass and less specific fibre recruitment.

During cycling the degradation of glycogen has been shown to follow an exponential, probably triphasic pattern (Bergstrom and Hultman 1967) as shown in Figure 2.6. There is an initial rapid disappearance of glycogen followed by a constant fall and finally, in the last minutes of exercise, a slower disappearance. Bergstrom and Hultman (1967) have attributed the initial fast rate to the relative anoxia in the working muscle, the constant fall to stabilisation of metabolic processes and the last phase to an increase in glucose output from the liver and to an increase utilisation of fat. Depletion of glycogen does not occur in all types of fibres simultaneously. During exhaustive cycling depletion occurs first in slow twitch (oxidative) fibres (Type 1) and then in fast twitch (glycolytic) fibres (Type 11) as exercise progresses (Gollnick, Armstrong, Saubert, Sembrowich, Shepherd and Saltin 1973). This depletion pattern is also seen during running (Costill, Gollnick, Jansson, Saltin and Stein 1973) with Type 11 fibres depleted only after 2-3 hours of running. During cycling Type 11 fibre depletion has been shown to increase as exercise intensity increases (Gollnick, Piehl and Saltin 1974) and this may also be so during running. At exhaustion Type 1 fibres have been shown to be completely depleted of glycogen, whereas adequate amounts of glycogen can be seen in Type 11
FIGURE 2.5 THE PATTERN ON MUSCLE GLYCOGEN DEPLETION DURING CYCLING AT DIFFERENT EXERCISE INTENSITIES (% of VO2 MAX).
ADAPTED FROM SALTIN AND KARLSSON 1971
FIGURE 2.6  THE TRIPHASIC PATTERN OF GLYCOGEN DEPLETION.
ADAPTED FROM BERGSTROM AND HULTMAN 1967
fibres (Costill et al 1973; Gollnick et al 1973). Therefore, 
exhaustion may not be concomitant with low levels of glycogen in all 
fibres, but more reduction in those fibres that have to be recruited 
to support locomotion at that exercise intensity. In addition, the 
uptake of glucose and FFA by the depleted fibres may not be 
sufficient to synthesize ATP at a fast enough rate to produce the 
tension needed to maintain the required force for a given exercise 
intensity (Essen, Pernow, Gollnick and Saltin 1975). Therefore, if 
exercise is to continue the individual has to slow down to an 
intensity that can be supported by aerobic metabolism of FFA.

2.5
GLYCOGEN RESYNTHESIS

While glycogenolysis is rapid glycogenesis is relatively slow.
Hultman and coworkers (Hultman, Bergstrom and Roch-Norlund 1971) have 
reported values of 43 000 μmol gu/kg/min for glycogenolysis and 
400–500 μmol gu/kg/min for glycogenesis. This discrepancy is probably 
due to the different control mechanisms operating on the rate 
limiting enzymes of glycogen breakdown and synthesis (Cohen, Nimmo 
and Proud 1979). Glycogen synthesis like glycogen depletion does not 
occur at the same rate in all fibres. It has been demonstrated 
(Conlee, Hickson, Winder, Hagberg and Holloszy 1978; Piehl 1974) that 
following exhaustive exercise glycogen resynthesis takes place first 
in Type 1 fibres probably as a result of higher glycogen synthetase 
levels in these fibres (Conlee et al 1978) because of their 
dependence upon glycogen as a metabolic fuel.

Glycogen synthetase (EC 2.4.1.11) is the rate limiting step in 
glycogen synthesis. It is a tetrameric enzyme, each sub unit having a 
molecular weight of 88 000. Glycogen synthetase catalyses the 
transfer of the glucosyl moiety from the glucose carrier, UDPG 
(uridine-diphosphoglucose), to a pre-existing glycogen molecule. The 
enzyme exists in two interconvertible forms: an inactive 
phosphorylated D form and an active dephosphorylated I form (Friedman 
and Larner 1963). The D form is dependent on G6P whereas the I form
is independent of G6P (Villar-Palasi and Larner 1961). Synthesis of glycogen requires the enzyme to be in the dephosphorylated state or I form.

2.6
THE PHOSPHORYLATION OF GLYCOGEN SYNTHETASE: ENZYME AND HORMONAL FACTORS

At least three kinases are responsible for the conversion of glycogen synthetase I to the D form: Glycogen synthetase kinase 3 (Embi, Rylatt and Cohen 1980), protein kinase (Schlender, Wei and Villar Palasi 1969; Soderling, Hickenbottom, Reiman, Hunkeler, Walsh and Krebs 1970) and phosphorylase kinase (Embi, Rylatt and Cohen 1979). Glycogen synthetase kinase 3 (GSK 3) alone produces a greater inactivation of glycogen synthetase than either protein kinase or phosphorylase kinase alone, though the effects are additive with the greatest inactivation occurring when all three kinases are present together (Embi et al 1980). These kinases are under both neural and (or) hormonal control.

The key to this dual control of glycogen synthetase is phosphorylase kinase. This enzyme is activated by calcium ions and phosphorylation. Calcium ions are released from the sarcoplasmic reticulum during muscle contraction and, therefore, phosphorylase kinase is activated when muscles contract. Activation of phosphorylase kinase causes phosphorylation of many proteins among them glycogen synthetase (Embi et al 1979). Phosphorylation of glycogen synthetase produces the inactive D form thereby inhibiting glycogenesis so glycogenolysis can proceed to produce the ATP required for contraction.

Stimulation of muscle cells by adrenaline causes an activation of the enzyme adenylate cyclase. Adenylate cyclase catalyses the formation of cyclic adenosine monophosphate (cAMP) from ATP. Cyclic AMP promotes the dissociation of inactive protein kinase into an active catalytic subunit and a regulatory subunit. The active
catalytic subunit is responsible for the phosphorylation of many proteins among them glycogen synthetase and phosphorylase kinase. Thus hormonal stimulation activates protein kinase and phosphorylase kinase, the calcium ions for complete activation of phosphorylase kinase now coming from the calcium binding protein, calmodulin. Active protein kinase and phosphorylase kinase are now able to phosphorylate glycogen synthetase thereby reducing its activity. Adrenaline also increases the activity of GSK 3 by some unknown mechanism (Parker, Embi, Caudwell and Cohen 1982). Glycogen synthetase kinase 3 is also responsible for phosphorylation of glycogen synthetase. Figure 2.7 shows the processes responsible for phosphorylation of glycogen synthetase.

2.7
THE DEPHOSPHORYLATION OF GLYCOGEN SYNTHETASE: ENZYME AND HORMONAL FACTORS

While the events leading up to the inactivation of glycogen synthetase are fairly well understood the sequence of events responsible for the activation of the enzyme in muscle are far from clear. Insulin has been shown to promote glycogen synthesis by increasing the percentage of glycogen synthetase in the I form (Roach, Rossell-Perez and Larner 1977; Goldberg, Villar Palasi, Sasko and Larner 1967). Diabetics, lacking insulin, generally have a lower percentage of glycogen synthetase I than non diabetics (Adolfsson and Ahren 1971; Roch-Norlund 1972). The activation of glycogen synthetase by insulin is surprisingly not associated with a decrease in cAMP (Goldberg et al 1967). Although insulin levels are low during exercise there is an increase in its activity post exercise and so glycogen synthesis is stimulated. The activation of glycogen synthetase by insulin has recently been associated with a decrease in GSK 3 activity (Cohen 1982). Glycogen synthetase kinase 3, as previously described, phosphorylates glycogen synthetase. More surprisingly GSK 3 is also responsible in vitro, together with ATP and magnesium ions, for the activation of protein phosphatase 1 (Hemmings, Yellowlees, Kernochan and Cohen 1981) which
FIGURE 2.7. THE EVENTS LEADING UP TO THE PHOSPHORYLATION OF GLYCOGEN SYNTHETASE.
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deprophosphorylates phosphorylase kinase thereby causing its inactivation. Therefore, if GSK 3 were controlled in vivo by insulin then the action of GSK 3 on the activation of protein phosphatase 1 must require GSK 3 to be in a different form from that required for phosphorylation of glycogen synthetase.

Insulin does not use cAMP as a second messenger yet the interaction of insulin with its receptor causes the phosphorylation /dephosphorylation of many intracellular proteins. So far the second messenger has not been identified. However, it would need to activate a protein kinase that stimulates a phosphatase or vice versa to explain the phosphorylation/dephosphorylation caused by one stimulus.

Alternatively it is suggested that the insulin-receptor complex itself activates a protein kinase possibly by causing its dissociation from the plasma membrane (Denton, Brownsey and Belsham 1981). Each molecule of this protein kinase would then be able to phosphorylate many molecules of target protein thus allowing amplification of the stimulus without the need of a second messenger. Recently it has been demonstrated that the receptor itself becomes phosphorylated after interacting with insulin and it is thought that this phosphorylation may be an important step in the regulation of glycogen synthesis by insulin (Kasuga, Karlsson and Kahn 1982).

2.8 EFFECT OF EXERCISE ON GLYCOGEN SYNTHESIS

Using the one-legged cycling model glycogen depletion has also been shown to be necessary for glycogen synthesis (Bergstrom and Hultman 1966) as glycogen synthesis only takes place in the exercised leg. Recently Maehlum and Hermansen (1978) demonstrated that even if exhaustive exercise is followed by 12 hours of starvation glycogen resynthesis occurs albeit to a small extent. Low levels of muscle glycogen are such a potent stimulus for resynthesis that after exhaustive exercise when liver glycogen stores are also depleted
resynthesis of muscle glycogen predominates over liver glycogen resynthesis (Fell, McLane, Winder and Hollszy 1980). The stimulation of glycogen synthesis by glycogen depletion can be explained by the fact that glycogen synthetase I activity has been shown to be inversely proportional to glycogen concentration within the muscle (Danforth 1965; Huijung, Nuttall, Villar Palasi and Larner 1969).

Exercise also promotes an increase in permeability of muscle cells to glucose, the substrate for glycogen resynthesis, and other sugars (Goldstein, Mullick, Huddleston and Levine 1953; Adolfsson 1973; Young, Garthwaite, Bryan, Cartier and Holloszy 1983). Holloszy and Narahara (1965), using frog sartorius muscle, showed that the permeability of muscle membrane to tritiated 3-methylglucose (a non-metabolisable glucose analogue) was proportional to the frequency and duration of applied stimulus until a plateau was reached.

In normal conditions insulin is required for transport of glucose into cells. However, after intense muscular work the stimulus for glucose uptake is so potent that an increase in uptake is seen even in untreated diabetics (Ingle, Nezamis and Morley 1951) though to a lesser extent than non-diabetics (Berger, Haag and Ruderman 1975).

How, therefore, does exercise increase glucose uptake? Glucose is transported across the cell membrane in association with a mobile sugar transporter. Insulin and exercise increase the rate of glucose transport without increasing the apparent Km (binding capacity) suggesting that these stimuli increase the number of active sugar transporters in the membrane (Cheung, Conover, Regen, Whitfield and Morgan 1978). Goldstein (1961) proposed that exercising muscle releases a substance (muscular activity factor) into the blood which bathes the cell surface and causes an increase in glucose uptake. It is unlikely that such a humoral factor exists as glycogen resynthesis would take place in the rested leg after one legged exercise which is not the case (Bergstrom and Hultman 1966). Recently it has been suggested (Garthwaite and Holloszy 1982) that the calcium ions liberated from the sarcoplasmic reticulum during muscle contraction
initiate a reaction which leads to an increase in the rate of sugar transport. However, the concentration of calcium in muscle remains elevated for only a fraction of a second after stimulation whereas the increase in permeability to sugar occurs 10 to 20 minutes after stimulation and is still evident up to 5 hours later. Therefore, the reaction stimulated by calcium must be of much slower time course. It is also suggested that the reversal of this exercise induced increase in permeability requires protein synthesis. Indeed Garthwaite and Holloszy (1982) have shown that cyclohexamide and puromycin (inhibitors of protein synthesis) block the reversal of exercise induced permeability to 3-methylglucose.

It has been established that an increase in glucose transport reverses the exercise induced increase in permeability (Fell, Terblanche, Ivy, Young and Holloszy 1982; Young et al 1983). In the study by Young and colleagues (1983) rats swam for 2-3 hours after which some were deprived of food and sacrificed 60 minutes after exercise while the other rats were fed either a lard or Purina (60% CHO) diet for 18 hours. The rate of sugar transport was measured in the rats hind limbs and compared with non-exercised groups of rats fed the same diets. Exercise was shown to increase by threefold the transport of glucose in the hind limb muscle. Carbohydrate feeding decreased glucose transport 18 hours after exercise (98 mol/h) compared to fat fed rats (200 mol/h).

This reversal of exercise induced permeability is not thought to be related to glycogen content as exercised and non-exercised rats fed low CHO diets had similar muscle glycogen levels but different glucose uptake rates. Young and colleagues (1983) suggest that it is the increased glucose uptake by cho fed rats that reverses the exercise induced permeability not the increased glycogen content.

This could occur if loaded transporters within the membrane were more susceptible to immobilisation than empty transporters. Loaded transporters have been shown to move more rapidly than empty
transporters (Plagemann, Wohlheuter, Graff, Erbe and Wilkie 1981) and so would, therefore, be more likely to collide and interact with a transporter immobilising protein.

In summary, glycogen resynthesis following exhaustive exercise is promoted by several cellular and extracellular factors. Low glycogen levels stimulate synthesis by promoting the conversion of glycogen synthetase D to the I form by inhibition of the phosphorylase. Exercise itself increases the permeability of the muscle cell membrane by some unknown mechanism. Insulin also plays an important role by promoting the increase in permeability of the muscle cell membrane to glucose and activating the enzyme glycogen synthetase.

2.9 EFFECT OF DIET ON MUSCLE GLYCOGEN

In the first few hours following exhaustive exercise glycogen resynthesis is determined by cellular factors, thereafter substrate supply is limiting. Glucose is normally considered the major substrate for glycogen resynthesis. However, Hermansen and Vaage (1977) have demonstrated that after maximal exhaustive exercise at 90% VO2 max lactate is converted to glycogen within the muscle.

If subjects fast or consume a low CHO diet after exercise then glycogen resynthesis still takes place up to 4 hours post exercise (Costill, Craig, Fink and Katz 1982; Maehlum and Hermansen 1978; Hultman and Bergstrom 1967). Only a slight increase in glycogen synthesis is seen after 4 hours if little CHO is consumed and initial glycogen values are still not restored after one week on a fat and protein diet (Hultman and Bergstrom 1967). If CHO is included in the diet following exhaustive exercise then initial glycogen values are regained at a much faster rate and they can be exceeded (supercompensated) if a high CHO diet is consumed (Costill and Miller 1980; Ivy, Sherman, Miller, Farrell and Frishberg 1982). This procedure of consuming a high CHO diet following exhaustive exercise is often known as carbohydrate-loading. Bergstrom and Hultman (1966)
demonstrated that diet and depletion are necessary for glycogen resynthesis by consuming a high CHO diet for three days following exhaustive one-legged exercise. Glycogen resynthesis in the exercised leg was rapid reaching normal values within 24 hours. Thereafter, synthesis was slower but higher then normal values were attained in the exercised leg. No such results were seen in the rested leg. From these studies it appears that resynthesis of muscle glycogen follows a biphasic pattern and that repletion of glycogen to pre-exercise levels is rapid and is related to the severity of depletion. Further synthesis of glycogen in excess of pre-exercise levels is slow and is related to dietary CHO intake. Similar results have been obtained by other workers using two legged exercise (Piehl 1974; MacDougall, Ward, Sale, Sutton 1977) and it is generally concluded that muscle glycogen stores are restored to normal concentrations 24-46 hours following prolonged running if a high CHO diet is consumed.

2.19
METHODS TO ENHANCE MUSCLE GLYCOGEN STORES

The effect of diet on glycogen stores after exercise-induced depletion has also been investigated to determine the combination of diet and exercise that produces maximum glycogen resynthesis. Ahlborg and coworkers (1967) examined the effect of three different diets on glycogen resynthesis after exhaustive exercise. In Ahlborg's study one group (group A) consumed a high CHO diet (>90% CHO) for between 3 and 6 days, the other two groups (groups B and C) consumed a low CHO diet (<1% CHO) for 1 and 3 days respectively, exercised again, and then followed a high CHO diet for between 3 and 6 days. After 3 days of consuming a high CHO diet group C had a higher muscle glycogen repletion (192%) than groups B (182%) and A (164%) when compared to initial levels (100%). Higher values still (262%) were seen in subjects in group C who had consumed a high CHO diet for 7 days. From this study it appears that the glycogen repletion is enhanced if exhausted exercise is followed by a period of low CHO diet prior to a high CHO diet.
Because the low CHO phase is associated with several unpleasant symptoms e.g. nausea, dizziness and fatigue Sherman and colleagues (1981) have examined whether a series of depletion-taper runs combined with a low, though not too severe, CHO diet will result in supercompensation of muscle glycogen. Each depletion taper was performed at 73% VO2 max and consisted of runs of 90, 40, 40, 20, 20 minutes and a day of rest on the 6 days prior to a 13 mile performance run. One of three diets was consumed during the 6 days. One group (Group A) consumed a low CHO (15%) diet for three days followed by three days of a high CHO (70%) diet. Another group (Group B) consumed a mixed (50% CHO) diet for three days and then consumed a high CHO (70%) diet for the next three days. The third group (Group C) consumed a mixed (50% CHO) diet for the whole six days. Groups A resulted in the largest increase (121.6 mmoles kg-1 wet) in muscle glycogen prior to the performance run when compared to initial values. Groups B and C resulted in increases of 71.0 and 26.1 mmoles kg-1 wet respectively. This suggests that muscle glycogen stores can be elevated to high levels without the low CHO phase.

Such a dietary regimen may be very important during the competitive season when events are very close together and the individual needs to restore muscle glycogen levels as quickly as possible after a prolonged run in order to participate in another endurance event. Indeed Costill and colleagues (Costill, Bowers, Branam and Sparks 1971) have shown that exercise on successive days results in greater reliance on FFA to meet the energy requirements for running.

Several investigations have been carried out to determine the quantity of CHO required to achieve maximum glycogen repletion. Early cycling studies (Hultman 1967; Saltin and Hermansen 1967) showed a linear relationship between CHO ingestion and glycogen repletion. It has been suggested by Costill and coworkers (Costill, Sherman, Fink, Maresh, Witten and Miller 1981) that the relationship is in fact sigmoidal (Figure 2.8) and that consumption in excess of 600g CHO per
FIGURE 2.8 THE RELATIONSHIP BETWEEN THE AMOUNT OF CARBOHYDRATE CONSUMED AND GLYCOGEN REPLETION FOLLOWING EXHAUSTIVE EXERCISE.
ADAPTED FROM COSTILL ET AL 1981
day will not result in any further increase in glycogen synthesis. Another study (Blom, Vaage, Kardel and Hermansen 1980) has demonstrated that consuming 1.4 to 2.0 grammes of glucose per kg body weight every 2 hours for 8 hours after exhaustive exercise at 70% VO₂ max will not result in significantly greater resynthesis (4.6 and 5.6 mmole kg⁻¹·wt⁻¹ respectively). It has also been suggested that there is a relationship between frequency of feeding and glycogen storage during a high CHO diet. O'Dea and Puls (1979) have reported that nibbling-fed rats incorporated more glucose into muscle glycogen than meal-fed rats, however, no such relationship has been observed in humans (Costill et al 1981).

The mechanism for supercompensation of glycogen stores is still unknown. Insulin is thought to play some role as supercompensation does not take place in untreated diabetics (Chen, Iannuzzo and Williams 1977; Maehlum et al 1978). Glycogen synthetase I activity is inversely proportional to glycogen levels in the muscle (Danforth 1965; Bergstrom, Hultman and Roch-Norlund 1972) and so during supercompensation when glycogen levels are high glycogen synthetase I activity is low. Forms of glycogen synthetase intermediate, in extent of phosphorylation, to I and D forms have been shown to exist in vitro (Roach, Takeda and Larner 1976; Brown, Thompson and Mayer 1977) and in vivo (Kochan, Lamb, Lutz, Perrill, Reiman and Schlender 1979). These are sensitive to G6P and insulin and are present during supercompensation and so could explain the persistent glycogen synthesis in the absence of glycogen synthetase I.

2.11 ADVANTAGES AND DISADVANTAGES OF INCREASED MUSCLE GLYCOGEN STORES

As a result of this increase in the rate of glycogen resynthesis, high CHO diets following exercise also increase time to exhaustion during a subsequent bout of exercise (Christensen and Hansen 1939; Goforth, Hodgdon and Hilderbrand 1980; Bergstrom et al 1967). Glycogen depletion, as previously described, limits exercise at work
loads above 70% VO₂ max and so an increase in muscle glycogen above normal levels such as occurs following high CHO diets will delay fatigue (Ahlborg et al 1967; Bergstrom et al 1967; Pruett 1970a).

Furthermore, high glycogen levels could also help to combat the effects of dehydration during prolonged exercise (Sharman 1981).

Early work performed by McBride and colleagues (1941) on rat liver demonstrated that 2.7 grammes of water were associated with each grammme of stored glycogen. More recently Olsen and Saltin (1970) have reported values of 2-5 grammes of water per grammme of muscle glycogen stored in human subjects. It has been suggested (Sharman 1981) that this bound water is liberated as the glycogen is utilised and so may offset the effects of dehydration. It has also been suggested that that the additional water stored with glycogen may cause a sufficient increase in body weight as to be detrimental to performance (Brotherhood and Swanson 1979). Cureton (1978) has demonstrated that a 5% change in body weight can significantly increase the oxygen uptake of an individual at submaximal work loads.

However, consuming a high CHO diet does have its disadvantages. If glycogen levels are high then during exercise these will be used in preference to FFA as an energy source (Rennie and Johnson 1974; Williams, Maughan, Kelman, Campbell and Hepburn 1976). This increased utilisation has been attributed to the increase in plasma insulin concentrations associated with high CHO diets (Pruett 1970; Rennie and Johnson 1974) which will suppress FFA mobilisation (Hagenfeldt and Wahren 1971). Recently it has been suggested that the size of the glycogen store may influence its own rate of utilisation (Klausen and Sjøgaard 1980). If CHO are used preferentially this will lead to an increase in blood lactate level (Kelman, Maughan and Williams 1975; Galbo, Holst and Christensen 1979) which could limit endurance. In addition cardiac abnormalities following a high CHO diet have been reported (Mirkin and Spring 1973) which are thought to result from an increased glycogen synthesis in cardiac muscle.
2.12
REPORTS OF DIETARY MANIPULATION IN PREVIOUS STUDIES

Although many investigations have been carried out into the effect of dietary manipulation on endurance capacity and muscle glycogen stores, few have reported any details of the diets used or dietary compliance of the subjects. The usual dietary protocol for these studies is a period of a mixed normal diet, followed by a period on a low CHO diet and then a period on a high CHO diet. Exercise is performed after the mixed, low CHO and high CHO phases and the performances compared with each other.

Few studies have reported the composition of the mixed diet (Maughan et al 1978; Jette et al 1978; Rennie and Johnson 1974; Pruett 1971) and only two studies (Pruett 1971; Sherman et al 19819 made any attempt to control this dietary phase, the subjects in other studies eating ad libitum. There is little information in the literature about the diets consumed during the low and high CHO phases of the carbohydrate-loading diet. Some studies have provided the subjects with food composition lists (Rennie and Johnson 1974; Maughan et al 1978; Jette et al 1974) a few studies have fed subjects in the laboratory (Hultman and Bergstrom 1967; Pruett 1970) while other studies have reported no details of instructions given (Karlsson and Saltin 1971; Bergstrom et al 1967). If the purpose of the study is to investigate the effect of dietary manipulation on performance then it is imperative that all subjects achieve successful dietary manipulation. Wootton and colleagues (Wootton, Shorten and Williams 1981) have demonstrated that when given minimal dietary advice many individuals were unable to successfully manipulate their diet to achieve the desired increase in CHO intake. This suggests that in studies demanding such dietary manipulation nutritional support of the subjects is very important if the desired dietary goals are to be achieved.

Some workers have reported CHO intake on the low phase
contributing less than 1% of the energy intake (Bergstrom et al 1967; Ahlborg et al 1967; Karlsson ans Saltin 1971). While on the high phase CHO intakes contributing in excess of 90% of the energy intake have been reported (Ahlborg et al 1967; Hultman and Bergstrom 1967). Such diets as these are perfectly acceptable for the laboratory situation but if such procedures are to be used by the athletic population then more realistic diets need to be used.
CHAPTER 3

GENERAL METHODS

This chapter of the thesis is divided into 5 sections: 3.1 Dietary analysis, 3.2 Expired air collections, 3.3 Blood metabolite assays, 3.4 Running tests and 3.5 Data analysis.

3.1 DIETARY ANALYSIS

For all dietary data collection the weighed food intake method was used. The validity and reliability of this method has been reviewed by Marr (1971). This method does incur errors but nevertheless gives a fairly accurate representation of the food intake. Errors occur as a result of the subject rather than a trained investigator weighing the food and alteration of normal food consumption by the subject caused by inconvenience of weighing or to appear to consume a better diet. Food tables also add inaccuracies as they contain average nutrient values for each food but foods vary in nutrients due to season and cooking method. A reliability test on the 7d weighed food intake was performed by Adelson (1969) who found the greatest reliability for energy, fat and protein intake and the least for calcium and vitamins.

In the present study subjects were provided with a set of scales (Salter Ltd.) accurate to 10g with a re-zero function, a food diary in which to record the weight of foods eaten and an instruction sheet. Each subject was given a demonstration of the method to be used to record and weigh each food and drink item. Subjects were also asked to record the time when each meal or snack was consumed and any training performed each day. No formal appointment was made to see the subjects but they were encouraged to call into the laboratory should they encounter any problems.

The individuals records were coded according to the food composition tables of Paul and Southgate (1978). The composition of any processed food not found in these tables was obtained from the
manufacturers. The records were analysed using a computer program on a PDP 11/03 computer (Digital).

3.2
EXPIRED AIR COLLECTIONS

The standard Douglas bag technique was used to determine oxygen uptake and carbon dioxide production (Consolazio et al 1963 pp 16-21). A low resistance respiratory valve (Jakeman and Davies 1979) with a rubber mouth piece attached was connected to a 200 litre capacity Douglas bag with a two way tap by means of a low resistance light weight tubing (Falconia).

The percentages of oxygen and carbon dioxide in each sample were determined using a paramagnetic oxygen analyser (Sybron-Taylor Ltd., Model 570A) and an infra-red carbon dioxide analyser (Mines Safety Appliance Ltd., Lira Model 303). Both analysers were calibrated before and during each series of analyses with nitrogen, a calibration gas and atmospheric air. The volume of each air sample was determined by evacuating the contents of each Douglas bag through a dry gas meter (Parkinson Cowen Ltd.).

Oxygen uptake and carbon dioxide production were calculated using the Haldane transformation (Consolazio et al 1963 p 6-9). Carbohydrate and fat metabolism during exercise were calculated from respiratory data without measurement of urinary nitrogen (Consolazio et al 1963 p 314). These calculations, therefore, assumed that the contribution of protein to energy metabolism was negligible and that all carbon dioxide produced was from oxidative metabolism. The measurement of percentages of oxygen and carbon dioxide in each sample are described in Appendix A together with the relevant equations for calculation of oxygen uptake and carbon dioxide production.
3.3 BLOOD METABOLITE ASSAYS

Duplicate 25 μl capillary blood samples were collected where possible, deproteinised in 250 μl of perchloric acid (0.4 M) and centrifuged before storage at -20°C. Venous samples were obtained by venopuncture from an antecubital vein. The sample was transferred to a tube containing coagulant (Lithium heparin), spun in a refrigerated centrifuge (Koolspin, Burkard Scientific) and the plasma stored at -20°C. All samples obtained from an individual during an experiment were analysed within a single batch for purposes of standardisation. Therefore, all samples, except those for haemoglobin and haematocrit analysis, were stored at -20°C until the end of the study. Samples were analysed using a Locarte or Perkin Elmer (Model 110M) fluorimeter or an Eppendorf photometer (Model 1101M).

All assays were performed on duplicate samples. All enzymes used were supplied by Boehringer Corporation Ltd. and stored at 4°C. Buffer solutions and diluents were made in bulk and stored at room temperature. Fresh reaction mixture was made up and used for each batch. All glassware was cleaned by boiling in 50% w/w nitric acid.

Analysis for lactate was carried out using a modification of the fluorimetric assay described by Olsen (1971). Glucose concentrations were determined using the glucose oxidase method (Boehringer Corporation kit). Plasma free fatty acid (FFA) concentrations were determined using a modification of the method described by Chromy and colleagues (1977). Plasma glycerol concentrations were determined fluorimetrically using the method of Laurell and Tibling (1966). Plasma cholesterol concentrations were determined photometrically using a kit supplied by Boehringer Corporation Ltd. Haemoglobin (Hb) concentrations were determined using the cyanmethoglobin method (Boehringer Corporation Ltd.). Changes in plasma volume were estimated from Hb and haematocrit values as described by Dill and Costill (1974).
All metabolite assays are described in detail in Appendix B.

3.4
RUNNING TESTS

All running tests were performed on a motor driven treadmill (Quinton, Model 24-72).

3.4.1
MAXIMUM OXYGEN UPTAKE (VO₂ max) TEST

The subjects ran at a comfortable pace which varied according to the subjects ability. The grade of the treadmill was increased progressively from 3.5% by 2.5% every 3 minutes. Expired air samples were collected the last 60 seconds of each 3 minute period. Perceived rate of exertion (PRE) using the Borg scale (Borg 1973) was also recorded during this 60 seconds. Heart rates were measured on a Camtrace oscilloscope (Cambridge instruments Ltd.) throughout the test from 3 chest electrodes and recorded every 30 seconds. The subjects were requested to indicate clearly, by raising one finger, when they could exercise for only one minute longer. A final expired air collection was made for 60 seconds immediately after this signal had been given. The expired air collections were analysed for oxygen, carbon dioxide and volume.

3.4.2
SUBMAXIMAL HORIZONTAL RUNNING

For this test the method described by Taylor et al (1955) was used. Each subject ran continuously at 4 submaximal speeds with the treadmill in the horizontal position. Each speed was maintained for 4 minutes and an expired air collection was made for the last 60 seconds of each 4 minute work period. Heart rate was monitored continuously from 3 chest electrodes and displayed on a Camtrace oscilloscope. The results of this test enabled a regression equation
to be calculated from which speeds that would elicit work loads corresponding to 60% and 70% of maximum oxygen uptake could be derived.

3.4.3
ENDURANCE RUN

The endurance test required each subject to run for as long as possible on a level treadmill at a speed equivalent to 70% VO₂ max.

Preparation
For the 3 days prior to each run the subjects were asked to abstain from strenuous exercise and alcohol. Each subject arrived in the laboratory after an overnight fast of approximately 12 hours duration. The two endurance runs were performed at the same time of day. All subjects sat on a comfortable chair on the treadmill with their left hand in hot water. This procedure was adopted in order to increase peripheral blood flow, and so facilitate blood sampling. A 4 minute resting expired air collection was also obtained for the determination of pre-exercise VO₂. The hand was dried and resting capillary blood samples were taken. These were analysed for lactate, glucose and haemoglobin as outlined previously (section 3 of this chapter). A resting venous blood sample was also taken for analysis of haemoglobin, haematocrit, free fatty acids (FFA), glycerol and cholesterol as described in Appendix B. After the blood collections the subjects were allowed to perform stretching exercises and any last minute adjustments.

Running test
A 5 minute running warm up at 60% VO₂ max was then performed on the treadmill. The treadmill speed was then increased to that eliciting 70% VO₂ max and the subjects ran until they could no longer maintain the prescribed running speed ie they ran to exhaustion. Heart rate measurements were recorded throughout the test using chest electrodes and a Camtrace oscilloscope. The subjects had free access to water during the test, the time and volume of each intake was recorded. A
wet sponge was also freely available. Electric fans were used to cool the subjects when needed.

One minute expired air collections were taken in the last minute of the warm up, every 15 minutes during the test at 70% VO2 max and finally in the last 60 seconds of exercise. Capillary blood samples were obtained every 15 minutes during the first hour of exercise at 70% VO2 max, every 30 minutes thereafter and immediately after exercise. These samples were deproteinised then frozen and stored at -20°C prior to analysis for glucose and lactate as described. Venous samples were taken immediately before and after exercise, treated as previously described (section 3 of this chapter) for analysis of FFA, glycerol, cholesterol, haemoglobin and haematocrit. A schematic representation of the protocol is shown in Figure 3.1.

3.5 DATA ANALYSIS

A paired t test was used for correlated data to determine significance between trials. When data did not meet the assumptions of the t test a Mann Whitney U test was used to determine significant differences.
Dietary Manipulations and Running Tests

FIGURE 3.1 A SCHEMATIC REPRESENTATION OF THE PROTOCOL USED IN THIS STUDY
CHAPTER 4

AN ANALYSIS OF THE DIETS OF RECREATIONAL AND MARATHON RUNNERS

4.1 Introduction

Physical activity demands optimal functional capabilities of the individual. Correct nutrition is, therefore, important in order to maintain health and, therefore, enable the athlete to train and compete. Much controversy surrounds the question of what is the optimal diet for athletes and many supplements and foods have at one time or another been advocated as producing drastic increases in work and athletic performance. Diet alone cannot be expected to produce such improvements though it is justifiable to assume that deficiencies in diet could limit performance. The general consensus today is that the optimal diet for athletes differs little from the diets of less active individuals.

In an attempt to describe the energy intakes of active people the opportunity to examine the diets of a total of 47 individuals regularly engaged in running activities was taken. Some of these individuals were subjects in the experiment reported in this thesis while others were taking part in other experiments taking place in the laboratory.

The aim of this study was, therefore, to describe the dietary intakes of recreational and marathon runners and to compare these against the recommendations for a healthy diet as defined in the National Advisory Committee on Nutrition Education (NACNE) report.

4.2 Subjects

Twenty six males (20 recreational runners and 6 marathon runners) and 21 females (15 recreational runners and 6 marathon runners)
volunteered to take part in this study. The recreational runners trained at least 3 times a week. The male marathon runners were members of an athletic club with average best marathon times of 143.5 (±4.0) mins. The female marathon runners were all members of the British marathon squad.

4.3 Method

Each subject performed a 7 day weighed food intake using a technique similar to that of Marr (1971) and described previously (chapter 3 section 1).

4.4 Results

The results are expressed as a mean of the 7 day food intake.

The distribution of energy, fat, CHO and protein intakes are shown in Figure 4.1 for male subjects and Figure 4.2 for female subjects. No significant differences were found in any of the parameters between either male recreational and marathon runners or female recreational runners and marathon runners. Table 4.1 shows the average values for the above nutrients together with their percentage contribution to the energy intake.

The distribution of percentage contributions of the three major nutrients to the energy intake are illustrated in Figure 4.3 for male subjects and in Figure 4.4 for female subjects. No significant differences were found for any of the parameters between male recreational runners and marathon runners and female recreational runners and marathon runners.

Because of the small sample sizes of marathon runners only the diets of the recreational runners were examined further. All food groups represented in the results below are as according to Paul and Southgate (1978).
FIGURE 4.2 THE DISTRIBUTION OF MAJOR NUTRIENT INTAKES OF FEMALE RECREATIONAL (R) AND MARATHON (M) RUNNERS.
Figure 4.1. The distribution of major nutrient intakes of male recreational (R) and marathon runners.
Table 4.1 Daily intake of major nutrients of recreational runners (RR) and marathon runners (MR) (mean +SD)

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Males</th>
<th></th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR</td>
<td>MR</td>
<td>RR</td>
<td>MR</td>
<td>RR</td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td>Energy (MJ)</td>
<td>mean</td>
<td>13.2</td>
<td>12.9</td>
<td>8.2</td>
<td>8.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+SD</td>
<td>3.5</td>
<td>2.3</td>
<td>1.5</td>
<td>2.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat (g)</td>
<td>mean</td>
<td>124.2</td>
<td>108.0</td>
<td>77.3</td>
<td>85.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+SD</td>
<td>26.2</td>
<td>27.7</td>
<td>20.5</td>
<td>37.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO (g)</td>
<td>mean</td>
<td>377.5</td>
<td>438.2</td>
<td>240.0</td>
<td>241.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+SD</td>
<td>147.1</td>
<td>68.7</td>
<td>48.5</td>
<td>59.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(%)</td>
<td>45.1</td>
<td>53.3</td>
<td>44.5</td>
<td>49.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein (g)</td>
<td>mean</td>
<td>112.6</td>
<td>92.8</td>
<td>69.6</td>
<td>80.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+SD</td>
<td>32.8</td>
<td>21.6</td>
<td>15.2</td>
<td>17.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(%)</td>
<td>14.1</td>
<td>12.3</td>
<td>14.5</td>
<td>15.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+SD</td>
<td>1.8</td>
<td>2.7</td>
<td>1.9</td>
<td>3.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
FIGURE 4.3. THE PERCENTAGE CONTRIBUTION OF FAT, CARBOHYDRATE AND PROTEIN TO THE DAILY ENERGY INTAKE OF MALE RECREATIONAL (R) AND MARATHON (M) RUNNERS.
FIGURE 4.4. THE PERCENTAGE CONTRIBUTION OF FAT, CARBOHYDRATE AND PROTEIN TO THE DAILY ENERGY INTAKE OF FEMALE RECREATIONAL (R) AND MARATHON (M) RUNNERS.
Figure 4.5 shows the percentage energy intake derived from fat contained in various food groups for the two extreme percentage intakes for males and Figure 4.6 shows the corresponding data for females.

Figure 4.7 shows the percentage energy intake derived from protein contained in various food groups for the two extreme percentage contributions for males and Figure 4.8 shows the corresponding data for females.

Figure 4.9 shows the percentage energy intake derived from CHO contained in various food groups for the two extreme percentage intakes for males and Figure 4.10 shows the corresponding data for females.

Available CHO can be divided into simple and complex CHO. When the contributions of simple and complex CHO to the total CHO intake are investigated (Figure 4.11) it is shown that simple sugars contribute 42.1 (±9.6) % and 44.7 (±9) % to male and female subjects energy intakes respectively and complex CHO contribute 44.2 (±14.0) % and 43.8 (±10.9) % to male and female energy intakes respectively. These represent mean simple sugar (sucrose, fructose, mannose, maltose, lactose) intake of 154.0 (±42.7) g/d and 110.7 (±38.3) g/d for male and female subjects respectively. Mean complex CHO intake are 185.6 (±138.2) g/d and 105.6 (±28.5) g/d for male and female subjects respectively.

When examining simple sugar intakes the most important sugar to consider is sucrose. Sucrose is most widely found in foods such as soft drinks, confectionery, table sugar and biscuits and cakes. Figure 4.12 also illustrates the percentage contribution of these foods to the total CHO intake and the results show that on average 22.5 (±11.1) % of the male total CHO intake and 26.9 (±25.5) % of the females total CHO intake comes from these foods.

The distribution of daily fibre intakes is shown in Figure 4.13.
FIGURE 4.5. TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF FAT TO THE TOTAL DAILY ENERGY INTAKE OF MALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.

(BCBP= BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; P= PRODUCTS; VEG.= VEGETABLES; POTS= POTATOES; F.POTS= FRIED POTATOES IE. ROAST, CHIPS, CRISPS; CONF= CONFECTIONERY)
FIGURE 4.6. TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF FAT TO THE ENERGY INTAKE OF FEMALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.
(BCBP = BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; P = PRODUCTS; VEG. = VEGETABLES; POTS = POTATOES; F.POTS = FRIED POTATOES IE. ROAST, CHIPS, CRISPS; CONF = CONFECTIONERY)
FIGURE 4.7  TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF PROTEIN TO THE ENERGY INTAKE OF MALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.
(BCBP= BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; P= PRODUCTS; VEG.= VEGETABLES; POTS= POTATOES; F.POTS= FRIED POTATOES IE. ROAST, CHIPS, CRISPS; CONF= CONFECTIONERY)
FIGURE 4.8  TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF PROTEIN TO THE ENERGY INTAKE OF FEMALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.

(BCBP= BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; P= PRODUCTS; VEG.= VEGETABLES; POTS= POTATOES; F.POTS= FRIED POTATOES IE. ROAST, CHIPS, CRISPS; CONF= CONFECTIONERY)
FIGURE 4.9  TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF CARBOHYDRATE TO THE ENERGY INTAKE OF MALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.
(BCBP= BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; VEG.= VEGETABLES INCLUDING POTATOES; S&P= SUGARS & PRESERVES; CONF= CONFECTIONERY; S.DRINKS= SOFT DRINKS)
FIGURE 4.10 TWO EXTREME EXAMPLES OF THE PERCENTAGE CONTRIBUTION OF CARBOHYDRATE TO THE ENERGY INTAKE OF FEMALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY CONTRIBUTION FROM VARIOUS FOOD GROUPS.

(BCBP = BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS; VEG. = VEGETABLES INCLUDING POTATOES; S&P = SUGARS & PRESERVES; CONF = CONFECTIONERY; S.DRINKS = SOFT DRINKS)
FIGURE 4.11  CONTRIBUTION OF SIMPLE AND COMPLEX CARBOHYDRATES TO THE TOTAL DAILY CARBOHYDRATE INTAKE. VALUES ARE MEAN DAILY INTAKES FOR MALE AND FEMALE RECREATIONAL RUNNERS.
CONTRIBUTION OF BISCUITS, CAKES, BUNS AND PASTRIES AND PUDDINGS (BCBP), SUGARS AND PRESERVES (S&P), SOFT DRINKS (S.DRINKS) AND CONFECTIONERY TO THE TOTAL DAILY CARBOHYDRATE INTAKE.
FIGURE 4.13  DISTRIBUTION OF DAILY FIBRE INTAKES IN MALE AND FEMALE RECREATIONAL RUNNERS.

Legend
- Males
- Females
Average fibre intakes were 33.4 (±16.1) g/d and 23.5 (±8.7) g/d for male and female subjects respectively.

The contribution of various food groups to the two extremes of fibre intake for male and female subjects is shown in Figures 4.14.

4.5 DISCUSSION

Several investigators have stated that active individuals require a greater energy intake than sedentary individuals as a result of the fact that exercise not only increases the daily energy expenditure of the individual but also increases resting metabolic rate by as much as 10-15%... and may remain at this high level for several hours following exercise (Miller and Mumford 1960; Astrand and Rodahl 1977). The recommended daily intake (RDI) of energy for the general male population is 10.5 MJ and 9.0 MJ for females (DHSS 1979). The mean energy intake of males in this survey was well above this recommendation. The mean intake for females, however, falls well below the recommendation for females involved in most occupations (DHSS 1979). Only seven subjects attained this value and only one female had an energy intake that meets the recommendation of 10.5 MJ cited for very active individuals.

Most studies previously reported have been conducted on physical education students or groups of athletes training for a variety of events. To the authors knowledge no studies have reported dietary intakes of marathon runners per se. The energy intakes of the males in this study are similar to those reported in other studies (Wootton et al 1981; Kirsch and von Ameln 1981; Clement and Asmundson 1980). The values obtained for females are similar to those found by Clement and Asmundson (1980) but are lower than those usually reported (Barry et al 1981; Eastman 1979). The National Food Survey (NFS) records an average energy intake of 10.3 MJ/d which is very slightly lower than the 10.9 MJ obtained when the male and female data is combined.
FIGURE 4.14  EXTREME FIBRE INTAKES OF MALE AND FEMALE RECREATIONAL RUNNERS. VALUES ARE MEAN DAILY INTAKES.  
(BCBP= BISCUITS, CAKES, BUNS & PASTRIES, PUDDINGS;  
VEG.= VEGETABLES)
In view of the fact that the marathon runners are far more active than the recreational runners it is surprising that there were no differences in energy intakes. The female marathon runners especially had much lower than expected energy intakes. As none of the subjects was experiencing weight loss or fatigue it can be assumed that their energy intakes were adequate. The marathon runners may well have had fairly sedentary jobs which would have low energy costs thus allowing more energy to be available for running. Weighed food intakes do incur some errors due to inconvenience and inaccuracies in weighing but these would be unlikely to effect all individuals to such an extent as to cause such a difference in intake from those recommended and previously recorded.

There is no evidence to show that active individuals require a greater intake of fat than sedentary individuals. During steady state running at a low percentage of VO₂ max fat metabolism does provide a large proportion of the energy requirement (Havel et al 1961; Gollnick 1977). However, this should not be a problem as most male subjects would have a body fat in excess of 10% of his body weight and most females a body fat content in excess of 23% which would provide enough energy alone to run several marathons.

Several national and international committees have advised that fat intakes should be reduced to a level providing not more than 35% of the energy intake in order to reduce coronary heart disease (Royal College of Physicians and British Cardiac Society 1976, NACNE 1983). As energy intakes increase then so too will the amount of fat consumed to provide some of this extra energy but the percentage contribution of fat to the energy intake should still remain the same. Mean fat intakes of male and female subjects were very slightly higher than the recommended amount though lower than those found in other studies on active individuals (Barry et al 1981; Eastman 1979). When the extreme fat intakes of the recreational runners are examined it is noticed that the high intakes of fat of both male and female subjects is due to a disproportionate consumption of meat products the fat of which contributes 12.5% and 12.2% of the male and female's
energy intakes respectively. Those individuals with the lowest fat intake consumed more of their fat as visible fats than those individuals with high intakes. They had almost excluded other fatty foods from their diet. The decrease in mean fat intakes when compared to other studies is probably due to a greater awareness of the subjects to the health problems associated with high fat diets.

Do athletes require more protein than sedentary individuals? There are two possible mechanisms by which additional protein in the diet may aid performance. Firstly to enhance protein synthesis leading to increased muscle mass and secondly as an energy source for muscular contraction. Much controversy surrounds protein as an energy source though Lemon and Nagle (1981) state that although protein is clearly not as important as fat and carbohydrate as a fuel for metabolism, under some conditions protein may contribute significantly to the total energy expenditure. Protein supplementation, to aid the development of an increased muscle mass, is more likely to benefit strength related exercise than endurance exercise as the former may lead to an imbalance between the rate of protein synthesis and breakdown (Goodman and Ruderman 1982). Protein requirements may change as training continues and it has been suggested that during the early stages of training protein intakes may need to be increased in order to support increase production of, for example, myoglobin and erythrocytes though optimal intakes at this stage may still be as low as 1.2 g/kg/d (Haymes 1983). For athletes undergoing intense strength training it has been suggested that optimal intakes may be as high as 2.0 g/kg/d (Haymes 1983), although the needs of most athletes will be covered by a normal protein intake (Horstman 1972; Durnin 1978).

At present the recommended contribution to the daily energy intake by protein is 10%. Only one of the individuals in this study had a protein intake less than this figure. On examination of the diets of recreational runners the highest value for female subjects was due to the large contribution of milk and milk products to her daily diet. This food group was responsible for providing 22% of the daily energy
intake of which 8% was protein. Cereal products also contributed significantly to this individuals protein intake. As expected the lowest protein intakes were due to a relatively low consumption of high protein foods compared to other individuals. The male with the lowest intake was consuming a high proportion of his energy as biscuits, cakes and meat products which are relatively low in protein. The female with the lowest protein intake was consuming a high proportion of her energy as sugars and preserves which again are low in protein. The tendency towards a high protein diet has been shown by other studies on both athletes and non athletes (Eastman 1979; Wootton et al 1981; Ferro Luzi 1972). It has long been a myth of the athletic world that high protein intakes are necessary for optimal performance.

It is encouraging, therefore, that neither the recreational runners nor the marathon runners in this study were taking protein supplements nor admitted to consciously trying to increase their protein intakes in order to improve performance. The high protein intakes observed here are most likely to be a consequence of the nature of the British diet rather than due to any food fads.

There is no evidence to suggest that current protein intakes are too high or too low. A decrease in calcium retention is associated with high protein diets (Linkswiler 1976) which could have a negative effect on performance, for example cramp, which is associated with, among other causes, low calcium levels. However, this decrease in retention is probably counter balanced by the increased absorption of calcium accompanying high protein intakes. Moreover, a high percentage of energy from protein would mean lower percentages of energy from CHO and fat which could have adverse effects on performance. The healthy diet advocated by NACNE (1983) and DHSS (1979) would mean a larger proportion of protein coming from cereal and vegetable sources and less from animal sources which would be accompanied by CHO, thereby minimising the problem.

Considerable evidence supports the fact that increased consumption
of CHO by active individuals leads to improved endurance. Therefore, it would be advisable for active individuals to consume a higher intake of CHO than the 50% contribution to energy intake recommended for the general population (NACNE). As training intensity increases and especially prior to competition CHO consumption should increase. Mean CHO intakes for both male and female recreational runners in this survey are well below the NACNE recommendation of 50% of the energy intake. Low CHO intakes are common among athletes (Barry et al 1981; Wootton et al 1981; Jette 1978) despite evidence to suggest that increased CHO intakes are beneficial. The NFS reported CHO intakes providing 44.5% of the energy intake (excluding confectionery products and alcohol) which is also lower than that recommended.

Available CHO exists in two forms: simple sugars (maltose, sucrose, fructose) and starches. It is desirable that the main CHO source is complex rather than simple sugars. High CHO intake could be achieved by consuming mainly simple sugars, starches or a mixture of both as illustrated by the food intakes of the two high CHO consumers in this study. The male consumed 70% of his CHO intake as starches and dextrins mainly due to consumption of large quantities of cereal products, for example 1 kg of rice with a meal was not unusual. On the other hand 63% of the CHO consumed by the female with the highest CHO intake came from simple sugars which was due to the addition of sugar to drinks. Other sweet foods also contributed significantly to her diet and the results show that 52.9% of her CHO intake and 34% of her energy intake came from foods such as biscuits, confectionery products, soft drinks, sugars and preserves. This was far in excess of both the other individuals in this study and of the 20.8% contribution to the energy intake found by the NFS. High intakes of foods such as these would result in a high intake of sucrose which has been associated with dental caries (Newburn 1982). Moreover, such foods are "empty calories" virtually devoid of vitamins and minerals and so if they constitute a large proportion of the energy intake the individuals concerned could run the risk of becoming deficient in certain nutrients. Males tended to consume more of their CHO as biscuit and cakes etc than females. This increase was not associated
with a snacking pattern of food intake as they did not have an increased frequency of feeding. Therefore, this discrepancy is more likely to be due to either consumption of these foods at meal times or simply consumption of larger quantities at the times when the females are also consuming them. Low CHO intake are undesirable from an athletic and health point of view. Some individuals had very low intakes of CHO. These individuals had high percentage contribution of energy from fat and/or protein at the expense of CHO. Low CHO intakes (less than 40% of the energy intake) have been shown to pose problems for the athlete in training (Costill and Miller 1980) as a result of the low levels of muscle glycogen associated with such a diet. Several of the individuals in this study had CHO contributions to the energy intake of less than 40% which could limit their endurance capacity.

While low fibre intakes are not related to a decrease in performance they can be detrimental to health. Many diseases of the gastrointestinal tract common to the western world are related to a low fibre content of the diet. A high fibre intake produces bulky stools that are thought to result from the water retaining properties of fibre. Such diets also decrease transit time which could be beneficial as pathogens would be in contact with the gut for a shorter time and indeed some correlation has been found between low fibre intakes and some cancers (Royal College of Physicians 1981).

The recommended fibre intake is 30 g/d. Few subjects achieved this recommendation. The highest fibre intakes of the males and females were achieved due to a high consumption of cereals products, vegetables and fruit, all high in fibre. Those individuals with the lowest intakes of fibre were eating a larger proportion of refined foods which are low in fibre.

In summary, the analysis revealed that in terms of energy intake, there was no significant difference between the diets of recreational runners and those of marathon runners. Furthermore, the total energy intakes were relatively modest and within normal range which is
surprising when one considers their daily energy expenditure. Within the group of subjects there were examples of individual diets which had certain inadequacies, for example low CHO intakes, that may not only limit performance but could cause health problems in later life. Such inadequacies are not confined to the recreational runner but are also observed in club and national athletes both in this country and others (Ferro Luzzi et al 1972; Barry et al 1981; Stordy 1981). There appears, therefore, to be a need for greater nutritional education and monitoring of diets as part of the training regimen in order to maintain health and maximum performance.
CHAPTER 5

A SYSTEM FOR DIETARY MANIPULATION

5.1 Introduction

The traditional method used to increase an individual's dietary carbohydrate (CHO) intake is to prescribe the consumption of large quantities of high CHO foods such as pasta, potatoes and bread. Although nutritionally sound this type of diet requires regular meals and planning of meals in order to consume the prescribed quantity of CHO. The busy lifestyle of an athlete in training for endurance events together with the bulkiness of the diet often makes the traditional method far from the most practical way of achieving an increase in CHO intake. The traditional diet also requires nutritional knowledge to ensure that sufficient quantities of the correct foods are consumed in order that an increase in CHO intake will be achieved. Lack of sound dietary advice and knowledge may account for the fact that some athletes find such dietary modification ineffective at producing an increase in endurance (Wooton et al 1981). One short term solution to this problem is to supplement the normal diet with confectionery products which provide CHO in an "easy to eat" form. While accepting that confectionery products are not to be recommended as part of the long term habitual diet of active individuals, they can provide the necessary additional CHO when the more conventional high CHO foods are not available. Replacement of muscle glycogen stores is sometimes so important for the athlete that on certain occasions provision of the necessary CHO by consumption of confectionery products is justifiable.

The overall aim of this study, as mentioned earlier, was to evaluate the efficacy of high CHO diets on recovery from prolonged exercise, with particular reference to the use of confectionery products as a means of increasing the CHO intake. Three diets were to be examined in this study; a control mixed diet and two high CHO
diets. One high CHO diet was to be accomplished by supplementing the normal diet with confectionery products the other by consuming conventional carbohydrate-rich foods. Supplementation of the normal diet with confectionery products would not only increase the CHO content of the diet but would also increase the energy content of the diet. Therefore, the conventional high CHO diet had to be designed so that it was of similar energy content and CHO content to the confectionery diet, whilst the control diet had to be of similar energy content but without the increase in CHO.

Rather than prescribe diets that were unfamiliar to the subjects and which may, therefore, upset their preparation for the prolonged runs a dietary system was developed so that their normal diets could be modified to achieve the necessary dietary goals.

5.2 METHODS

Subjects
Thirty (15 males, 15 females) of the recreational runners whose diets are included in the results of the previous chapter volunteered to take part in the study.

Protocol
The study required the subjects to perform two runs to exhaustion (run 1 and run 2) three days apart. Each run was performed on a motor driven tradmill at a speed equivalent to 70% of each individual's VO₂ max. For three days prior to run 1 a prescribed mixed diet was consumed. Thereafter, the subjects were divided into three equal groups, each group consuming one of the three prescribed experimental diets ie. control, confectionery or traditional high CHO, for three days. After this period the subjects were required to run again (run 2) and to try to match or improve on their run 1 performance time.
Diets

Two confectionery products were used, their compositions are shown in Table 5.1.

Costill and colleagues (1981) have recently demonstrated that a daily CHO intake of 600g results in maximum glycogen repletion. For similar subjects in this study ie those consuming 12.6 MJ (3000 kcal) per day consumption of 600g of CHO would represent a 67% increase in CHO intake. It was decided, therefore, to attempt to increase the CHO intake of each individual, in the appropriate experimental groups, by approximately 70%. As the confectionery products were to be eaten in addition to the individuals normal diet this would necessitate an increase in energy intake as well as an increase in CHO intake. Knowing the composition of the confectionery products it was calculated that a 70% increase in CHO intake would be concomitant with a 40% increase in energy intake. In order to compare the influence of the confectionery diet with the control diet and the traditional diet the energy composition of the latter two had also to be increased by 40% to maintain isocaloricity with the confectionery diet. Therefore, the aim was to examine whether the flexible exchange system could be used to achieve high energy and high CHO diets.

Prior to the study each subject carried out a 7d weighed food intake as described previously (Chapter 3 section 1). This was carried out in order to assess the subjects normal energy and CHO intakes so that appropriate mixed and experimental diets could be prescribed.

All diets prescribed prior to runs 1 and 2 were based on food exchanges. An exchange is a dietary term used to describe foods that can be substituted for one another. In all 6 food exchange groups were used. The exchanges within each of the 6 food groups were approximately equal in energy and carbohydrate content. However, there was a certain amount of variation due to the large number of foods offered and so subjects were encouraged to choose a variety of foods within each food exchange group. Subjects were instructed to
<table>
<thead>
<tr>
<th>Product</th>
<th>ENERGY (KJ)</th>
<th>ENERGY (KCALS)</th>
<th>Fat (g)</th>
<th>CHO (g)</th>
<th>Protein (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Product1</td>
<td>1.9</td>
<td>459</td>
<td>19.53</td>
<td>70.16</td>
<td>4.91</td>
</tr>
<tr>
<td>Product2</td>
<td>1.7</td>
<td>413</td>
<td>8.1</td>
<td>90.6</td>
<td>0.43</td>
</tr>
</tbody>
</table>

Table 5.1. The nutritional composition of the confectionery products used (per 100g)
consume a certain number of exchanges from each food group, the exact number depending on their normal energy and CHO intake. The system of food exchanges is shown in Appendix C.

For three days prior to run 1 a mixed, prescribed diet was consumed. This contained approximately the same energy content and quantity of CHO per day as the average daily intake of the individual, which had been established from analysis of the 7 day weighed food intake.

For the three days between run 1 and run 2 those subjects assigned to the control group (Group 1) consumed a diet similar in quantity of CHO to the mixed diet prescribed before run 1 but with an increase in energy intake of 40%. This additional energy intake was provided by fat and protein (fat and meat exchanges). Those subjects assigned to the confectionery group (group 2) continued to consume the mixed diet prescribed prior to run 1 but supplemented their diet with an appropriate amount of confectionery to increase their CHO intake by 70% and their energy intake by 40%. Those subjects assigned to the high CHO group (group 3) consumed a diet containing an increase in CHO rich foods (bread and fruit exchanges) designed to provide an increase in CHO intake of 70% and a 40% increase in energy intake. All food and drink consumed before and during the experiments was recorded and weighed as previously described (Chapter 3 section 1).

5.2 Results

The mean daily energy and CHO intakes of the subjects for the three days prior to run 1 and for the three days prior to run 2 are shown in table 5.2. For comparison the results of the 7 day weighed food intake of the subjects are shown in table 5.3.

Energy intake

The 7 day weighed food intake resulted in an average daily energy
Table 5.2. Seven day weighed food intakes (mean daily intake ±SD)

<table>
<thead>
<tr>
<th></th>
<th>ENERGY</th>
<th>Fat</th>
<th>CHO</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>KJ</td>
<td>KCALS</td>
<td>g</td>
<td>%</td>
</tr>
<tr>
<td>Group1</td>
<td>mean</td>
<td>10.6</td>
<td>2533</td>
<td>110</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>2.8</td>
<td>662</td>
<td>28</td>
</tr>
<tr>
<td>Group2</td>
<td>mean</td>
<td>9.6</td>
<td>2289</td>
<td>87</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>3.7</td>
<td>889</td>
<td>38</td>
</tr>
<tr>
<td>Group3</td>
<td>mean</td>
<td>10.9</td>
<td>2596</td>
<td>104</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>2.7</td>
<td>636</td>
<td>33</td>
</tr>
</tbody>
</table>
Table 5.3. Mixed and experimental diets (mean daily intake ±SD)

<table>
<thead>
<tr>
<th></th>
<th>Run 1</th>
<th>Run 2</th>
<th>Run 1</th>
<th>Run 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>KJ</td>
<td>KCAL</td>
<td>KJ</td>
<td>KCAL</td>
</tr>
<tr>
<td>Group1 mean</td>
<td>12.5</td>
<td>2985</td>
<td>15.8</td>
<td>3763</td>
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<tr>
<td></td>
<td>±SD</td>
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<td>±SD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.8</td>
<td>435</td>
<td>3.9</td>
<td>923</td>
</tr>
<tr>
<td>Group2 mean</td>
<td>9.9</td>
<td>2347</td>
<td>15.0</td>
<td>3572</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
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<td>±SD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.2</td>
<td>292</td>
<td>2.6</td>
<td>615</td>
</tr>
<tr>
<td>Group3 mean</td>
<td>12.1</td>
<td>2874</td>
<td>14.4</td>
<td>3439</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td></td>
<td>±SD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.0</td>
<td>481</td>
<td>3.4</td>
<td>817</td>
</tr>
</tbody>
</table>

361 408
79  69
283 582
50  62
350 523
95  120
intake of 10.6 (±2.8) MJ, 9.6 (±3.7) MJ and 10.9 (±2.7) MJ for groups 1, 2 and 3 respectively. Prior to run 1 the average energy intakes for groups 1, 2 and 3 were 10.2 (±2.9) MJ, 9.0 (±1.4) MJ and 10.3 (±2.4) MJ respectively. The energy intakes prior to run 1 are not significantly different from those obtained during the 7 day weighed food intake. Prior to run 2 the average daily energy intakes were 13.5 (±4.3) MJ, 12.6 (±2.6) MJ and 13.9 (±2.7) MJ for groups 1, 2, and 3 respectively. These values represent average increases in daily energy intake of 32%, 41% and 34%.

Carbohydrate intake

Table 5.3 shows the mean CHO intakes in grammes of the subjects as determined by the 7 day weighed food intake. The mean values are 295 (±87) g, 279 (±106) g and 313 (±68) g for groups 1, 2, and 3 respectively. Prior to run 1 the mean CHO intakes of the respective groups are 300 (±85) g, 265 (±45) g and 301 (±86) g. The values obtained for daily CHO intakes prior to run 1 are not significantly different from those obtained during the 7 day weighed food intake. Prior to run 2 the CHO intakes of groups 1, 2 and 3 were 339 (±92) g, 462 (±81) g and 507 (±120) g respectively which represent average increases in daily CHO intakes of 13%, 74% and 68%.

5.3 Discussion

The purpose of this part of the study was to examine a method of modifying the individual's normal diet so that the CHO intake was increased by 70% and the energy intake was increased by 40%. The quantity of CHO i.e. 70% was chosen as a result of information provided by previous studies. The 40% increase in energy was dictated by the amount of confectionery required to achieve a 70% increase in CHO intake. As the effect of such a diet on recovery from prolonged exercise was to be investigated in another study, strict control of the individual's energy and CHO intakes had to be achieved. To this end a system of food exchanges was developed.
A high CHO diet can be described in two different ways. If the high CHO diet and the normal diet are to be isoenergetic then a high CHO diet can be described as one that contains a large percentage of its energy as CHO. If isocaloricity is not important then as long as the diet contains substantially more grammes of CHO than usual then the individual can be said to be consuming a high CHO diet. In this case unless the diet contains too little energy the actual intake is unimportant and the increase in CHO may well be accompanied by an increase in energy intake.

It has been suggested by Costill et al (1981) that 600g of CHO per day results in maximum glycogen repletion and that consuming more than this amount will not result in further glycogen synthesis. More than half of the subjects who took part in this study had an average daily energy intake of 2500 or less. If these individuals were to take in 600g of CHO per day while maintaining isocaloricity then foods containing at least 90% of their energy intake as CHO would need to be consumed! In real terms this would mean a daily diet of three cups of sugar and a knob of butter! Moreover, such an intake of CHO while maintaining isocaloricity would be impossible for those individuals whose energy intake was less than 2250 kcals per day. Such an intake could only be achieved if the individual were to vastly overfeed or consume nothing but sugar either of which could have serious consequences to health. Conversely some individuals regularly consume 600g of CHO during their normal diet. Does this mean that these individuals cannot carbohydrate load? This study attempted to overcome this problem by prescribing percentage increases in CHO and energy intake, as previously described, and, therefore, the actual amounts consumed varied according to the individuals normal intake.

Endurance capacity has been shown to be related to preceeding diet (Christensen and Hansen 1939, Ahlborg et al 1967). It was ,therefore, essential that the diet of each subject was as normal as possible in respect to energy and CHO content. From the results it can be seen
that prior to run 1 the energy and CHO intakes prescribed were similar to the subjects normal intakes and, therefore, no great changes in diet occurred. Prior to run 2 the two groups attempting to increase their energy intake by eating more food were less successful at achieving the 40% increase in energy intake required than the confectionery group. This was probably because as a large choice of foods was given there was a variation in energy content of foods within a food exchange group, therefore, as the number of exchanges to be eaten increased there was less chance of achieving the energy intake required. The confectionery group, however, were provided with a supplement with which to increase their energy intake and so fewer foods had to be consumed and consequently there was more chance of achieving the required energy increase. The two groups attempting to increase their CHO intake prior to run 2 were both successful. The subjects in the control group, however, also increased their CHO intake very slightly. The additional 40g CHO consumed per day is unlikley to have made a significant difference to muscle glycogen resynthesis especially when compared with the other groups whose CHO intake increased by approximately 70%. This slight increase was possibly due to an increased consumption of high CHO foods (eg. bread, potatoes, pasta) which were eaten with the prescribed extra fat and protein. Despite the slight deviations from the required mean increases, food exchange groups appear to be an effective method of controlling a subjects food intake. They provide the subject with a large choice of foods whilst allowing control to be kept on the subjects intake. For those subjects prescribed a large number of exchanges tighter control can be achieved if fewer choices are given or if certain food exchange groups are further sub-divided into high and low CHO or energy foods so that possible deviation from the mean requirement is eliminated.
6.1 Introduction

Rapid recovery from prolonged exercise is very important for the athlete in order that they can participate fully in training or another endurance event.

As previously mentioned fatigue during prolonged exercise is associated with low muscle glycogen concentrations (Bergstrom et al 1967; Hultman 1967) and that the duration of exercise is related to initial muscle glycogen concentrations (Gollnick, Pernow, Essen, Jansson and Saltin 1980). Therefore, following exhaustive exercise one of the main problems is to restore muscle glycogen levels as soon as possible. Several investigators have shown that the quickest way to recover muscle glycogen concentrations is to consume a high CHO diet for up to 46 hours following exercise (Piehl 1974; MacDougal et al 1977; Ivy et al 1982). Although recovery of the substrate for energy metabolism during exercise has taken place during this time, the obvious question is whether the individual as a whole has recovered and is able to perform prolonged exercise again.

The aim of the present study was to investigate whether supplementation of the diet with confectionery products following exhaustive exercise could facilitate recovery. Recovery was defined not as the restoration of normal muscle glycogen concentration, as in other studies, but as the ability to reproduce or even improve on the performance time achieved on a previous run.
6.2 METHODS

Twenty subjects, 10 males and 10 females, volunteered to take part in this study. A summary of the physiological characteristics of the subjects is shown in Table 6.1. All subjects were well trained recreational runners and were familiar with all pertinent laboratory procedures.

Each subject performed four running tests. Prior to each test the height and weight of each subject was measured and chest electrodes placed in position. The running tests were as follows.

i) Graded uphill running test to determine VO₂ max (Taylor et al 1955).

ii) A horizontal submaximal running test on a level treadmill to determine the speed that would elicit 70% VO₂ max for each individual.

iii) Two endurance tests three days apart which involved running to exhaustion at a speed equivalent to 70% of each individual's VO₂ max.

The procedures employed during these tests are as described in chapter 3 section 4.

Diets

For three days prior to run 1 a prescribed mixed diet was followed as described in chapter 5. For the three days between run 1 and run 2 the subjects consumed either a prescribed control diet containing the same quantity of CHO as the diet prior to run 1 or a high CHO diet in which the normal CHO intake was supplemented with confectionery products. Both these diets are described in detail in chapter 5.
Table 6.1 A summary of the physiological characteristics of the subjects
(mean ±SD)

<table>
<thead>
<tr>
<th></th>
<th>AGE (yrs)</th>
<th>HEIGHT (cm)</th>
<th>WEIGHT (kg)</th>
<th>V̄O₂ max (l.min⁻¹)</th>
<th>HR max (b.min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>25.9</td>
<td>176.9</td>
<td>70.8</td>
<td>4.20</td>
<td>59.6</td>
</tr>
<tr>
<td>±SD</td>
<td>7.8</td>
<td>7.4</td>
<td>8.5</td>
<td>0.50</td>
<td>5.4</td>
</tr>
<tr>
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<td>164.3-189.7</td>
<td>54.8-87.9</td>
<td>3.2-5.15</td>
<td>53-70.9</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>23.3</td>
<td>164.4</td>
<td>57.3</td>
<td>2.79</td>
<td>49.1</td>
</tr>
<tr>
<td>±SD</td>
<td>4.8</td>
<td>7.0</td>
<td>6.7</td>
<td>0.29</td>
<td>7.1</td>
</tr>
<tr>
<td>range</td>
<td>19-35</td>
<td>154.5-174.0</td>
<td>50.6-72.9</td>
<td>2.63-3.1</td>
<td>36.5-58.5</td>
</tr>
</tbody>
</table>


6.3
RESULTS

Laboratory Conditions

During run 1 the average laboratory temperature was 19.7 (± 2.4) °C and humidity was 59.5 (±8.3) %. During run 2 the average temperature was 19.5 (±2.5) °C and humidity was found to be 61.5 (± 6.5) %. Neither laboratory temperature nor humidity was significantly different during run 2 compared with run 1.

Body weight

No significant increase in body weight was incurred due to the increase in energy intake before run 2. Mean body weight was 57.86 (±5.77) kg prior to run 1 and 57.91 (±5.87) kg prior to run 2. A significant decrease (p < 0.005) in weight was observed after each run. The mean loss in body weight was 1.27 kg for run 1 and 1.3 kg for run 2.

Performance

The running times to exhaustion and distances covered by the two groups are shown in Figure 6.1 and Table 6.2. The control group showed a slight increase in run time of 2.7% whereas the group following the confectionery diet showed a significant increase in running time of 22.8% (p < 0.01). By way of comparison individuals consuming the traditional diet as described in chapter 5 also showed an improvement in running performance which was 25.7% (p < 0.01). From the data for run 2 it can be seen that the control group were able to match run 1 performance after three days of rest but without and increase in CHO intake. Eight of the 10 subjects in the control group increased their performance time on run 2 by an average of 6.25 (±3.2) minutes (range 2-12 minutes). One subject showed a decrease in endurance capacity of 4 minutes on run 2 whilst another subject showed no change in running time. On the other hand 9 of the group
**FIGURE 6.1**

RUNNING TIMES AND DISTANCES RUN TO EXHAUSTION. VALUES ARE MEANS ±SD OBTAINED FROM SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.

*+ significantly different from run 1 (p < 0.01)*
Table 6.2 Running times and distances run to exhaustion (Mean ±SD)

<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th></th>
<th>Run 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time</td>
<td>Distance</td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td>mins</td>
<td>miles</td>
<td>mins</td>
</tr>
<tr>
<td>CONTROL</td>
<td>mean</td>
<td>119.2</td>
<td>14.89</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>19.5</td>
<td>1.82</td>
</tr>
<tr>
<td>CONF.</td>
<td>mean</td>
<td>114.5</td>
<td>14.12</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>15.6</td>
<td>2.27</td>
</tr>
</tbody>
</table>

* Significantly different from run 1 values p<0.01
consuming the confectionery diet were able to improve on their run 1 performance by an average of 29 (+20.9) minutes (range 4-73 minutes). While one subject in this group matched but was not able to improve on his run 1 performance. None of the subjects in the confectionery group showed a decrease in endurance capacity.

**Fluid intakes**

Fluid intakes are shown in Figure 6.2. There was no significant difference in fluid intake in mls min⁻¹ after the high CHO diet when compared to intakes during run 1.

**Plasma volume**

Changes in plasma volume were calculated using the method described by Dill and Costill (1974) and the results are shown in Table 6.3.

Unless specified the following parameters are represented for the first 60 minutes of exercise and at exhaustion. Sixty minutes was chosen as this was the run time achieved by all subjects.

**Heart rates**

Figure 6.3 shows the heart rate response to exercise, in beats per minute, of both groups during run 1 and run 2. Heart rates were shown to increase during exercise in both the control and confectionery groups. Over the duration of run 1 the confectionery group showed an average increase of 15 (+12) b min⁻¹ whilst the control group showed an increase in heart rate of 12 (+7) b min⁻¹. During run 2 the confectionery group showed an increase in heart rate of 15 (+9) b min⁻¹ and the control group showed an increase of 10 (+9) b min⁻¹. These increases in heart rate during exercise were not significant for either group.
FIGURE 6.2 FLUID INTAKES DURING THE TWO RUNS TO EXHAUSTION. VALUES ARE MEANS OBTAINED FROM SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.
Table 6.3  Effect of exercise on plasma volume (Mean % change ±SD)

<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th>RUN 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL</td>
<td>mean</td>
<td>-8.3</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>5.5</td>
</tr>
<tr>
<td>CONFECTIONERY</td>
<td>mean</td>
<td>-3.7</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>3.5</td>
</tr>
</tbody>
</table>
FIGURE 6.3  HEART RATES DURING THE TWO RUNS TO EXHAUSTION. VALUES ARE MEANS ±SD OBTAINED FROM SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.
The contribution of fat and CHO to the energy metabolism

The average R values obtained at 15 minute periods during each run are shown in Figure 6.4. Figure 6.5 shows the rate of CHO and fat utilisation and the percentage contribution of these substrates to the energy metabolism during run 1 and run 2. Table 6.4 shows the total energy utilisation and the energy utilisation for the first 60 minutes of exercise together with values for the rate of CHO and fat utilisation.

There was no significant difference in the quantity of fat and CHO used per minute by the control group during the first 60 minutes of run 2 compared to the first 60 minutes of run 1. The confectionery group, however, used significantly more CHO per minute \( (p<0.025) \) during the first 60 minutes of run 2 when compared to the same time interval for run 1. This increase in CHO utilisation was accompanied by a significant decrease in fat utilisation \( (p<0.025) \) and so the energy utilisation over this time period remained the same as run 1.

Carbohydrate metabolism

Blood lactate concentrations at rest and during exercise are shown in Figure 6.6. No significant difference was found in blood lactate levels after either the control diet or the confectionery diet when compared to those obtained after a mixed diet.

Figure 6.7 shows the blood glucose concentrations at rest and during exercise. No significant difference was found in blood glucose concentrations either at rest or during exercise after either the control diet or the confectionery diet when compared to those obtained after a mixed diet.
FIGURE 6.4  R VALUES DURING THE TWO RUNS TO EXHAUSTION. VALUES ARE MEANS ± SD OBTAINED FROM THE SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.

* significantly different from run 1 (p < 0.01)
FIGURE 6.5  CARBOHYDRATE AND FAT UTILISATION AND CONTRIBUTION TO THE TOTAL ENERGY EXPENDITURE DURING THE TWO EXHAUSTIVE RUNS. VALUES ARE MEANS ±SD OBTAINED FROM THE SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.

+ significantly different from run 1 (p < 0.025)
Table 6.4 Energy expenditure and substrate utilisation during exhaustive running (mean ±SD)

<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th></th>
<th>RUN 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CHO</td>
<td>fat</td>
<td>kcals</td>
<td>g/min</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>1.95</td>
<td>0.41</td>
<td>1364</td>
<td>696</td>
</tr>
<tr>
<td>±SD</td>
<td>0.63</td>
<td>0.1</td>
<td>285.5</td>
<td>171.8</td>
</tr>
<tr>
<td>Confectionery</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>1.75</td>
<td>0.48</td>
<td>1329</td>
<td>692</td>
</tr>
<tr>
<td>±SD</td>
<td>0.44</td>
<td>0.22</td>
<td>300.2</td>
<td>176.9</td>
</tr>
</tbody>
</table>

* significantly different from run 1 (p<0.025)
FIGURE 6.6  BLOOD LACTATE CONCENTRATIONS DURING THE TWO EXHAUSTIVE RUNS. VALUES ARE MEANS ±SD OBTAINED FROM THE SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.
FIGURE 6.7  BLOOD GLUCOSE CONCENTRATIONS DURING THE TWO EXHAUSTIVE RUNS. VALUES ARE MEANS ± SD OBTAINED FROM THE SUBJECTS IN CONTROL AND CONFECTIONERY GROUPS.
Fat metabolism

Cholesterol Concentrations

The results obtained for plasma cholesterol concentrations of male and female subjects during run 1 are shown in Table 6.5a. Average resting plasma cholesterol concentrations for male and female subjects were found to be 3.34 (±0.75) mM and 3.9 (±0.64) mM respectively. No significant difference in cholesterol concentrations of the male subjects was observed due to an acute bout of exercise, the female subjects, however, showed a significant decrease in plasma cholesterol concentrations (p<0.05). Table 6.5b shows the pre and post run 1 and run 2 plasma cholesterol results obtained for the control and confectionery groups. Dietary intervention was not shown to cause any significant difference in plasma cholesterol concentrations.

FFA Concentrations

Table 6.6 contains the average values for plasma FFA concentrations for the confectionery and control groups prior to and post each run. The FFA concentrations are significantly different post exercise when compared to pre exercise values for both groups. Dietary intervention did not cause a significant change in the plasma FFA concentrations of either groups.

Glycerol Concentrations

Average glycerol concentrations pre and post both runs are shown in Table 6.7. Post exercise glycerol concentrations are significantly different from pre exercise concentrations in both groups for both periods of exercise. When the pre exercise concentrations are examined it is seen that the plasma glycerol concentrations were unaffected by dietary intervention.
Table 6.5a  Effect of exercise (run 1) on plasma cholesterol concentrations mM (mean ±SD)

<table>
<thead>
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<tr>
<td>MALES</td>
<td>mean 3.34</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>±SD 0.75</td>
<td>1.52</td>
</tr>
<tr>
<td>FEMALES</td>
<td>mean 3.9</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>±SD 0.64</td>
<td>0.78</td>
</tr>
</tbody>
</table>

* significantly different from pre exercise value (p<0.05)
Table 6.5b  Effect of diet on plasma cholesterol concentration mM
(Mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th>RUN 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
</tr>
<tr>
<td>CONTROL</td>
<td>mean 3.65</td>
<td>4.41</td>
</tr>
<tr>
<td></td>
<td>±SD 0.93</td>
<td>1.10</td>
</tr>
<tr>
<td>CONF.</td>
<td>mean 3.87</td>
<td>4.01</td>
</tr>
<tr>
<td></td>
<td>±SD 1.06</td>
<td>0.73</td>
</tr>
</tbody>
</table>
Table 6.6 Effect of exercise and diet on plasma free fatty acid concentration mM (Mean ±SD)

<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th></th>
<th></th>
<th>RUN 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
<td>PRE</td>
<td>POST</td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>mean</td>
<td>0.321</td>
<td>1.159*</td>
<td>0.248</td>
<td>0.957*</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>0.156</td>
<td>0.467</td>
<td>0.225</td>
<td>0.518</td>
</tr>
<tr>
<td>CONF.</td>
<td>mean</td>
<td>0.238</td>
<td>1.038*</td>
<td>0.190</td>
<td>1.100*</td>
</tr>
<tr>
<td></td>
<td>±SD</td>
<td>0.125</td>
<td>0.355</td>
<td>0.093</td>
<td>0.426</td>
</tr>
</tbody>
</table>

* Significantly different from preexercise levels p<0.005
<table>
<thead>
<tr>
<th></th>
<th>RUN 1</th>
<th>RUN 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
</tr>
<tr>
<td>CONTROL</td>
<td>mean 0.06</td>
<td>0.624*</td>
</tr>
<tr>
<td></td>
<td>±SD 0.025</td>
<td>0.144</td>
</tr>
<tr>
<td>CONF.</td>
<td>mean 0.07</td>
<td>0.58*</td>
</tr>
<tr>
<td></td>
<td>±SD 0.04</td>
<td>0.35</td>
</tr>
</tbody>
</table>

* significantly different from preexercise concentrations p<0.005
6.4 DISCUSSION

The aim of this study was to investigate the effect of supplementing an individual's normal diet with CHO, in the form of confectionery, on metabolism and recovery from prolonged exercise.

The cardiovascular system is responsible for the transport of oxygen and substrates to the working muscle, the rate of which depends upon the metabolic demands of the muscle. Therefore, at the onset of exercise heart rate, oxygen uptake and ventilation rate increase. During the prolonged running, in this study, there was a slight drift upwards in the heart rate although the metabolic demand of the activity remained constant. This drift has been attributed to several factors. Saltin and Stenberg (1964) suggest that functional changes in the myocardium are responsible for this drift and indeed Ekelund and colleagues (Ekelund, Holmgren and Ovensfors 1967) have demonstrated an increase in the duration of systole as exercise duration increases. Changes in plasma volume have also been implicated in causing the drift when plasma volume changes are large (Saltin 1964). If plasma volume decreases then stroke volume will decrease which will result in an increase in heart rate to maintain the cardiac output. Large changes in plasma volume were not seen in the present study and, moreover, changes in plasma volume have been reported to take place at the onset of exercise (Costill 1978) and so could not account for the increase in heart rate towards the end of exercise. The drift is most likely to be caused by thermoregulatory changes. As core temperature rises blood flow to the skin increases in order to increase heat transfer (Zitnik et al 1971). This increase in blood flow to the periphery causes a decrease in stroke volume and, therefore the heart rate increases to maintain cardiac output (Rowell 1974).

Adenosine triphosphate (ATP) the energy source for muscular contraction is not stored in any great quantity in the body. Its concentration in muscle is only 5-7 mol/g muscle (ww) (Beis and
Newsholme 1975) which would be depleted by less than a second of maximal exercise unless it was resynthesised at a rate equal to its utilisation (Newsholme and Start 1973). Exercise, therefore, not only increases the flux through the ATP producing pathways but also causes mobilisation and transport of metabolic fuels from their storage sites to the working muscle. During exercise the main metabolic fuels are fat and carbohydrate.

Fatigue during cycling is closely related to a decrease in muscle glycogen concentration, however, the same clear relationship has not been found during running probably due to involvement of a larger muscle mass and involvement of other factors eg. neurophysiological and psychological. Nevertheless, both cycling and running studies have shown that exercise time to exhaustion is related to pre exercise muscle glycogen levels (Bergstrom et al 1967; Galbo et al 1979) which can be increased by consuming a high cho diet following a prolonged period of exercise (Ahlborg et al 1967; Gollnick et al 1980).

Respiratory exchange ratios (R) are commonly used to estimate substrate utilisation. The R value represents the gas exchange over the lungs for the whole body. For this reason some workers have expressed doubts as to its validity as a method of determining the relative concentrations of substrates used by the working muscles (Jones et al 1980). This could be true when the muscle mass involved is small but as the muscle mass utilised during running is large then the R value can be assumed to be representative of the substrate utilisation by the working muscles. Recently Jansson (1982) showed that there was good agreement between the respiratory quotient across the working muscle and the whole body R value.

Higher R values following high CHO diets are in agreement with the view that high CHO diets lead to an increase in CHO oxidation by the muscle as a result of an increase in muscle glycogen (Hultman 1967; Gollnick et al 1972; Gollnick et al 1980). This increased reliance on muscle glycogen is thought to be due to an increased production of
insulin associated with high CHO diets (Costill et al 1977; Galbo et al 1979) which in turn lowers plasma FFA concentrations during exercise (Steinberg 1963; Rennie and Johnson 1974).

The ability to match and increase the running time observed after only 72 hours of recovery on the high CHO diet could be a function of an increased availability and oxidation of muscle glycogen. Previous studies have shown supercompensation of muscle glycogen concentrations to occur after 3 days of a high CHO diet following exhaustive exercise (Bergstrom and Hultman 1967; Piehl 1974).

Therefore, it would not be unreasonable to assume that such changes had occurred in the muscle glycogen content of the individuals in the confectionery group in the present study. Few other studies have examined the effect of high CHO diets on endurance running. Goforth and colleagues recently demonstrated that running at 80% of $V_{O_2\text{max}}$ after a high CHO diet achieved an improvement in endurance of 9% (Goforth et al 1980) compared to the 26% improvement observed in this study. This discrepancy could be due to the different exercise intensities used and, therefore, a different emphasis on substrate utilisation. At 80% $V_{O_2\text{max}}$ there would be a greater reliance of the muscle on glycogen to provide the energy required and, therefore, these stores may well be utilised at a faster rate than they would be at a lower exercise intensity (Hermansen et al 1967; Saltin and Karlsson 1971). At 70% $V_{O_2\text{max}}$ fat would provide a greater proportion of the energy source than at 80% $V_{O_2\text{max}}$ and so would have a relative glycogen sparing effect. It is possible that at work loads lower than 70%, an even greater improvement in endurance would be obtained after a high cho diet.

It is interesting that the control group were also able to match their previous performance after maintaining their normal CHO intake during the three days of recovery. Exhaustive exercise itself has been shown to be a potent stimulus for glycogen repletion (Young et al 1983) and this together with a normal CHO intake may well be enough to restore glycogen levels after three days. Several
investigators (Ahlborg 1967; Ivy et al 1982) have shown that consumption of a normal CHO intake following exhaustive exercise does allow repletion of glycogen levels to normal, albeit at slower rates than if a high CHO diet was consumed. It would, therefore, not be unreasonable to assume that the control group may have only achieved glycogen repletion to pre-exercise concentrations which would explain why the control group were unable to match but not improve on their performance. If the improvement in performance was simply a result of the extra energy intake over the preceding three days, then increased running times would have been achieved in both groups.

The control group showed no change in R value after dietary intervention. This suggests that the increase in fat intake did not cause a shift in substrate utilisation towards fat. The R value and, therefore, muscle substrate metabolism appears to be influenced to a greater extent by the amount of CHO consumed rather than the proportion of fat consumed in relation to CHO.

It has been suggested that fatigue is more likely to be a result of a reduction in the glycogen concentration in fibres that can be recruited at that exercise intensity to maintain the tension required, rather than total depletion (Sherman 1980; Essen et al 1971). The fact that R values were still quite high at exhaustion supports the idea that during running factors other than total muscle glycogen depletion contribute to limiting endurance capacity.

Blood glucose is mainly used for energy production in the central nervous system but some glucose is transported to the working muscle where it contributes to the oxidative metabolism, the extent depending on the exercise intensity and previous diet (Wahren, Felig, Ahlborg & Jorfeldt 1971; Keul et al 1967). In the present study there was no significant change in blood glucose concentration during exercise when compared to resting values. This is probably because although glucose output by the liver increases at the onset of exercise in response to an increase in catecholamine levels (Galbo et al 1979; Pequignot, Peyrin, Mayet and Flanrios 1979) there is also an
increase in uptake of glucose by the working muscle (Felig and Wahren 1975) and, therefore, concentrations in the blood will remain fairly stable.

Some studies have reported blood glucose levels falling as exercise duration increases (Maehlum and Hermansen 1978; Pruett 1971) due to a mismatching between production and utilisation (Ahlborg et al 1974; Ahlborg and Felig 1982). Hypoglycaemic symptoms of nausea, dizziness, mental confusion begin to appear at a blood glucose concentration of about 2.8 mmol/l and are fully developed at a concentration of about 2.2 mmol/l. Values as low as 1.4 mmol/l have been reported at the end of exercise (Felig, Cherif, Minagawa & Wahren 1982). Felig and colleagues (1982) reported that 30% of their subjects had blood glucose concentrations equal to or less than 2.5 mmol/l during exercise but despite this they were still able to continue exercising for up to 120 minutes longer suggesting that factors other than hypoglycaemia were responsible for causing fatigue. None of the subjects in the present study experienced such low blood glucose concentrations the lowest being 3.6 mmol/l which did not cause any apparent symptoms of hypoglycaemia. Glucose uptake by the muscle is inversely proportional to the glycogen content of the muscle (Gollnick et al 1980; Jansson 1980) in the presence of an adequate insulin concentration (Berger et al 1975). The absence of hypoglycaemia in the present study could be due to either a limited uptake of glucose or that factors other than low glycogen caused fatigue. If catecholamine concentrations were sufficiently high at 70% VO2max as to limit glucose uptake by the muscle, via their inhibition of insulin release, when muscle glycogen was low, symptoms of fatigue would result before those of hypoglycaemia, due to insufficient energy production. Alternatively if factors other than low muscle glycogen were the cause of fatigue then glucose uptake by the muscle would be minimal and so hypoglycaemia would not result.

Blood glucose contributes significantly to the muscle metabolism after low CHO diets and during moderate exercise intensities whereas high CHO diets and high work intensities tend to decrease the
contribution to total energy production from blood glucose (Wahren 1973). No significant difference in resting blood glucose concentration was found after the high CHO diet compared to the normal diet in this study. Other studies have also reported either no change or a slight though non-significant decrease in resting blood glucose as a result of consumption of a high CHO diet (Maughan et al 1978; Rennie and Johnson 1974; Goforth et al 1980; Sherman 1980; Pruett 1971). During exercise there was no further increase in blood glucose concentration due to an increased CHO intake. This has also been observed by other workers (Goforth et al 1980; Maughan et al 1978). This could be due to either no change in release of glucose from the liver or an increased release accompanied by an increased rate of uptake by the working muscle. Jansson (1980) found no diet related difference in glucose extraction by working muscle as measured by arterial-venous differences during cycling if anything there was a tendency for a greater uptake after a low CHO diet. Moreover, Bergstrom and Hultman (1967) found no increase in liver glucose output after a high CHO diet although liver glycogen levels would have been high (Hultman and Nilsson 1971). Therefore, it would seem that glucose release and rate of uptake are unaffected by diet although contribution by blood glucose to the total energy requirement of the working muscles is increased after a period of low CHO intake.

During exercise blood lactate concentrations were shown to increase from resting values as a result of an increase in the rate of carbohydrate metabolism. The rise in blood lactate is similar to that found in other studies performed at similar exercise intensities (Bergstrom et al 1967; Hermansen et al 1967; Costill et al 1974). Lactate accumulates in the muscle as a result of an imbalance between the rate of pyruvate production by glycogenolysis and entry of pyruvate into the tricarboxylic acid (TCA) cycle (Keul et al 1967; Holloszy and Booth 1976). If pyruvate were allowed to accumulate in the muscle cell glycogenolysis would be inhibited as regeneration of NAD (nicotinamide adenine dinucleotide) the necessary cofactor would stop. The production of lactate from pyruvate by the enzyme lactate
dehydrogenase (EC 1.1.1.27) allows regeneration of NAD and thus
glycogenolysis can continue. Lactate accumulation is most pronounced
at high work intensities as the anaerobic contribution to the energy
demands of the muscle increases (Hermansen and Stensvold 1972;

Exercise after a high CHO diet has been associated with an
increase in blood lactic acid concentration (Rennie and Johnson 1974;
Kelman et al 1975; Bergstrom et al 1967; Hultman 1967). In this study
blood lactate levels were not found to be significantly different
either at rest or during exercise after the confectionery diet when
compared to the values obtained after a mixed diet. Differences in
blood lactate levels have been reported, both in running and cycling
studies, following consumption of high and low CHO diets (Jansson et
al 1980; Galbo et al 1979). Few studies have been carried out
comparing the effect of a high CHO diet with a normal diet. Of those
that have, lactate levels have been reported to be higher during
exercise after a high CHO diet when compared to a normal diet in
cycling studies (Kelman et al 1975; Maughan et al 1978). Such a
relationship has not been found in running studies (Sherman 1980) and
the results of the present study are, therefore, consistent with
these previously reported studies.

Resting plasma FFA and glycerol concentrations prior to run 1 were
similar to those reported for 12 hour fasted subjects in other
studies (Rennie and Johnson 1974; Maughan et al 1978; Pruett 1970b)
which confirms that instructions were followed.

The mobilisation of FFA and glycerol from adipose tissue is
controlled by both hormones and metabolites. Triacylglycerols (TG)
are hydrolysed to form FFA and glycerol in the ratio 3:1. The FFA are
transported to the working muscle where they are taken up in direct
proportion to their concentration in the bloodstream (Armstrong et al
1961) to be used as a metabolic fuel whereas the glycerol molecules
are transported to the liver where they are converted into glycogen
by gluconeogenesis.
Exercise is known to cause an increase in plasma catecholamine concentration (Galbo, Holst and Christensen 1975; Galbo et al 1976; Watson et al 1980) and a decrease in plasma insulin concentration (Pruett 1970a; Blom et al 1976; Galbo, Holst and Christensen 1979) with a resultant increase in lipolysis.

Plasma FFA and glycerol concentrations increased during exercise as expected. There was, however, a large intra-individual variation in increase and this could possibly reflect differences in magnitude of hormonal responses to exercise.

High CHO diets could decrease resting plasma FFA and glycerol concentrations as a result of increased release of insulin from the pancreas in response to an increased absorption of glucose from the intestines. Conversely high CHO diets have been shown to increase plasma TG concentrations (Hayford, Danney, Wiebe, Roberts and Thompson 1979, Jette et al 1978). Excess CHO contributes to TG production in the liver by one of two ways both stimulated by insulin. Glucose can be converted to either glycerol-3-phosphate or acetyl CoA, a major precursor for fatty acyl CoA. Glycerol-3-phosphate is acylated by acyl CoA to form triacylglycerols.

Previous studies have shown an increase in FFA and glycerol concentrations during the traditional low phase of carbohydrate-loading which normalise when the high CHO phase is followed (Pruett 1971; Maughan et al 1978; Jansson 1980). Few studies have used cho loading without the low phase but those that have have observed a slight though non-significant decrease in plasma FFA concentration due to the short period of increased CHO consumption (Sherman 1980). Previous studies using a high fat diet have demonstrated an increase in FFA and glycerol concentrations (Pruett 1970b; Maughan et al 1978; Jansson 1980) as a result of decreased insulin release (Galbo et al 1980). However, although the control diet used in this study contained a larger quantity of fat than the mixed diet it did not cause an increase in plasma FFA concentration.
It may be that sufficient CHO was available to prevent any decrease in plasma insulin.

The difference in response of cholesterol to exercise exhibited by females compared to males is interesting. A similar sex related difference has been reported by Robinson and colleagues (1974) who demonstrated that plasma cholesterol concentration decreased in female rats but not in male rats in response to exercise. As the mechanism of cholesterol metabolism during exercise is not clear it is difficult to explain this difference. The effect of acute exercise on cholesterol concentrations is a controversial subject. Several studies have shown total cholesterol concentrations to be unchanged by acute exercise, others have shown cholesterol concentrations to decrease (Dufaux, Assman, Svachten and Hollman 1982; Thompson, Cullinane, Henderson and Herbert 1980) whilst some workers have shown cholesterol concentrations to increase (Hurter et al 1972). Thompson (1980) demonstrated that there was a delay in the response of cholesterol to exercise which could explain the different responses. The existence of cholesterol in different forms: High density lipoproteins (HDL), Low density lipoproteins (LDL) and very low density lipoproteins (VLDL), which respond differently to exercise (Peltonen et al 1981; Huttunen et al 1979) must also account for the different reports. However, it seems most likely that if cholesterol does change during exercise it is HDL cholesterol that is affected (Enger, Stromme and Refsum 1980).

Several previous studies have shown that 2-5 grammes of water are stored in association with each gramme of muscle glycogen (McBride et al 1941; Olsen and Saltin 1970). It has been suggested (Sharman 1981) that this bound water is liberated as the glycogen is utilised and so may offset the effects of dehydration. However, no significant difference in fluid intake between the two runs by the confectionery group was observed in this study nor were there any significant changes in plasma volume. It has also been suggested that that the additional water stored with glycogen may cause a sufficient increase in body weight as to be detrimental to performance (Brotherhood and
Swanson (1979). Cureton (1978) has demonstrated that a 5% increase in body weight can significantly increase the oxygen uptake of an individual at submaximal work loads. Body weight was not found to be significantly different after either the confectionery or control diet when compared to that after the mixed diet. Any change in body weight did not affect oxygen uptakes as these were found to be very similar during run 1 and run 2.

In summary, supplementation of the diet with confectionery products appears to enhance recovery from prolonged exercise. This is shown by the fact that during run 2 those individuals in this group were able to match and improve on their running time to exhaustion achieved during run 1. In addition this diet produced significant changes in certain metabolic responses to exercise.
CHAPERR 7

SUMMARY

This study has highlighted many important points related to diet and exercise. The principal finding was that supplementation of the diet with CHO, in the form of confectionery, following prolonged exercise can facilitate recovery. This was shown by the fact that after a period of only three days, following exhaustive exercise, those individuals consuming a high CHO diet showed an improvement on their previous running time of 22.8%. A similar improvement in running time (25.7%) was also shown by those whose diets were supplemented with conventional high CHO foods. In comparison the control group showed only a small improvement in running time of 2.7%.

While one would not recommend consumption of large quantities of confectionery in the long term dietary planning of an individual, the results have shown that such a convenience food can aid recovery and so could be used to supplement the diet when the more conventional high CHO foods are unavailable.

In order to compare the efficacy of the confectionery diet on recovery from prolonged exercise with the more conventional high CHO diet and a control diet, careful dietary prescription was required. To achieve this, using diets familiar to the subjects, a system of dietary exchanges was designed. These were shown to provide the subject with a flexible diet whilst still allowing the dietary goals to be achieved.

In addition the examination of the normal diets of active individuals (recreational and marathon runners) revealed energy intakes lower than might be expected considering the individuals' daily activity levels. No difference was found in energy intakes of either male recreational and marathon runners (13.2 ±3.5 MJ vs 12.9 ±2.3 MJ) or female recreational and marathon runners (8.2 ±1.5 MJ).
vis 8.5 ± 2.4 MJ). Also several individuals in the study had daily CHO intakes lower than the 50% contribution of their energy intake as recommended by NACNE (1983). Such inadequacies, ie low energy and CHO intakes, in an individual's normal diet could limit endurance capacity during training.

Further research

Although it is well established that depletion of muscle glycogen plays a central role in the onset of fatigue during cycling, there is still little known about the mechanisms involved in the cause of fatigue during prolonged running, and these invite further investigation.

The ability of a high CHO diet to facilitate recovery after a much shorter time period than that used in this study also invite further investigation.

In addition the question of how much CHO is necessary to achieve maximum glycogen repletion needs to be reviewed and clearly a case exists for the amount of CHO required being related to muscle mass.
REFERENCES


NATIONAL ADVISORY COMMITTEE ON NUTRITION EDUCATION (1983). A discussion paper on proposals for nutritional guidelines for health education in Britain. Health Education Committee


APPENDICES
1. The expired air collected was allowed to stand for several minutes to ensure a homogeneous sample.

2. Samples of the expired air were passed through the carbon dioxide and oxygen analysers for 120 and 60 seconds respectively and the percentage of these gases in the sample was recorded.

   The flow rate was measured on a Gap flow meter (Platon Flowbits Ltd) and recorded.

3. The Douglas bag was evacuated through a vacuum pump connected to a dry gas meter and the volume noted. This volume was added to that used for analysis to obtain the volume of air expired in one minute.

   The temperature of the gas sample was also noted.

   To correct this volume to STPD the following equation is used

\[
V_e(\text{STPD}) = V_e \times \frac{273}{273 + t^\circ C} \times BP - SWVP^t^\circ C
\]

Where

- \( V_e \) is the volume of expired air (l min\(^{-1}\))
- \( t^\circ C \) is the temperature of the expired air
- \( BP \) is the barometric pressure (mm Hg)
- \( SWVP^t^\circ C \) is the saturated water vapour pressure at \( t^\circ C \).

4. In order to calculate \( VO_2 \) (oxygen uptake) and \( VCO_2 \) (carbon dioxide production) the volume of air inspired needed to be calculated. This was accomplished by using the Haldane transformation which makes use of the concentration of nitrogen (a metabolically inert gas) in the inspired and expired air.

\[
V_i = \frac{F_e N_2 \% \times V_E}{F_i N_2 \%}
\]
where: $V_i$ is the volume of inspired air
$V_e$ is the volume of expired air
$F_i N_2 \%$ is the percentage of nitrogen in the inspired air
$F_e N_2 \%$ is the percentage of nitrogen in the expired air

5. $V O_2 (l \ min^{-1})$ could now be calculated.

$$V O_2 = V_i O_2 - V_e O_2$$

$$V_i O_2 = \frac{V_i \times F_i O_2 \%}{100}$$

$$V_e O_2 = \frac{V_e \times F_e O_2 \%}{100}$$

6. Similarly $VCO_2 (l \ min^{-1})$ was calculated. Thus:

$$VCO_2 = V_e CO_2 - V_i CO_2$$

and so

$$VCO_2 = \frac{V_e \times F_e CO_2 \% - V_i \times F_i CO_2 \%}{100}$$

$F_i O_2 \%$, $F_i CO_2 \%$ and $F_i N_2 \%$ can be assumed to be 20.93\%, 0.03\% and 79.04\% respectively.

7. $V O_2$ and $VCO_2$ were then divided by body weight in kg and converted to millilitres to obtain values in millilitres per kilogram of body weight per minute (ml kg$^{-1}$ min$^{-1}$).

8. The respiratory exchange ratio ($R$) was calculated from the following equation:

$$R = \frac{VCO_2}{VO_2}$$
APPENDIX B

METABOLITE ASSAYS

LACTIC ACID ASSAY

An adaptation of the method described by Olsen (1971) was used. This depends on the fact that NADH is liberated by the following reaction and this is measured by its native fluorescence.

Lactate + NAD$^+\rightarrow$ Pyruvate + NADH

Solutions

Perchloric acid: 2.5% w/v.
Hydrazine buffer (1.1M, pH 9.0): 1.3g hydrazine sulphate, 5.0g hydrazine hydrate and 0.2g disodium ethylenediaminetetraacetic acid (EDTA) in 100 ml distilled water.
Reaction mixture: Prepared immediately before use. 2mg NAD$^+$ and 10μl LDH per ml hydrazine buffer.

Standards

From 1.0M Sodium L-lactate stock solution standards were made.

Deproteinization

25μl of blood was deproteinized by addition to 250 μl of perchloric acid. After mixing thoroughly the mixture was centrifuged. The samples were kept at -20°C until analysis.

Procedure

1. The samples were removed from the freezer and defrosted at room
2. The samples were mixed thoroughly and centrifuged.

3. 25 μl of the supernatant or standard was transferred to a clean test tube and 250 μl of reaction mixture was added.

4. The contents of the tubes were mixed and incubated at room temperature for 30 minutes.

5. Following incubation 1 ml of diluent was added and the samples read with a Perkin Elmer fluorometer against a series of standards and a blank treated in the same way as the samples.

6. After subtraction of the blank value from readings of the samples and standards the concentration of lactate in each sample was calculated from the standard curve.
GLUCOSE ASSAY

A colourimetric method based on the following principles was used.

\[
\text{Glucose} + O_2 + H_2O \rightarrow \text{Gluconate} + H_2O_2 \\
H_2O_2 + \text{ABTS}^* \rightarrow \text{Coloured complex} + H_2O
\]

Solutions

Phosphate buffer: 100 mmol/l, pH 7.0
POD: > 0.8 U/ml
GOD: > 10.0 U/ml
ABTS*: 1.0 mg/ml

Standard

A 0.505 mmol l\(^{-1}\) standard was used.

Deproteinisation

Blood was deproteinized as described for the lactate assay.

Procedure

1. Samples were removed from freezer and defrosted at room temperature for 1 hour.

2. Samples were mixed thoroughly and centrifuged.

3. 20 µl of supernatant and standard (reaction mixture for blank) was placed in a tube with 1 ml of reaction mixture and mixed well.

4. The mixture was then incubated at room temperature for 20 minutes.

5. The absorbance of the standard and samples was measured at Hg 436 nm with an Eppendorf photometer in a cuvette of 1 cm light path.
6. The concentration (mmol l\(^{-1}\)) of glucose in the samples was calculated using the following equation:

\[
c = 5.5 \times \frac{A_{\text{sample}}}{A_{\text{standard}}}
\]

\(\text{*} = \text{di-ammonium 2,2'-azino-bis(3-ethylbenzothiazoline-6-sulphonate)}\)

A Boehringer Mannheim diagnostic kit was used for this assay.
HAEMOGLOBIN ASSAY

The cyanmethaemoglobin method was used. This is a colorimetric method and is based on the following principle

\[ \text{Haemoglobin} + \text{cyanide} + \text{ferricyanide} \rightarrow \text{cyanmethaemoglobin} \]

Solutions

Colour reagent: 1.63 mmol/l phosphate buffer, 0.75 mmol/l potassium cyanide and 0.6 mmol/l potassium ferricyanide, 5% detergent.

Drabkins reagent:

Procedure

1. 0.02 ml of blood was added to 5.0 ml of Drabkin’s reagent and mixed well.

2. The solution was incubated at room temperature for at least 3 mins.

3. The absorbance (A) of the samples was measured in an Eppendorf photometer at Hg 546 nm in a cuvette with 1 cm light path against a blank of distilled water.

4. The concentration of haemoglobin in the samples was calculated using the following equation
   \[ c = 36.77 \times A \ (g/100ml) \]

This assay was carried out using a Boehringer Mannheim diagnostic kit.
**FFA ASSAY**

FFA were analysed using a modification of the photometric, colorometric assay (Chromy et al 1977).

**Solutions**

Extraction solvent (CHM): 280 ml chloroform, 210 ml n-heptane and 10 ml methanol.

Stable copper reagent: 1.878 g sodium citrate, 16.775 g triethanolamine, 8.125 g copper nitrate and 62.5 g sodium chloride made up to 250 ml with distilled water.

TAC: 10 mg 2-thiozolylazo-p-cresol in 100 ml ethanol.

**Standards**

From a 4 mM stock solution and CHM of palmitic acid 0.2 mM, 0.4 mM, 0.8 mM and 1.0 mM standards were made. These were kept refrigerated in glass bottles with plastic screw caps stable to CHM until need.

**Procedure**

1. 100 µl of plasma or standard (CHM for blank) was added to 3 ml CHM in acid washed screw-capped glass tubes.

2. 1 ml of stable copper reagent was added.

3. The tubes were shaken vigorously for 6 minutes and centrifuged at 6000 rpm in a refrigerated centrifuge for 5 minutes.

4. 1 ml of the upper phase was transferred to a tube containing 0.25 ml TAC and the resulting solution mixed well.

5. The samples and standards were read at Hg 578 nm in cuvettes of 1 cm light path in an Eppendorf photometer. The concentration of FFA in the samples was determined from the standard curve.
GLYCEROL ASSAY

A fluorometric assay modified from Laurell and Tibbling (1966) was used. This method is based on the following reactions:

Glycerol + ATP $\rightarrow$ Glycerol-1-Phosphate + ADP

Glycerol-1-Phosphate + NAD$^+$ $\rightarrow$ Dihydroxyacetone Phosphate + NADH

The dihydroxyacetone phosphate produced is trapped by hydrazine and the amount of NADH formed is determined by measurement of its native fluorescence.

Solutions
Zinc sulphate: 0.087M
Barium hydroxide: 0.083M
Hydrazine HCl buffer: 1M reagent grade with 1.5mM MgCl$_2$ adjusted to pH 9.4 with HCl.
Diluent: 0.01M NaOH with 1mM EDTA.
Glycerokinase: 1mg/ml (Boehringer Biochemica).
Glycerin 3 phosphate dehydrogenase: 1mg/ml (Boehringer Biochemica).
Reaction mixture: Per 5ml reaction mixture. 3.5ml Hydrazine HCl buffer, 1.5ml distilled water, 6mg ATP, 10mg NAD, 12.1mg cysteine, 5µl glycerokinase and 25µl glycerin 3 phosphate dehydrogenase.

Standards
From a 0.4mM stock solution 20%, 40%, 60%, 80% and 100% standards were made using distilled water.

Deproteinization
0.1ml of plasma or standard (distilled water as blank) was transferred to a small centrifuge tube containing 0.5ml zinc sulphate. The resulting solution was then mixed and chilled. Next 0.5ml of barium hydroxide was added, the solutions mixed and allowed to stand for 5 minutes until centrifugation at 12000 rpm for
Procedure

1. 0.1ml of reaction mixture and 0.2ml of supernatant were transferred to a test tube and mixed.

2. The tubes were capped and left at room temperature for 60 minutes.

3. 1ml of diluent was added and the samples and standards were read in a Lochart fluorimeter. After subtracting the blank from the readings of the standards and samples the concentration of glycerol in the samples was determined from the standard curve.
**CHOLESTEROL ASSAY**

An enzymatic colourimetric method based on the following reactions was used.

\[
\text{cholesterol ester} + H_2O \xrightarrow{\text{cholesterol esterase}} \text{cholesterol} + \text{RCOOH}
\]

\[
\text{cholesterol} + O_2 \xrightarrow{\text{cholesterol oxidase}} \text{4-cholestenone} + H_2O_2
\]

\[
2H_2O_2 + \text{4-aminophenazone} + \text{phenol} \xrightarrow{\text{POD}} \text{4-(p-benzoquinone-mono-imino)-phenazone} + 4H_2O
\]

**Solutions**

Reaction mixture: Tris buffer pH 7.7 (100mM), Magnesium aspartate (50mM), 4-aminophenazone (1mM), sodium cholate (10mM), phenol (6mM), 3,4-dichlorophenol (4mM), hydroxypolyethoxy-n-alkanes 0.3%, cholesterol esterase > 0.4 U/ml, cholesterol oxidase > 0.25 U/ml, peroxidase > 0.2 U/ml

**Procedure**

1. 0.02 ml of plasma (reaction mixture for blank) was added to 1 ml of reaction mixture and thoroughly mixed.

2. The mixture was then incubated for 30 minutes at room temperature.

3. The absorbance (A) of the sample was then measured in a glass cuvette of 1 cm light path at Hg 546 nm in an Eppendorf photometer. The concentration of cholesterol in the samples was determined from the standard curve.

This assay was carried out using a Boehringer Mannheim diagnostic kit.
APPENDIX C

FOOD EXCHANGE GROUPS

DIETS

In order to provide you with the maximum amount of choice possible this diet is based on 'exchanges', a dietary term used to describe foods that can be substituted for each other. An exchange is an equivalent or a substitute. There are six exchange groups used in this diet. Within each group, each exchange is approximately equal in calories and carbohydrate (CHO), but obviously there is a certain amount of variation. Because of this variation please try to choose a variety of foods from each exchange group to ensure that the desired calorie and CHO intake is achieved. Tea and coffee intake is unlimited but any milk or sugar you add must be checked off your daily exchange allowance. You are required to follow each diet plan for three days. Dietary advice will be given if required.

DAILY DIET PLAN

<table>
<thead>
<tr>
<th>PHASE I</th>
<th>PHASE II</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Days 1-3 before run1)</td>
<td>(Days 1-3 before run 2)</td>
</tr>
</tbody>
</table>

Bread exchanges
Meat exchanges
Veg. exchanges
Fruit exchanges
Fat exchanges
Milk exchanges

DAILY CALORIES
DAILY CHO (g)
FOOD EXCHANGE GROUPS

BREAD EXCHANGES

All foods within this group can be interchanged.

<table>
<thead>
<tr>
<th>AMOUNT (g)</th>
<th>FOOD</th>
<th>KCAL</th>
<th>CHO</th>
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<tbody>
<tr>
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MEAT EXCHANGES
All foods within this group can be interchanged.

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<th>CHO (g)</th>
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<tbody>
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<td>LIVER</td>
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VEGETABLE EXCHANGES
All foods within this group can be interchanged.

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FRUIT EXCHANGES
All foods within this group can be interchanged.

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<th>CHO (g)</th>
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<td>APPLE</td>
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FAT EXCHANGES
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**MILK EXCHANGES**

All foods within this group can be interchanged.

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<th>CHO (g)</th>
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<td>CUSTARD</td>
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<td>ICE-CREAM</td>
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**MISCELLANEOUS**

Foods have been put in this group which do not fit into one single food group.

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<td>CHEESECAKE</td>
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</tr>
<tr>
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<td>CRISPS</td>
<td>1 BREAD EXCHANGE+1 FAT EXCHANGE</td>
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<td>DOUGHNUT</td>
<td>1 BREAD EXCHANGE+1 FAT EXCHANGE</td>
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<td>WHITE SAUCE</td>
<td>1 MILK EXCHANGE+1 FAT EXCHANGE</td>
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<tr>
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<td>PANCAKES</td>
<td>1 BREAD EXCHANGE+1 FAT EXCHANGE</td>
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<td>1 MEAT EXCHANGE+1 BREAD EXCHANGE</td>
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<tr>
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<td>CORNISH PASTIE</td>
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<td>QUICHE LORRAINE</td>
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<td>1 BREAD EXCHANGE+1 MILK EXCHANGE</td>
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Below are some examples of the number of exchanges that would be prescribed per day for an individual whose normal daily energy intake was 12.6 MJ of which 46% was from CHO.

**MIXED DIET**

<table>
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<tr>
<th>Bread exchanges</th>
<th>17</th>
<th>Meat exchanges</th>
<th>8</th>
<th>Vegetable exchanges</th>
<th>3</th>
<th>Fruit exchanges</th>
<th>5</th>
<th>Fat exchanges</th>
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<th>Milk exchanges</th>
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<td>Daily CHO intake</td>
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**CONTROL**

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<th>Vegetable exchanges</th>
<th>3</th>
<th>Fruit exchanges</th>
<th>6</th>
<th>Fat exchanges</th>
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<th>Milk exchanges</th>
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**HIGH CHO-CONFECTIONERY**

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<th>4 MARS BARS + 1 PKT OPAL FRUITS</th>
<th>Bread exchanges</th>
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<th>Vegetable exchanges</th>
<th>3</th>
<th>Fruit exchanges</th>
<th>5</th>
<th>Fat exchanges</th>
<th>5</th>
<th>Milk exchanges</th>
<th>3</th>
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</thead>
<tbody>
<tr>
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**HIGH ENERGY-HIGH CHO**

<table>
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<th>Fat exchanges</th>
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<th>Milk exchanges</th>
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<tbody>
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